ENVE 576
Indoor Air Pollution
Fall 2014

Week 11: November 4, 2014
1. SVOCs
2. Health effects of indoor and outdoor air pollution

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

Built Environment Research @ IIT

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

Built Environment Research Group
www.built-envi.com

Updates

Last time:

- Particle filtration

Today:

- Finish particle filtration (stand-alone air cleaners)
- SVOCs
- Health effects and epidemiology

Take home-exam release today

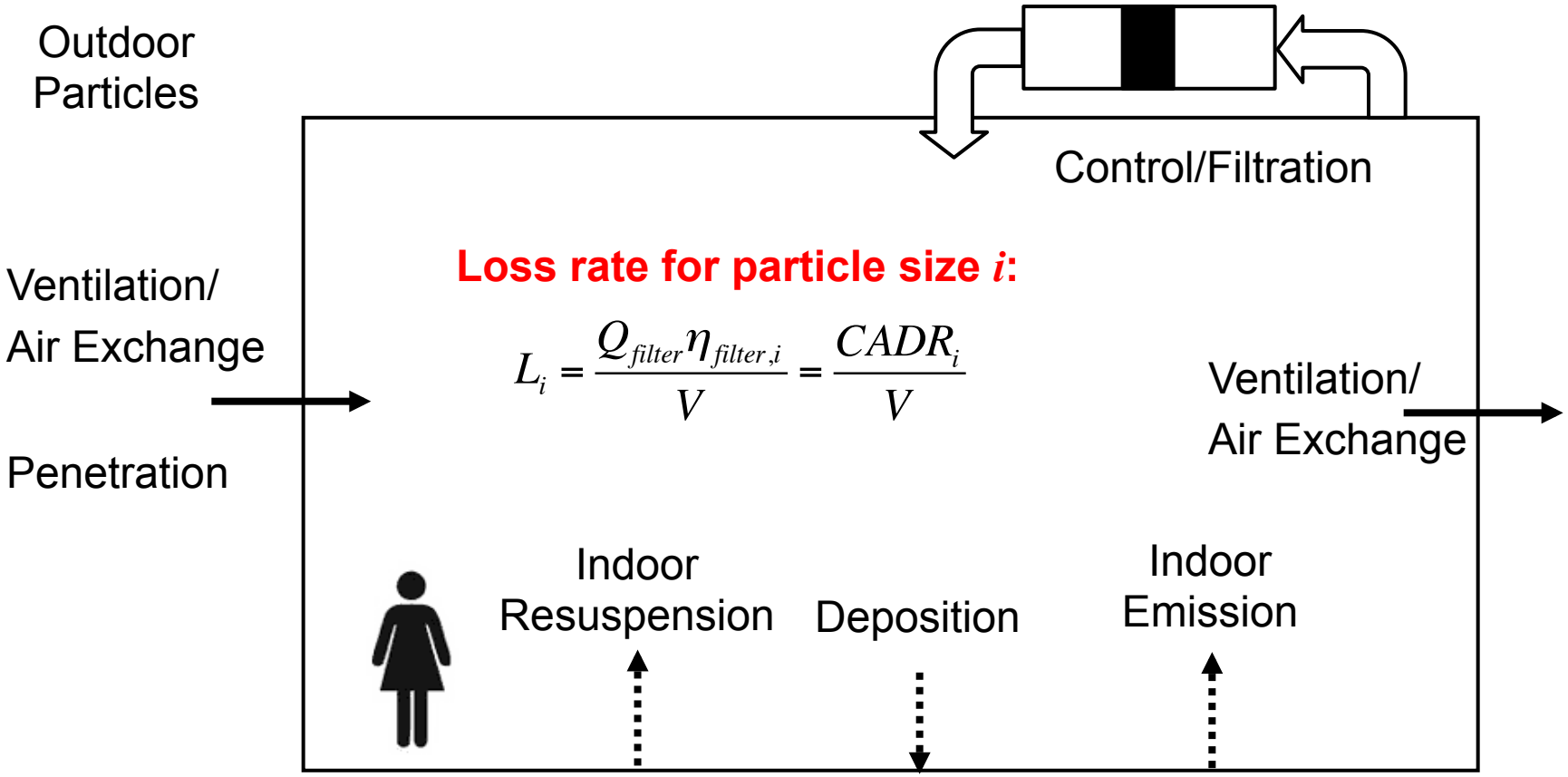
- Due Tuesday November 11

Updated schedule

Course Topics and Tentative Schedule

Week	Date	Lecture Topics	Reading*	Assignment:
1	Aug 26	Introduction to topic/field • Indoor and outdoor atmospheres • Fundamental air principles	1–3	
2	Sep 2	Reactor models • Steady-state and dynamic Ventilation and air exchange rates Human exposure patterns • Inhalation and intake fractions	4–6	
3	Sep 9	Overview of indoor pollutants/constituents • Particulate matter • Gas-phase compounds ⇒ Organic and inorganic • Biological	7	HW1 due
4	Sep 16	Gaseous pollutants • Sources • Emission models	8–11	Blog #1 due
5	Sep 23	Gaseous pollutants • Adsorption/desorption • Reactive surface deposition • Homogenous chemistry • Byproduct formation (including SOA)	12–14	HW2 due
6	Sep 30	Particulate matter • Single particle physics and dynamics • Particle size distributions • Respiratory deposition	15–17	Blog #2 due
7	Oct 7	*Lecture cancelled due to illness*		HW3 due
8	Oct 14	*Conference travel – guest lecture* • *Dr. Stephanie Kunkel: Indoor air microbiology*	18–20	
9	Oct 21	Particulate matter • Particle sources (indoor and outdoor) • Deposition and resuspension • Penetration/infiltration	21–26	
10	Oct 28	Particulate matter • Filtration and air cleaners	27–29	HW4 due
11	Nov 4	SVOCs Health effects • Epidemiology and physical responses	30–32	Blog #3 due Exam assigned
12	Nov 11	IAQ in developing countries IAQ measurement techniques	33–35	Exam due
13	Nov 18	IAQ measurement techniques (cont.) Infectious disease transmission	39–41	
14	Nov 25	Applications • Standards and manufacturer ratings Modeling software	36–38	Blog #4 due
15	Dec 2	*Lecture cancelled: Review panel travel*		
Final	Dec 9 5-7 PM	No final exam – final presentations		Final project due Blog #5 due

Particle filtration and air cleaners



Total efficiency for an example filter

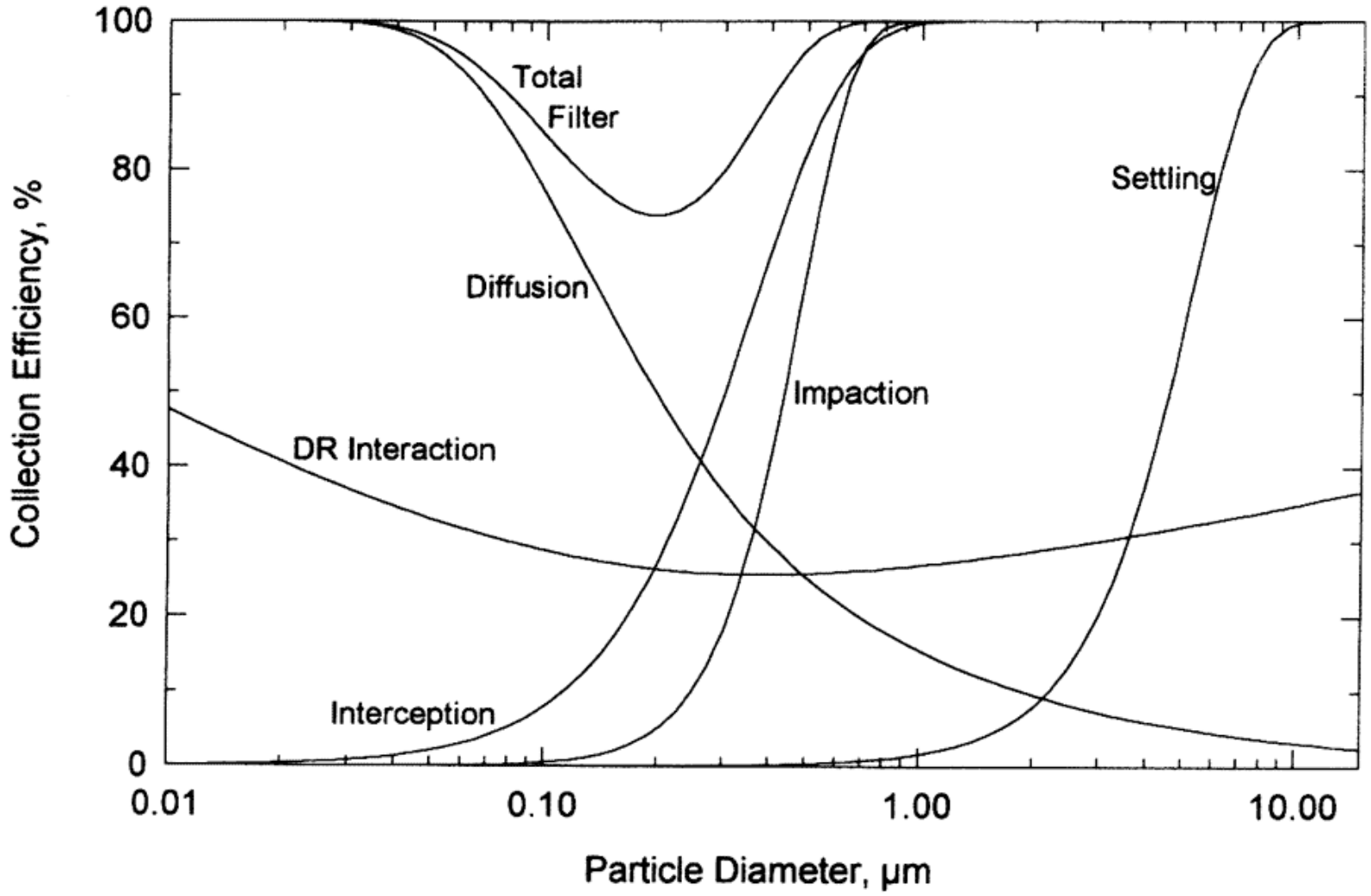


FIGURE 9.8 Filter efficiency for individual single-fiber mechanisms and total efficiency; $t = 1$ mm, $\alpha = 0.05$, $d_f = 2$ μm, and $U_0 = 0.10$ m/s. [10 cm/s].

STAND-ALONE AIR CLEANERS

Stand-alone air cleaners

- Another major type of filter is a stand-alone air cleaner
  - i.e. ‘room air cleaners’ or ‘portable air cleaners’

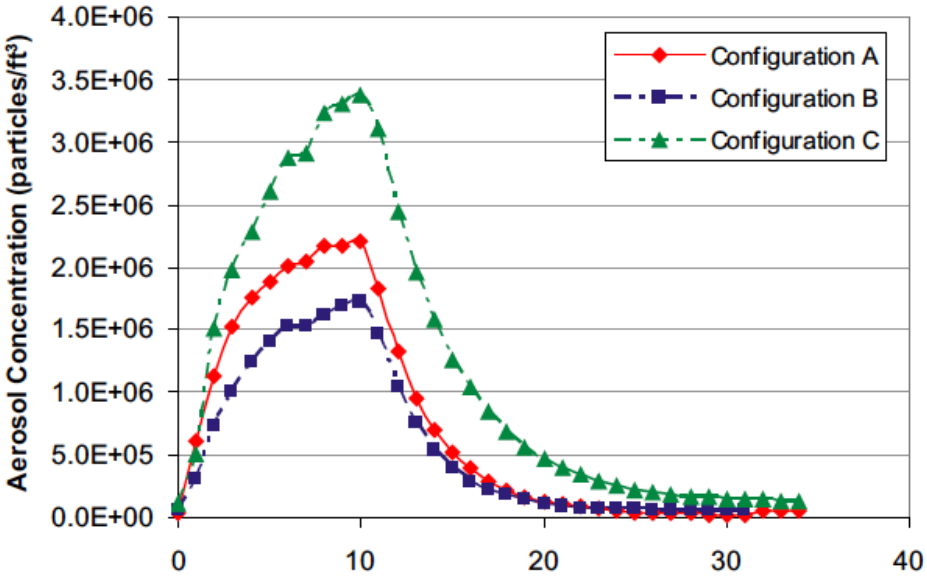
Photo from M.S. Waring and J.A. Siegel

Recent testing of portable air cleaners

- A few recent studies on particle removal by portable air cleaners
  - First dates back to 1985 (Offermann et al., *Atmos Environ*)
- Basic procedure involves elevating aerosol concentrations
  - Measuring subsequent decay with and without air cleaner operating

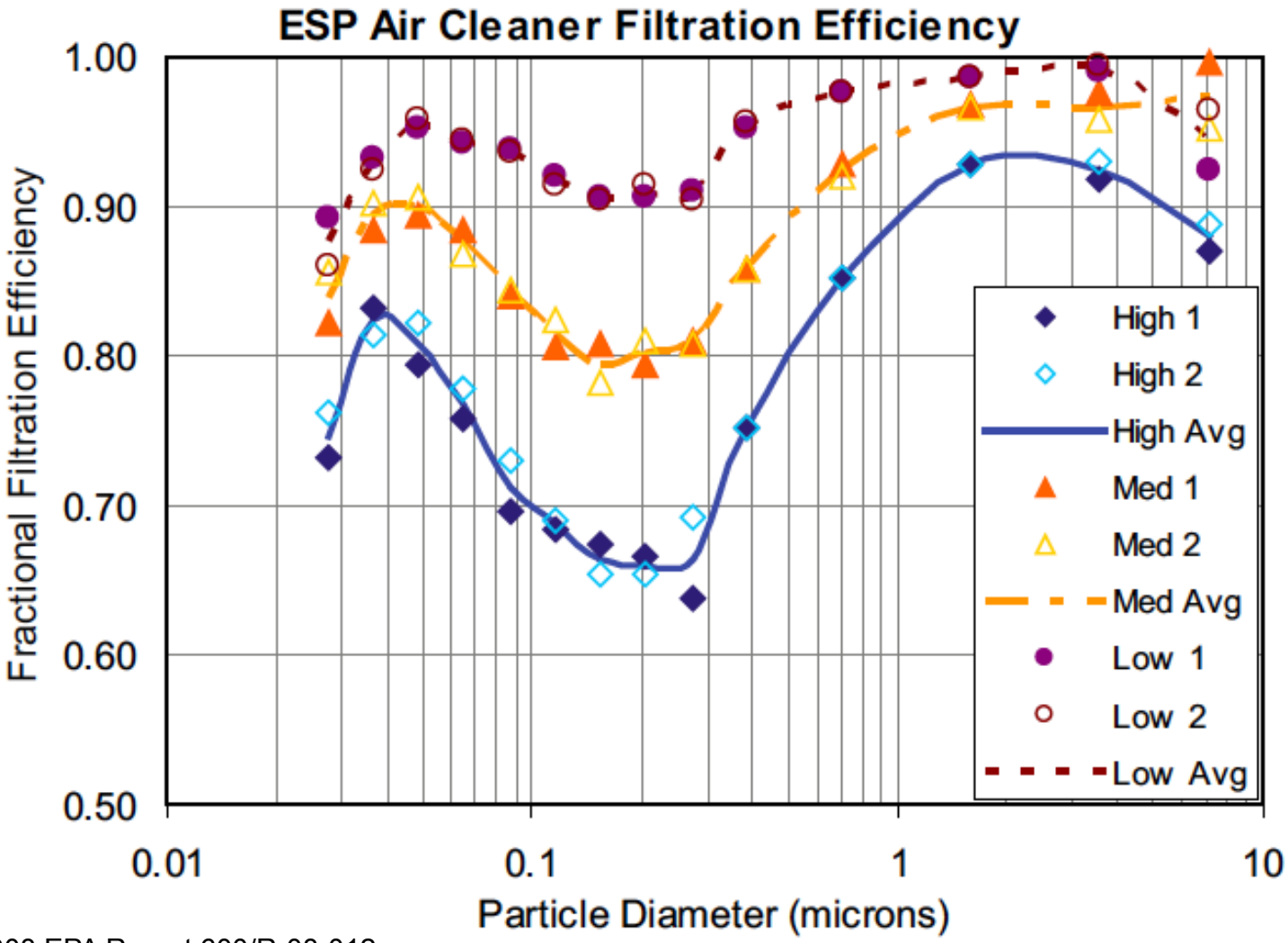
Kogan et al., 2008 EPA Report 600/R-08-012

$$CADR = V\left(L_{AC} - L_{noAC}\right)$$



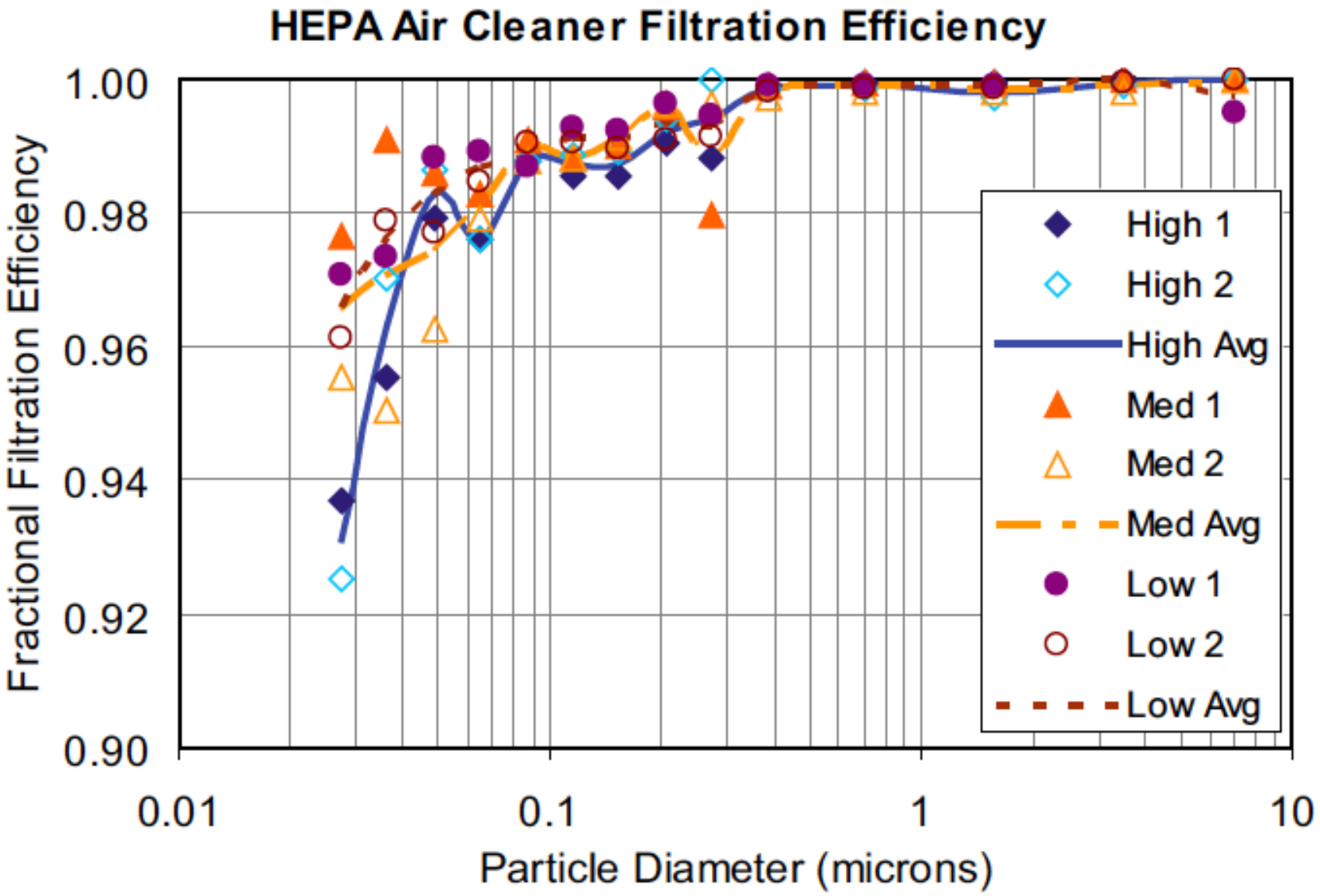
Recent testing of portable air cleaners

$$CADR = V\left(L_{AC} - L_{noAC}\right) \longrightarrow \eta = \frac{CADR}{Q_{AC}}$$



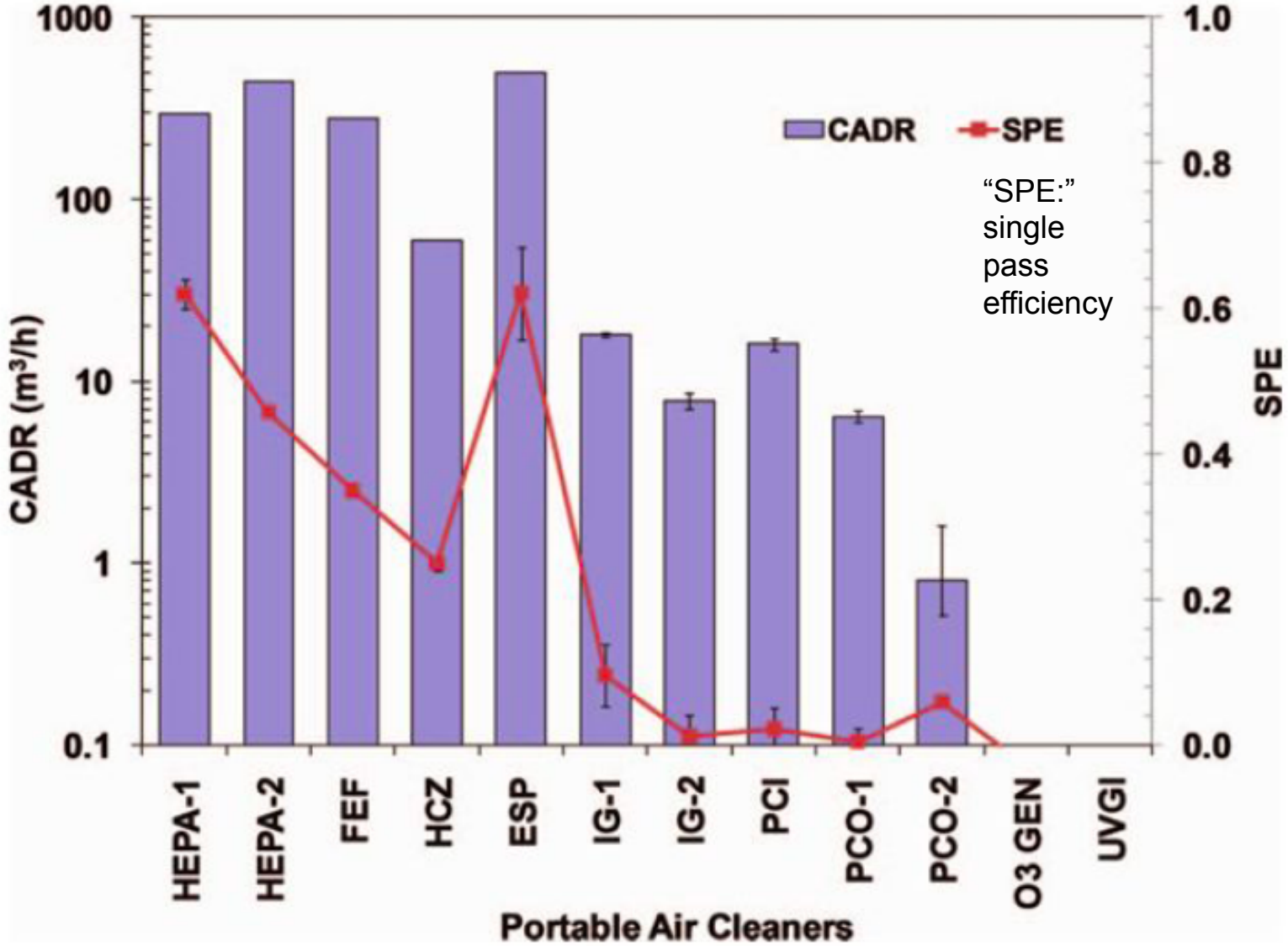
Kogan et al., 2008 EPA Report 600/R-08-012

Recent testing of portable air cleaners



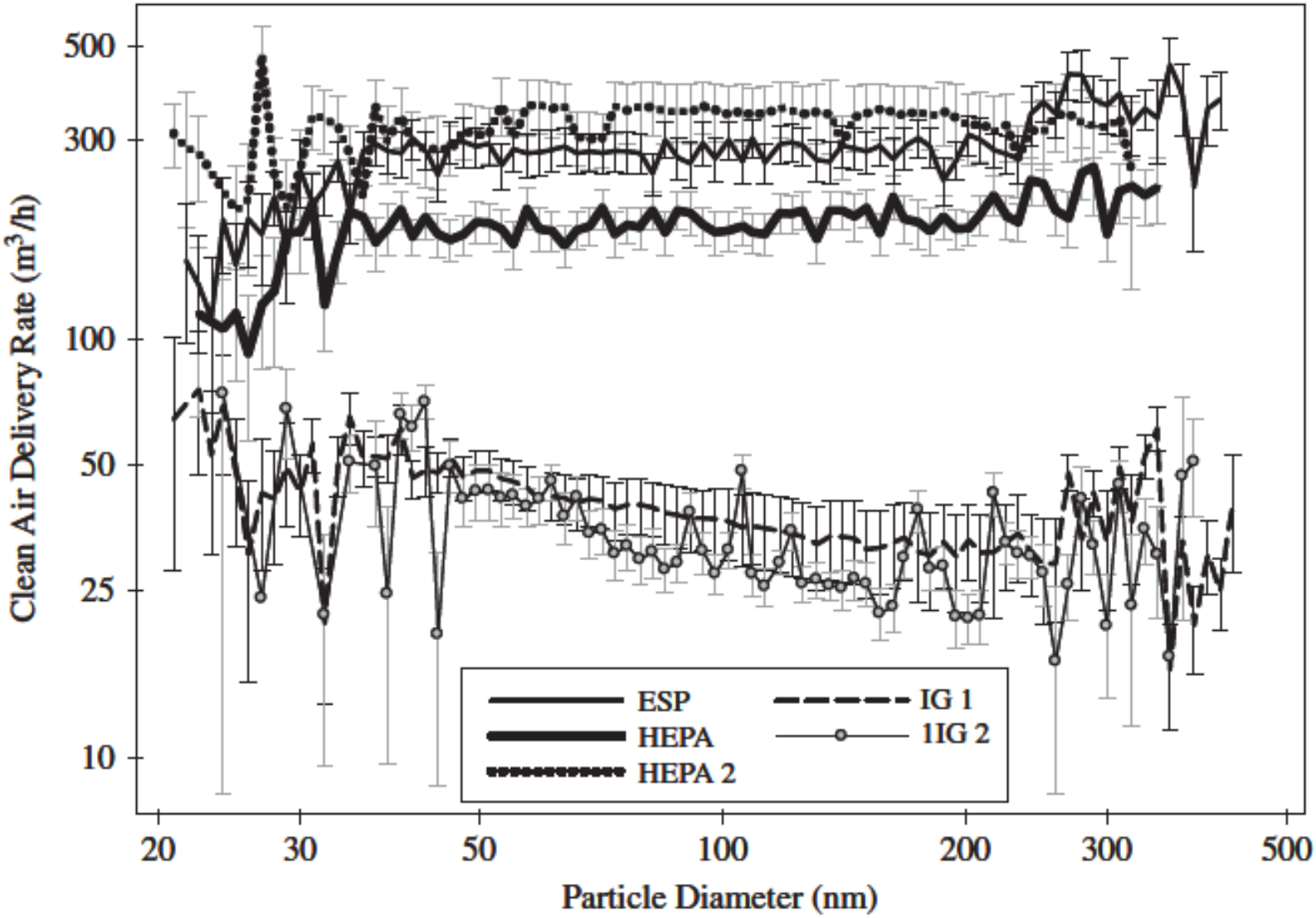
Kogan et al., 2008 EPA Report 600/R-08-012

Recent testing of portable air cleaners: UFPs



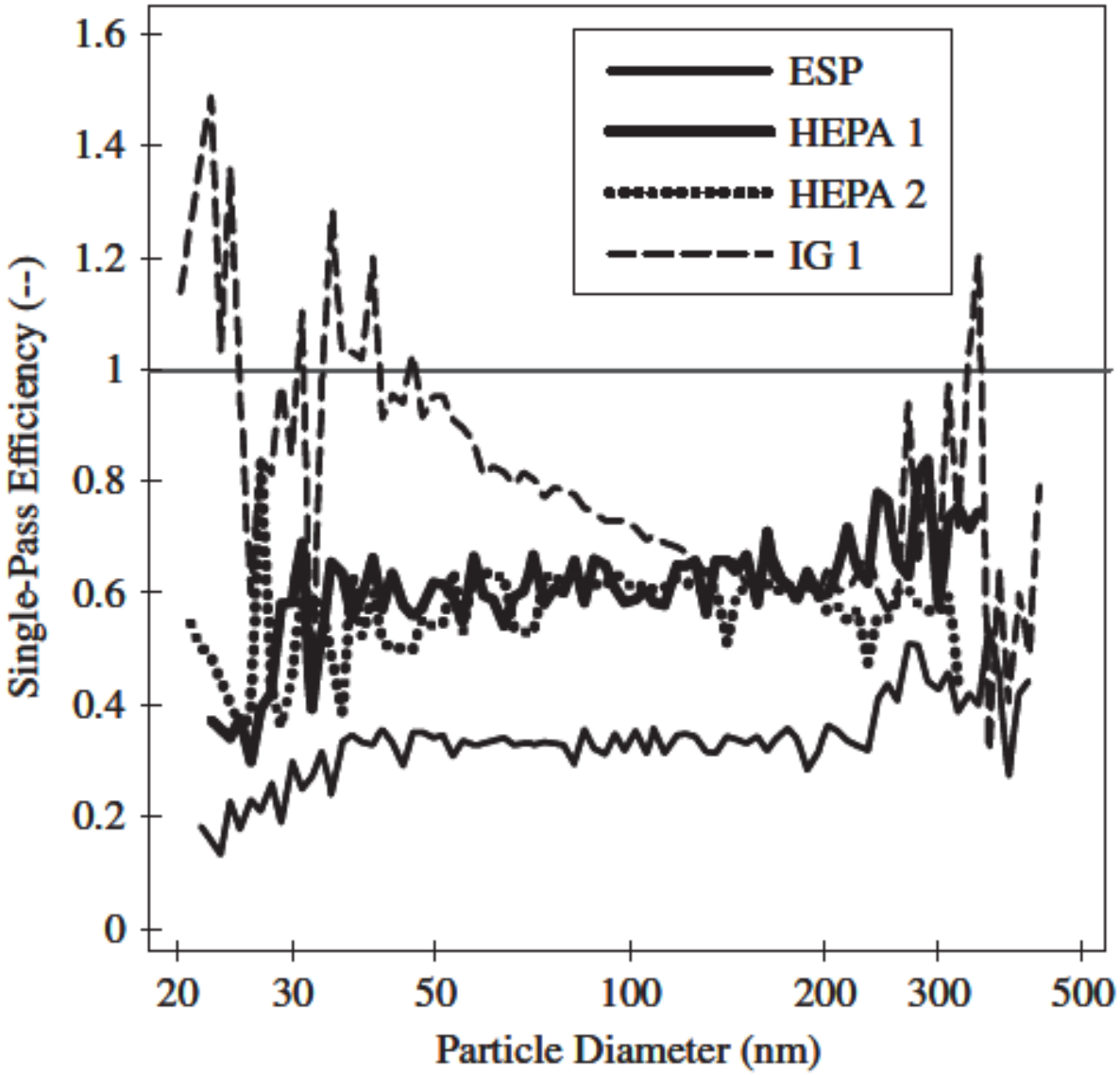
Sultan et al., 2011 *HVAC&R Research*

Recent testing of portable air cleaners



Waring et al., 2009 *Atmos Environ*

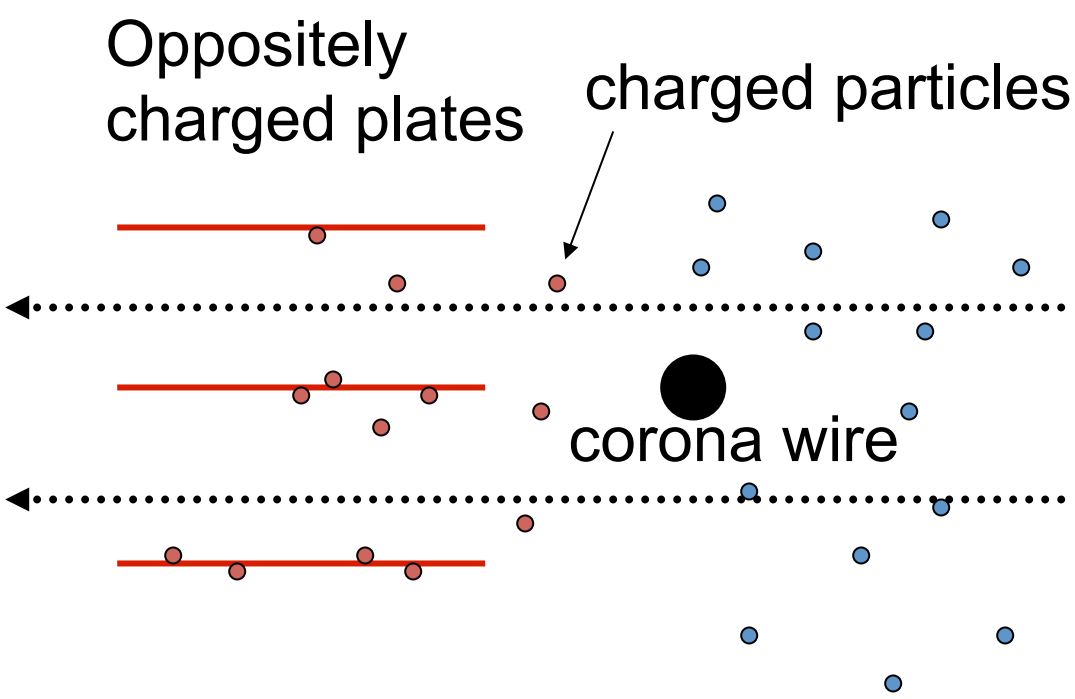
Recent testing of portable air cleaners



Waring et al., 2009 *Atmos Environ*

Ozone emissions for electronic air cleaners

- "Ion generating air cleaners" and electrostatic precipitators
  - Utilize high voltage to 'excite' oxygen (make singlet O out of O_2)
  - O_2 then forms with O to form O_3 (ozone)



Waring et al., 2009 *Atmos Environ*

Ozone emissions from electronic air cleaners

- Ozone generation rates

Ozone emission rates for ionizers tested in the first phase, as well as predicted ozone concentration increases, C^*, and equivalent outdoor ozone increases, ΔC_{out}, for a hypothetical residential 50 m^3 room and 392 m^3 home

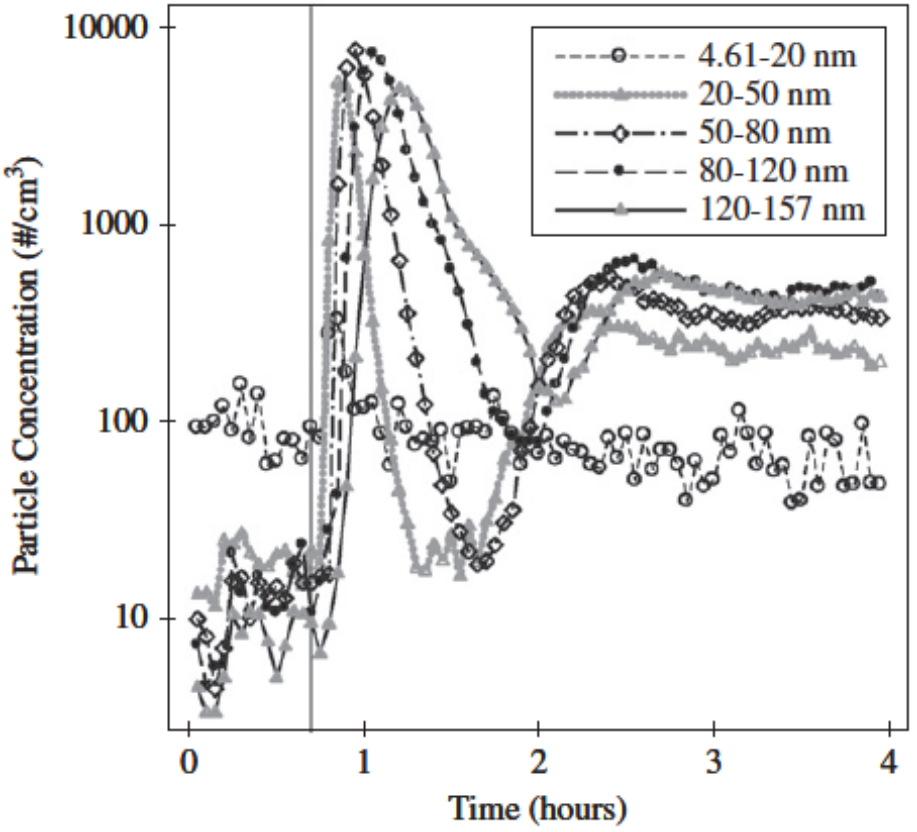
Air cleaner	Ozone emission rate ($mg\,h^{-1}$)	$V = 50\,m^3$		$V = 392\,m^3$	
		C^* (ppb)	ΔC_{out} (ppb)	C^* (ppb)	ΔC_{out} (ppb)
ESP	3.8±0.2	8.6	77	1.1	9.9
IG 1	3.3±0.2	7.5	67	1.0	8.6
IG 2	4.3±0.2	9.7	88	1.2	11

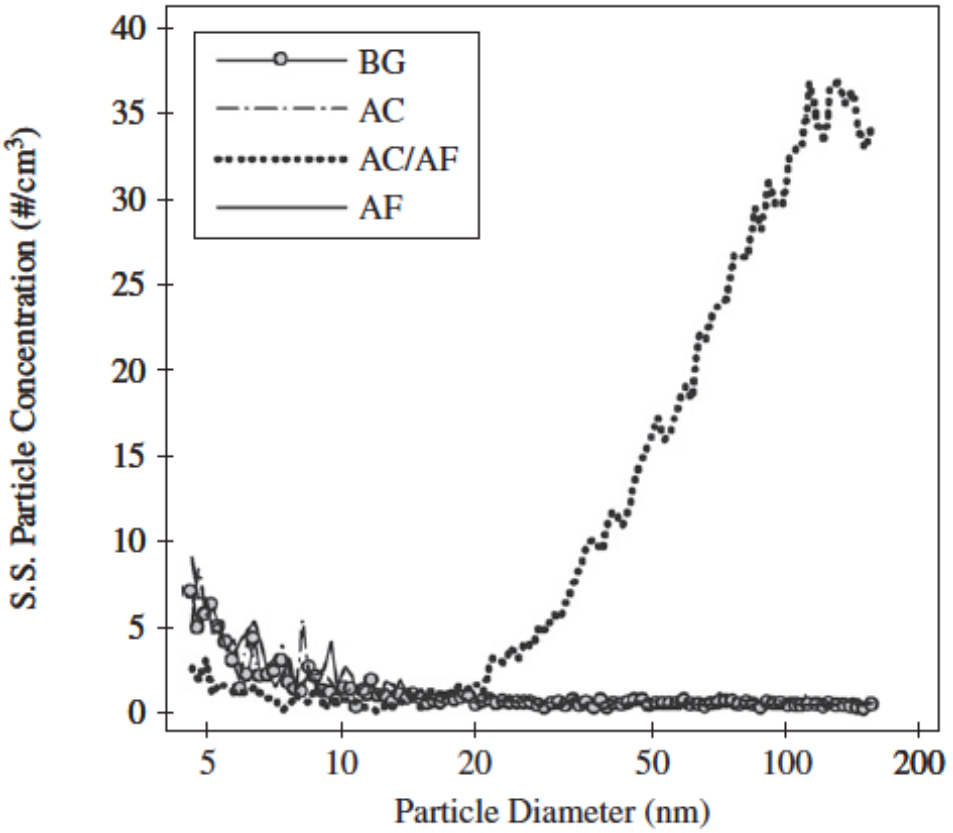
- Byproduct formation from reactions between ozone and terpene products
  - Formation products include SOA (secondary organic aerosols)
  - Can your particle-removing air cleaner actually lead to generation of particles??

Waring et al., 2009 *Atmos Environ*

Ozone emissions from electronic air cleaners and SOA

Operating an ozone generating air cleaner in the presence of terpene based products leads to formation of particles!





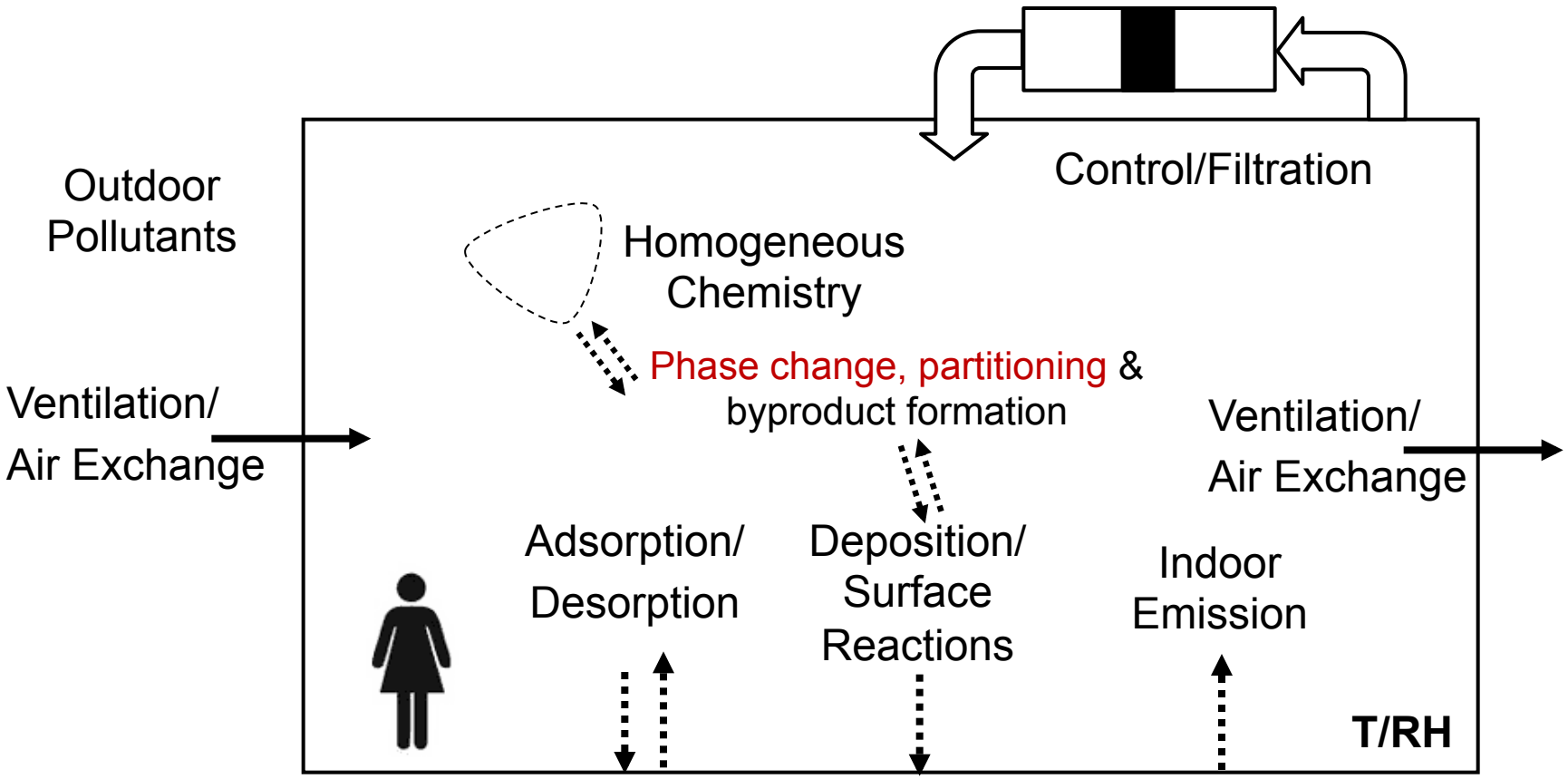
Waring et al., 2009 *Atmos Environ*

More resources on particle filtration

- ASHRAE Standard 52.2
- ASHRAE Technical Committee 2.4 Particulate Air Contaminants and Particulate Contaminant Removal Equipment
  - https://www.ashrae.org/standards-research--technology/technical-committees/section-2-0-environmental-quality/tc-2-4-particulate-air-contaminants-and-particulate-contaminant-removal-equipment
- National Air Filtration Association (NAFA)
  - http://www.nafahq.org
- EPA Guide to Air Cleaners
  - http://www.epa.gov/iaq/aircleaners/

SEMI-VOLATILE ORGANIC COMPOUNDS

Indoor environment: Mass balance



What are semi-volatile organic compounds?

- Semi-volatile organic compounds (SVOCs) are organic molecules that can have meaningful abundances in both the gas phase and condensed (particle) phases
  - Sometimes called particulate organic matter (POM)
  - Compounds with boiling points from 240 to 400°C
  - Compounds with saturation vapor pressures from 10^{-2} to 10^{-7} kPa

  Generally: as $p_{vap,sat}$ ↑ BP ↓ → More likely to be in gas phase than solid phase
- SVOCs are generally under-studied relative to VOCs and aerosols
  - Doesn't mean they're not important → largely due to analytical limitations
- We've already touched on some of these
  - e.g., polycyclic aromatic hydrocarbons (PAHs) originating from combustion
- SVOCs also occur as active ingredients in pesticides, cleaning agents, and personal care products
  - And as major additives in materials such as floor coverings, furnishings, and electronics components

Weschler and Nazaroff, 2008 *Atmospheric Environment*

What are semi-volatile organic compounds?

- Most SVOCs have a slow rate of release from sources

- Exposures can occur via inhalation
  - Both gases and SVOCs adsorbed onto particles
- Exposures can also occur via dermal and ingestion pathways

- Some are known to be toxic
  - Dioxins, benzo[a]pyrene, pentachlorophenol
    - Many have been removed from production over the years

- Others have emerging indicators of concern
  - More than 100 SVOCs have been found in the US population's blood in large biomonitoring studies

Some SVOCs of emerging concern

- Phthalate esters (BBzP, DEHP) (often used as plasticizers)
  - Allergic symptoms in children
  - Slowed male reproductive development
  - Altered semen quality

- Perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA)
  - Was a key ingredient in Scotchgard
  - Low birth weight, chronic kidney disease

- Polychlorinated biphenyl (PCBs), brominated flame retardants (BFRs), di-2-ethylhexyl phthalate (DEHP), bisphenol A, and some pesticides
  - Have been linked to endocrine disrupting activity
  - SVOCs that have chemical structures similar to those of human hormones and can either mimic or block endocrine (hormonal) activity
  - EDs may be important contributors to neurodevelopment and behavioral problems ranging from autism to attention deficit disorder
    - Incomplete and sometimes controversial evidence

SVOC classifications, sources, and potential health effects

SVOC Compounds	Uses	Sources	Potential Health Effects
Alkylphenols *Example:* *4-nonylphenol, 4-octylphenol*	Nonionic Surfactants	Detergents, Disinfectants, Surface Cleaners	May Interfere With, Mimic or Block Hormones
Organochlorines *Example:* *DDT, Chlordane*	Pesticides, Termiticide, Bactericide (Some Have Been Banned or Restricted in the 1980s)	Outdoor and Indoor Air, Tracked-In Dust, Disinfecting Products	Neurotoxicity, Effects on Developing Reproductive Systems And on Lactation, Cancer
Organophosphorus Compounds *Example:* *Tris(2-chloroethy)phosphate (TCEP), Tris(chloropropyl)phosphate (TCPP)*	Plasticizers, Antifoaming Agents, Flame Retardants, Pesticides	Polymeric Materials, Fabrics, Polyurethane Foams, Electronics (Cable Sheathing and Casings), Outdoor and Indoor Air, Dust	Effects on Neurodevelopment and Growth in Developing Tissue, Relate To Respiratory Disease in Children Through Dysregulation of the Autonomic Nervous System
Phthalates *Example:* *Di(2-ethylhexyl)-phthalate (DEHP), Di-iso-nonyl-phthalate (DINP)*	Plasticizers, Solvents, Fixing Fragrances (Use of DEHP And BBP Reduced Due to the Concern on Health Effects)	Flexible PVC, PVC Flooring, Wall Covering, Electrical Cable and Casings, Personal Care Products	Effects on the Development of Male Reproductive Tract, Prenatal Mortality, Reduced Growth and Birth Weight, May Relate to Asthma and Allergies in Children
Polybrominated Diphenyl Ethers (PBDEs) *Example:* *Hexabromodiphenyl ether (BDE-153), Tetrabromodiphenyl ether (BDE-47)*	Flame Retardants (Use of Penta- and Octa-BDEs Have Been Restricted)	Carpet Padding, Wall Coverings, Electronics (Casings), Furniture (Foam Cushioning and Mattress)	Effects on the Development of Brain And Nerve Tissues, Permanent Learning and Memory Impairment, Behavioral Changes, Delayed Puberty Onset, Fetal Malformations, Thyroid Hormone Disruption

Xu and Zhang **2011** *ASHRAE Journal*

SVOC classifications, sources, and potential health effects

SVOC Compounds	Uses	Sources	Potential Health Effects
PolychlorinatedBiphenyls (PCBs) *Example: 2,2',5,5'-tetrachloro-1,1'-biphenyl (PCB 52), 2,2',4,4',5,5'-hexachloro-1,1'-biphenyl (PCB 153)*	Heat Transfer Fluids, Stabilizers, Flame Retardants, (Have Been Banned or Restricted in the 1970s)	Floor Finishes, Foam Cushioning and Mattresses, Oil-Filled Transformers, Capacitors	Developmental Neurotoxicants, Effects on Immune, Reproductive, Nervous, and Endocrine Systems, Cancer (Including Breast Cancer)
Polycyclic Aromatic Hydrocarbons (PAHs) *Example: Benzo(a)pyrene, Pyrene*	Combustion Byproducts	Outdoor Air, Cooking, Smoking	Cataracts, Kidney and Liver Damage, Jaundice, Increased Risk Of Skin, Lung, Bladder, and Gastrointestinal Cancers
Pyrethroids *Example: Cyfluthrin, Permethrin*	Insecticides	Outdoor and Indoor Air, Tracked-In Dust, Cleaning Products	Weak Anti-Androgenic, Anti-Estrogenic, or Estrogenic Effect
Parabens *Example: Butyl paraben, Methyl paraben*	Bactericides, Antimicrobial Agents, Preservatives	Personal Care Products, Canned Food, Fabrics	Weak Environmental Estrogens

Xu and Zhang **2011** *ASHRAE Journal*

Semi-volatile organic compounds found indoors

Table 1
Selected semivolatile organic compounds observed or expected in indoor environments, organized by product class and chemical class, with examples.

Chemical class	Specific chemical	CAS No.	Formula	log P_s[a]
Biocides and preservatives				
Antimicrobials	Triclosan	3380-34-5	$C_{12}H_7Cl_3O_2$	−8.9
Antioxidants	Butylated hydroxytoluene (BHT)	128-37-0	$C_{15}H_{24}O$	−6.7
Fungicides	Tributyltin oxide (TBTO)	56-35-9	$C_{24}H_{54}OSn_2$	−10.9
Wood preservatives	Pentachlorophenol (PCP)	87-86-5	C_6HCl_5O	−7.4
Combustion byproducts				
Environmental tobacco smoke	Nicotine	54-11-5	$C_{10}H_{14}N_2$	−4.7
Polychlorinated dibenzo-*p*-dioxins	2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD)	1746-01-6	$C_{12}H_4Cl_4O_2$	−11.4
Polycyclic aromatic hydrocarbons	Benzo[*a*]pyrene (B*a*P)	50-32-8	$C_{20}H_{12}$	−10.5
Polycyclic aromatic hydrocarbons	Phenanthrene	85-01-8	$C_{14}H_{10}$	−6.6
Polycyclic aromatic hydrocarbons	Pyrene	129-00-0	$C_{16}H_{10}$	−7.5
Degradation products/residual monomers				
Phenols	Bisphenol A	80-05-7	$C_{15}H_{16}O_2$	−10.5
Flame retardants				
Brominated flame retardants	2,2′,4,4′,5,5′-Hexabromodiphenyl ether (BDE-153)	68631-49-2	$C_{12}H_4Br_6O$	−13.8
Brominated flame retardants	2,2′,4,4′,5-Pentabromodiphenyl ether (BDE-99)	60348-60-9	$C_{12}H_5Br_5O$	−12.0
Brominated flame retardants	2,2′,4,4′-Tetrabromodiphenyl ether (BDE-47)	5436-43-1	$C_{12}H_6Br_4O$	−10.5
Chlorinated flame retardants	Perchloropentacyclodecane (mirex)	2385-85-5	$C_{10}Cl_{12}$	−10.6
Phosphate esters	Tris(chloropropyl) phosphate	13674-84-5	$C_9H_{18}Cl_3O_4P$	−6.3
Heat-transfer fluids				
Polychlorinated biphenyls (PCBs)	2,2′,5,5′-tetrachloro-1,1′-biphenyl (PCB 52)	35693-99-3	$C_{12}H_6Cl_4$	−7.8
Polychlorinated biphenyls (PCBs)	2,2′,4,4′,5,5′-hexachloro-1,1′-biphenyl (PCB 153)	35065-27-1	$C_{12}H_4Cl_6$	−9.8
Microbial emissions				
Sesquiterpenes	Geosmin	23333-91-7	$C_{12}H_{22}O$	−5.3
Personal care products				
Musk compounds	Galaxolide	1222-05-5	$C_{18}H_{26}O$	−7.5
Petrolatum constituents	*n*-Pentacosane	629-99-2	$C_{25}H_{52}$	−10.2

Weschler and Nazaroff **2008** *Atmos Environ*

Semi-volatile organic compounds found indoors

Table 1
Selected semivolatile organic compounds observed or expected in indoor environments, organized by product class and chemical class, with examples.

Chemical class	Specific chemical	CAS No.	Formula	log P_s^a
Pesticides/termiticides/herbicides				
Carbamates	Propoxur	114-26-1	$C_{11}H_{15}NO_3$	−6.8
Organochlorine pesticides	Chlordane	57-74-9	$C_{10}H_6Cl_8$	−7.8
Organochlorine pesticides	*p,p′*-DDT	50-29-3	$C_{14}H_9Cl_5$	−9.7
Organophosphate pesticides	Chlorpyrifos	2921-88-2	$C_9H_{11}Cl_3NO_3PS$	−7.9
Organophosphate pesticides	Diazinon	333-41-5	$C_{12}H_{21}N_2O_3PS$	−8.0
Organophosphate pesticides	Methyl parathion	298-00-0	$C_8H_{10}NO_5PS$	−6.6
Pyrethroids	Cyfluthrin	68359-37-5	$C_{22}H_{18}Cl_2FNO_3$	−12.4
Pyrethroids	Cypermethrin	52315-07-8	$C_{22}H_{19}Cl_2NO_3$	−12.4
Pyrethroids	Permethrin	52645-53-1	$C_{21}H_{20}Cl_2O_3$	−10.7
Synergist	Piperonyl butoxide	51-03-6	$C_{19}H_{30}O_5$	−10.1
Plasticizers				
Adipate esters	Di(2-ethylhexyl) adipate (DEHA)	103-23-1	$C_{22}H_{42}O_4$	−9.9
Phosphate esters	Triphenylphosphate (TPP)	115-86-6	$C_{18}H_{15}O_4P$	−9.2
Phthalate esters	Butylbenzyl phthalate (BBzP)	85-68-7	$C_{19}H_{20}O_4$	−10.0
Phthalate esters	Dibutyl phthalate (DBP)	84-74-2	$C_{16}H_{22}O_4$	−8.0
Phthalate esters	Di(2-ethylhexyl) phthalate (DEHP)	117-81-7	$C_{24}H_{38}O_4$	−11.5
Sealants				
Silicones	Tetradecamethylcycloheptasiloxane (D7)	107-50-6	$C_{14}H_{42}O_7Si_7$	–
Stain repellents, oil and water repellents				
Perfluorinated surfactants	*N*-ethyl perfluorooctane sulfonamidoethanol (EtFOSE)	1691-99-2	$C_{12}H_{10}F_{17}NO_3S$	−6.8
Perfluorinated surfactants	*N*-methylperfluorooctane sulfonamidoethanol (MeFOSE)	24448-09-7	$C_{11}H_8F_{17}NO_3S$	−6.4
Surfactants (nonionic), emulsifiers, coalescing agents				
Alkylphenol ethoxylates	4-Nonylphenol	104-40-5	$C_{15}H_{24}O$	−7.1
Coalescing agents	3-Hydroxy-2,2,4-Trimethylpentyl-1-Isobutyrate (Texanol)	25625-77-4	$C_{12}H_{24}O_3$	−5.6
Terpene oxidation products				
	Pinonaldehyde	2704-78-1	$C_{10}H_{16}O_2$	−4.1
Water disinfection products				
	3-Chloro-4-(dichloromethyl)-5-hydroxy-2(5H)-furanone (MX)	77439-76-0	$C_5H_3Cl_3O_3$	−9.3
Waxes, polishes and essential oils				
Fatty acids	Stearic acid (octadecanoic acid)	57-11-4	$C_{18}H_{36}O_2$	−11.0
Fatty acids	Linoleic acid	60-33-3	$C_{18}H_{32}O_2$	−10.2
Sesquiterpenes	Caryophyllene	87-44-5	$C_{15}H_{24}$	−4.6

Weschler and Nazaroff **2008** *Atmos Environ*

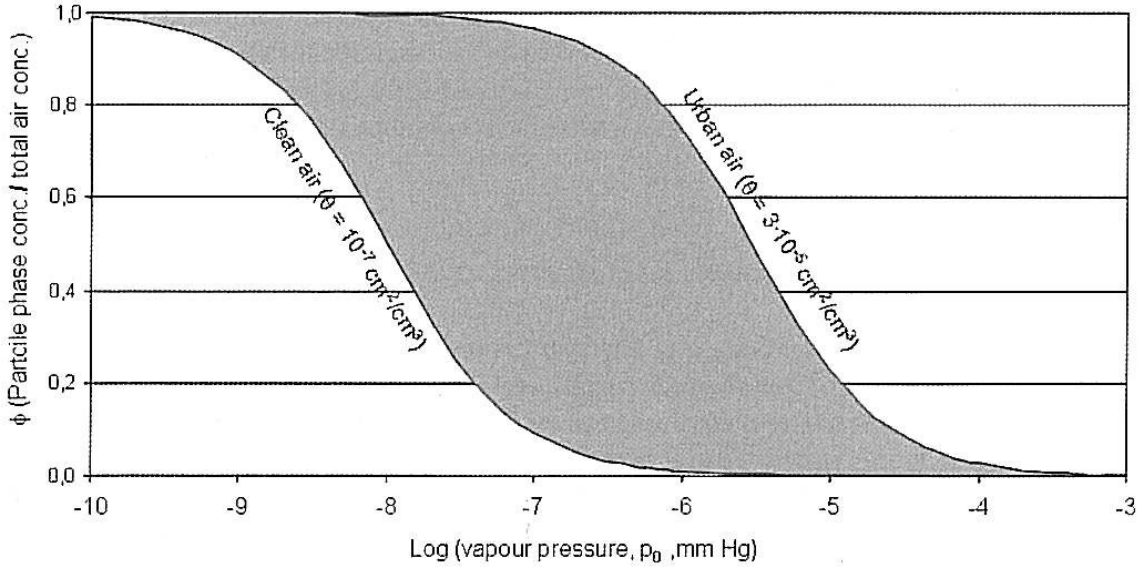
SVOC 'partitioning'

SVOCs can exist in both gas and particle phases

$$\phi = \frac{\text{particle phase concentration}}{\text{total gas + particle phase concentration}} = \frac{k * SA_{particles}}{p_{vap,sat} + k * SA_{particles}}$$

ϕ = concentration of a compound in the particle phase relative to the total air concentration (gas + particle) [dimensionless]
k = constant that depends on MW of compound
$SA_{particles}$ = aerosol surface area per volume [cm^2/cm^3]
$p_{vap,sat}$ = saturation vapor pressure of compound (mm Hg)

Generally: as VP ↑ BP ↓ → More likely to be in gas phase than solid phase



Organic gases: VOCs

- VOCs, VVOCs, SVOCs, and POM are all categorized by their boiling points
  - Lower molecular weight (and low boiling point) compounds are more likely in the gas-phase

Table 1. Classification of indoor organic pollutants

Category	Description	Abbreviation	Boiling-point range (°C)[a]	Sampling methods typically used in field studies
1	Very volatile (gaseous) organic compounds	VVOC	<0 to 50–100	Batch sampling; adsorption on charcoal
2	Volatile organic compounds	VOC	50–100 to 240–260	Adsorption on Tenax, carbon molecular black or charcoal
3	Semivolatile organic compounds	SVOC	240–260 to 380–400	Adsorption on polyurethane foam or XAD-2
4	Organic compounds associated with particulate matter or particulate organic matter	POM	>380	Collection on filters

[a] Polar compounds appear at the higher end of the range.

SVOC 'partitioning'

We can also describe particle/gas partitioning as a function of the total aerosol mass concentration suspended in the air:

$$K_p = \frac{\left(F / TSP\right)}{c_g} = \frac{c_p}{c_g}$$

K_p = thermodynamic particle-gas partition coefficient [$m^3/\mu g$]
F = equilibrium particle concentration of a compound [ng/m^3]
TSP = concentration of total suspended particles [$\mu g/m^3$]
c_g = equilibrium gas phase concentration (ng/m^3)
c_p = concentration within the particle phase (ng/m^3)

$$\frac{F}{c_g} = K_p(TSP)$$

How do we get K_p?

Remember: as VP ↑ BP ↓ → More likely to be in gas phase than solid phase

Weschler **2003** *Atmos Environ*

SVOC ‘partitioning’

- Ratio between organic compound’s particle phase concentration and its gas phase concentration:

$$\frac{F}{c_g} = K_p(TSP)$$

K_p = thermodynamic particle-gas partition coefficient [$m^3/\mu g$]
F = equilibrium particle concentration of a compound [ng/m^3]
TSP = concentration of total suspended particles [$\mu g/m^3$]
c_g = equilibrium gas phase concentration (ng/m^3)

log(K_p) is higher for lower log($p_{vap,sat}$)

K_p is therefore higher for lower $p_{vap,sat}$

Higher K_p means greater fraction F in the particle phase

Lower vapor pressure more likely to be in solid phase… makes sense, right?

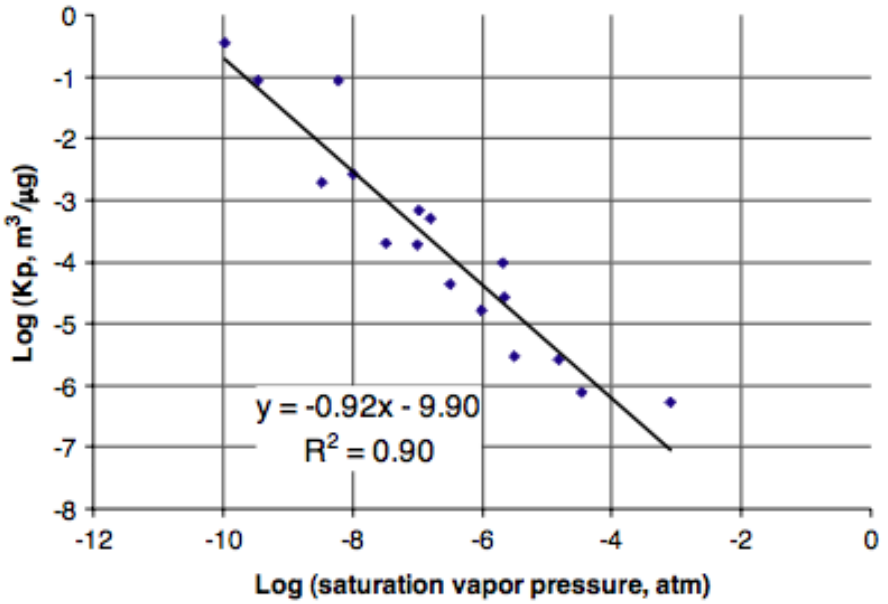


Fig. 1. Log(K_p) versus log (saturation vapor pressure at 31°C) for a series on *n*-alkanes and PAHs sorbed to particles generated from gasoline vapors. Data taken from Liang et al. (1997).

Weschler **2003** *Atmos Environ*

SVOC 'partitioning'

- Rule of thumb: higher MW compounds will have lower vapor pressures and thus be more likely to be in the particle phase (higher K_p, F, and c_p)

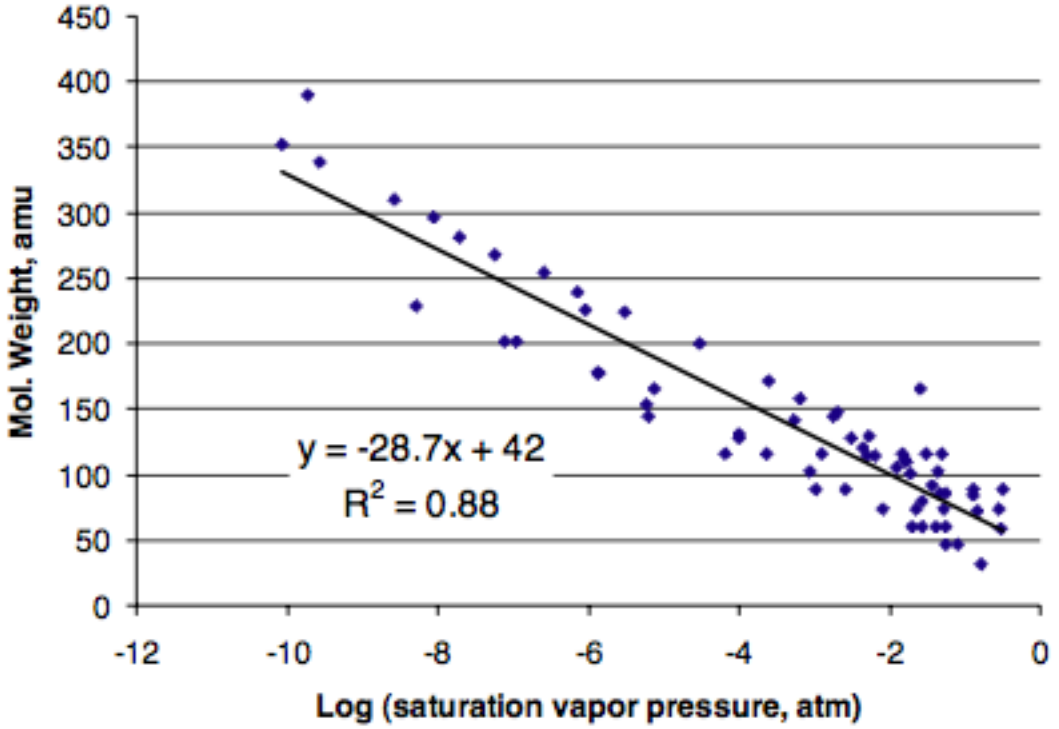


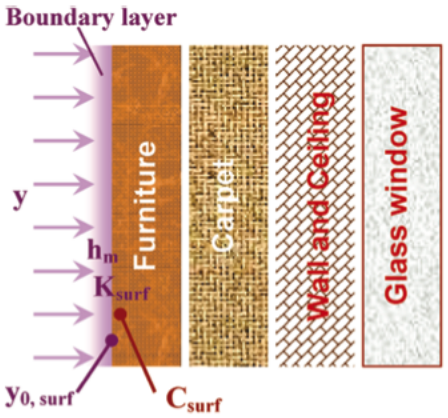
Fig. 6. Log (molecular weight, amu) versus log (saturation vapor pressure at 25°C) for compounds plotted in Figs. 1–3 and 5, as well as DEHP.

Weschler **2003** *Atmos Environ*

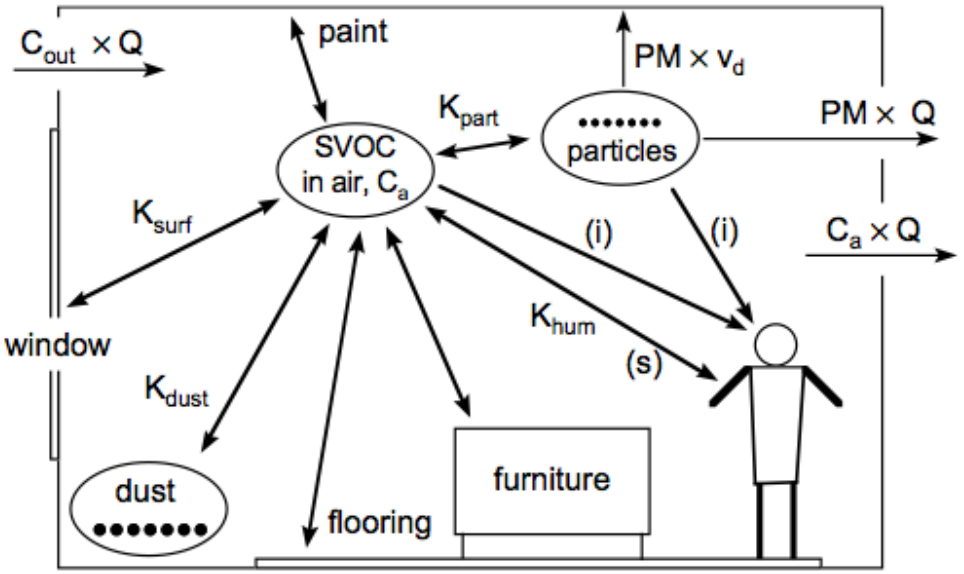
SVOCs in indoor environments

Mass balance. SVOCs emitted from indoor materials exist as:

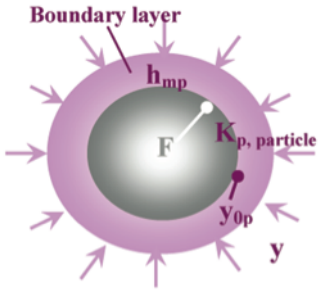
- Gases
- Attached to particles
- Adsorbed to surfaces



a. Adsorption to interior surfaces



(i) - inhalation intake; (s) - skin permeation



b. Adsorption to airborne particles

Fig. 3. Schematic illustration of some key aspects of indoor SVOC dynamics. The figure emphasizes the partitioning of an SVOC between the gas phase and different indoor sorptive compartments (airborne particles, settled dust, fixed surfaces, and human surfaces). Also shown are SVOC exchanges with outdoor air associated with ventilation. Important, but not illustrated, are emissions from indoor sources.

Weschler and Nazaroff **2008** *Atmos Environ*; Xu et al. **2009** *Environ Sci Technol*

Predicted gas, particle, and surface concentrations of different SVOCs

Distribution of selected organic compounds between the gas phase and the surfaces of airborne particles, a carpet and walls within a typical room

Compound	Mol. weight (amu)	Vapor pressure at 25°C (atm)	Assumed gas phase concentration ($\mu g\,m^{-3}$)	Mass in gas phase (μg)	Mass on particles (μg)	Mass on carpet (μg)	Mass on walls (μg)
MTBE	88	3.2E−01	10	400	2.3E−5	17	19
Toluene	92	3.7E−02	10	400	1.4E−4	100	70
Ethylbenzene	106	1.3E−02	10	400	3.6E−4	260	140
Propylbenzene	120	4.5E−03	10	400	8.9E−4	610	260
Naphthalene	128	1.0E−04	5	200	1.2E−2	7400	1390
Acenaphthene	154	5.9E−06	5	200	0.13	8.0E+4	8000
Hexadecane	226	9.1E−07	5	200	0.66	3.8E+5	2.6E+4
Phenanthrene	178	1.4E−06	1	40	0.093	5.4E+4	4000
Octadecane	254	2.5E−07	1	40	0.40	2.3E+5	1.1E+4
Pyrene	202	7.6E−08	1	40	1.1	6.2E+5	2.4E+4
Heneicosane	296	8.7E−09	0.5	20	3.6	1.9E+6	4.6E+4
Chrysene	228	5.0E−09	0.5	20	5.8	3.0E+6	6.4E+4
Tetracosane	338	2.8E−10	0.01	0.4	1.4	6.9E+5	7800
DEHP	390	1.9E−10	0.07	3.0	14	6.7E+6	6.9E+4
Pentacosane	352	8.7E−11	0.01	0.4	3.8	1.8E+6	1.6E+4

Values derived for a $3 \times 3.65 \times 3.65\,m^3$ room containing $20\,\mu g\,m^{-3}$ of airborne particles (TSP), a $10\,m^2$ carpet with pad, and painted gypsum board walls. See text for further details.

Weschler **2003** *Atmos Environ*

What are typical indoor SVOC concentrations?

Indoor concentrations and body burden of selected semivolatile organic compounds.

Chemical	Typical reported concentrations in indoor environments				US body burdens (95%ile)[a] – blood (ng g^{-1} serum); urine (μg g^{-1} creatinine)
	Air (ng m^{-3})	References	Dust (μg g^{-1})	References	
Biocides and preservatives					
Triclosan	–	–	0.2–2	Canosa et al., 2007	360 (urine)[b]
Tributyltin oxide (TBTO)	–	–	0.01–0.1	Fromme et al., 2005	–
Pentachlorophenol (PCP)	0.4–4	Rudel et al., 2003; Morgan et al., 2004	0.2–2	Rudel et al., 2003	2.3 (urine)
Combustion byproducts					
Nicotine	200–2000	Leaderer and Hammond, 1991; Gehring et al., 2006	10–100	Hein et al., 1991; Matt et al., 2004	2.2 (blood)[c]
Benzo[*a*]pyrene (BaP)	0.02–0.2	Naumova et al., 2002; Morgan et al., 2004	0.2–2	Rudel et al., 2003; Mannino and Orecchio, 2008	0.18 (urine)
Phenanthrene	10–100	Naumova et al., 2002	0.2–2	Mannino and Orecchio, 2008	1.7 (urine)[d]
Pyrene	1–10	Naumova et al., 2002; Rudel et al., 2003	0.2–2	Mannino and Orecchio, 2008	0.24 (urine)
Degradation products/residual monomers					
Bisphenol A	0.5–5	Morgan et al., 2004	0.2–2	Rudel et al., 2003	11 (urine)[e]
Flame retardants					
2,2′,4,4′,5,5′-Hexabromodiphenyl ether (BDE-153, hexa BDE)	0.002–0.02	Wilford et al., 2004; Shoeib et al., 2004; Allen et al., 2007	0.03–0.3	Stapleton et al., 2005; Wilford et al., 2005; Wu et al., 2007	0.44 (blood)[f]
2,2′,4,4′,5-Pentabromodiphenyl ether (BDE-99, pentaBDE)	0.03–0.3	Wilford et al., 2004; Shoeib et al., 2004; Allen et al., 2007	0.4–4	Rudel et al., 2003; Stapleton et al., 2005; Wilford et al., 2005; Wu et al., 2007	0.28 (blood)[f]
2,2′,4,4′-Tetrabromodiphenyl ether (BDE-47, tetra BDE)	0.06–0.6	Wilford et al., 2004; Shoeib et al., 2004; Allen et al., 2007	0.3–3	Stapleton et al., 2005; Wilford et al., 2005; Wu et al., 2007	1.1 (blood)[f]
Perchloropentacyclodecane (Mirex)	–	–	–	–	0.41 (blood)
Tris(chloropropyl) phosphate	6–60	Wensing et al., 2005	0.3–3	Wensing et al., 2005	–

Weschler and Nazaroff **2008** *Atmos Environ*

What are typical indoor SVOC concentrations?

Indoor concentrations and body burden of selected semivolatile organic compounds.

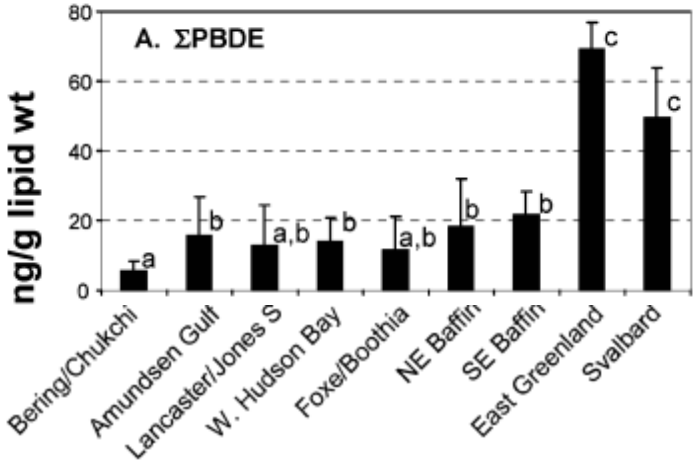
Chemical	Typical reported concentrations in indoor environments				US body burdens (95%ile)[a] – blood (ng g^{-1} serum); urine (µg g^{-1} creatinine)
	Air (ng m^{-3})	References	Dust (µg g^{-1})	References	
Personal care products					
Galaxolide	25–250	Fromme et al., 2004	0.5–5	Fromme et al., 2004	–
Pesticides/termiticides/herbicides					
Propoxur	0.8–8	Rudel et al., 2003	0.05–0.5	Rudel et al., 2003	<1 (urine)
Chlordane	0.5–5	Morgan et al., 2004; Offenberg et al., 2004	0.04–0.4	Rudel et al., 2003	0.35 (blood)
p,p′-DDT	0.2–2	Rudel et al., 2003	0.1–1	Rudel et al., 2003	0.18 (blood)
Chlorpyrifos	1–10	Morgan et al., 2004	0.08–0.8	Julien et al., 2008; Morgan et al., 2004	9.2 (urine)
Diazinon	1–5	Morgan et al., 2004	0.02–0.2	Julien et al., 2008	<1 (urine)
Methyl parathion	0.05–0.5	Rudel et al., 2003	0.01–0.1	Rudel et al., 2003	2.9 (urine)
Cyfluthrin	0.1–1.0	Morgan et al., 2004	0.08–0.8	Julien et al., 2008; Morgan et al., 2004	Common metabolite: 2.6 (urine)
Cypermethrin	–	–	0.08–0.8	Julien et al., 2008; Rudel et al., 2003	
Permethrin	0.1–0.7	Rudel et al., 2003; Morgan et al., 2004	0.2–2	Rudel et al., 2003; Julien et al., 2008	3.8 (urine)
Piperonyl butoxide	0.1–1.0	Rudel et al., 2003	0.1–1.0	Rudel et al., 2003	–
Plasticizers					
Di(2-ethylhexyl) adipate (DEHA)	5–15	Rudel et al., 2003	2–10	Rudel et al., 2003	–
Triphenylphosphate (TPP)	0.1–1	Wensing et al., 2005	2–20	Wensing et al., 2005	–

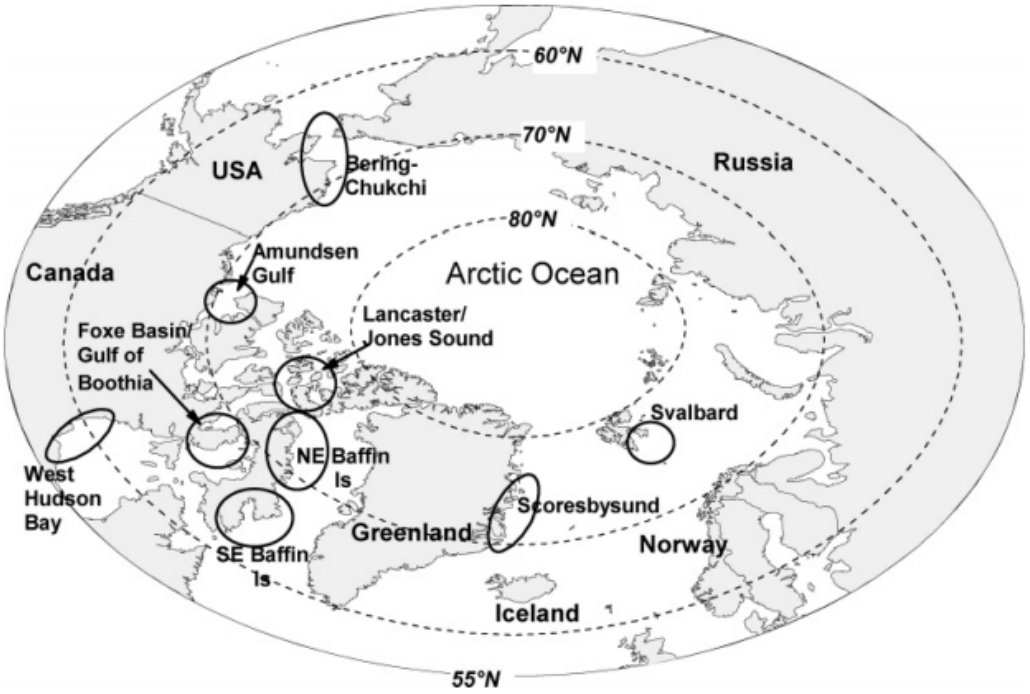
Weschler and Nazaroff **2008** *Atmos Environ*

Indoor and outdoor connections

Brominated Flame Retardants in Polar Bears (*Ursus maritimus*) from Alaska, the Canadian Arctic East Greenland, and Svalbard

Muir et al., **2006** *Environ Sci Technol*





- What do flame retardants in polar bears have to do with indoor air pollution??

Indoor and outdoor connections

Indoor Air Is a Significant Source of Tri-decabrominated Diphenyl Ethers to Outdoor Air via Ventilation Systems

Björklund et al., **2012** *Environ Sci Technol*

Table 4. Estimated Emissions of PentaBDE to Outdoor Air in Sweden (See SI for Full Description of Calculations and References)

source	emission factor	activity (kg/year)	annual emission (kg/year)	comment
metals manufacturing	35–716 μg/tonne product	1.7×10^9	0.06–1	concerns the sum of 20 congeners (di-octaBDEs), with BDE-47 and -99 being the most predominant
municipal incineration	no information	0.8–18	not possible to estimate	
electronics recycling	no information	9×10^4–5.6×10^5	not possible to estimate	
e-waste fires	8.4–50.2 μg/kg burnt material, assuming no extinguishing water	1.48×10^6	0.01–0.07	concerns sum of BDEs (47,85,99,100,138,153,154). nondetected congeners were assigned a value of 0 (d.l. = 1.5 μg/kg burnt)
landfill fires	4.96 – 394 μg/kg C burned	7×10^4–7×10^5	3.5×10^{-4}–0.028	concerns BDE-47 only
indoor environment - households	*10–260 pg/m³*	*1.7×10^{12}–9.4×10^{12} m³/year*	*0.024–0.92*	*concerns BDE-28, -47, -99, -153*
indoor environment–public buildings	*84–1600 pg/m³*	*2.7×10^{12}– 8.7×10^{12} m³/year*	*0.26–8.7*	*concerns BDE-28, -47, -99, -153*
total			**0.35–11**	
percentage total contribution of indoor air			81–82	

For one of the first times we're aware of, indoor air pollution in modern countries is linked strongly to outdoor air pollution in remote regions of the world!

- Potential effects go beyond human beings

ADVERSE HEALTH EFFECTS AND AIR POLLUTION

Adverse health effects

- 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

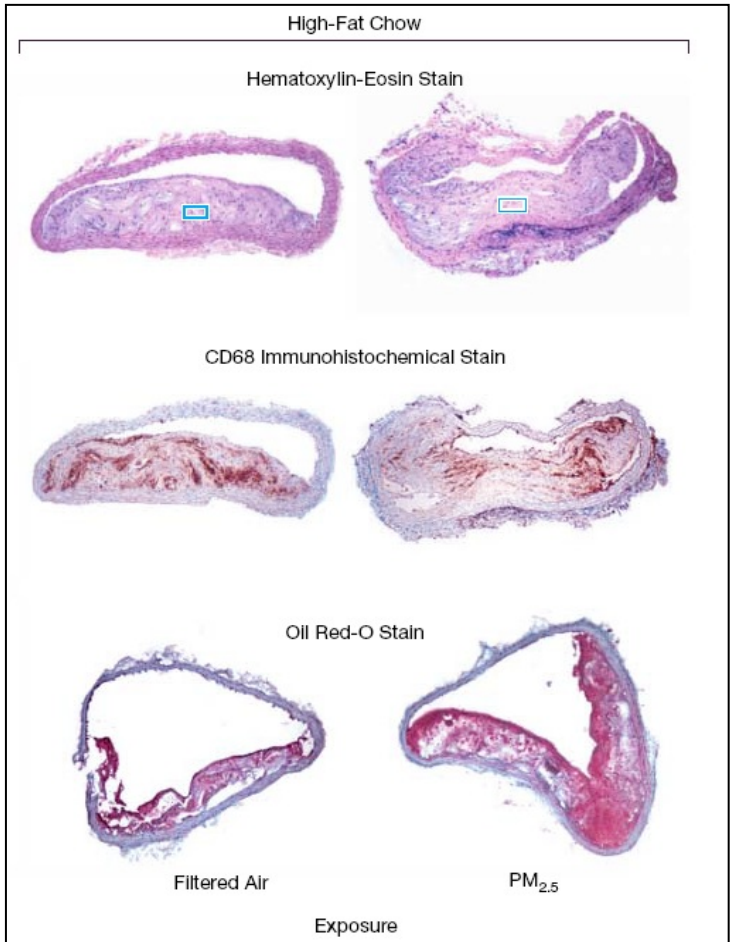
How do air pollutants cause health effects?

- PM or ozone induce airway inflammation
- Oxidative stress is induced by transition metals or PAHs
- Modifications of intracellular proteins/enzymes
- 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



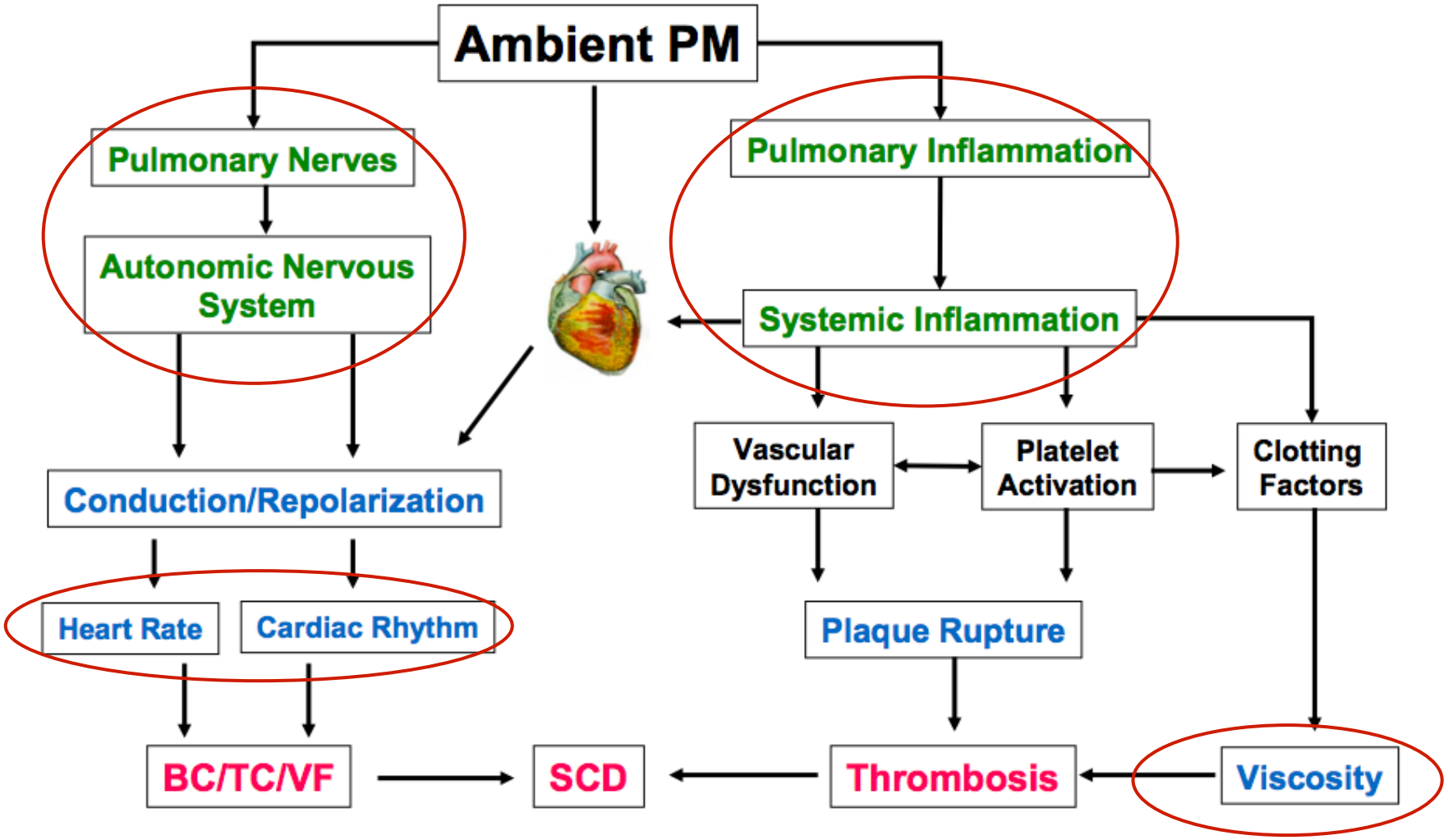
Sun et al. *JAMA,* 2005; Kunzli et al. 2005

How does PM cause health effects?

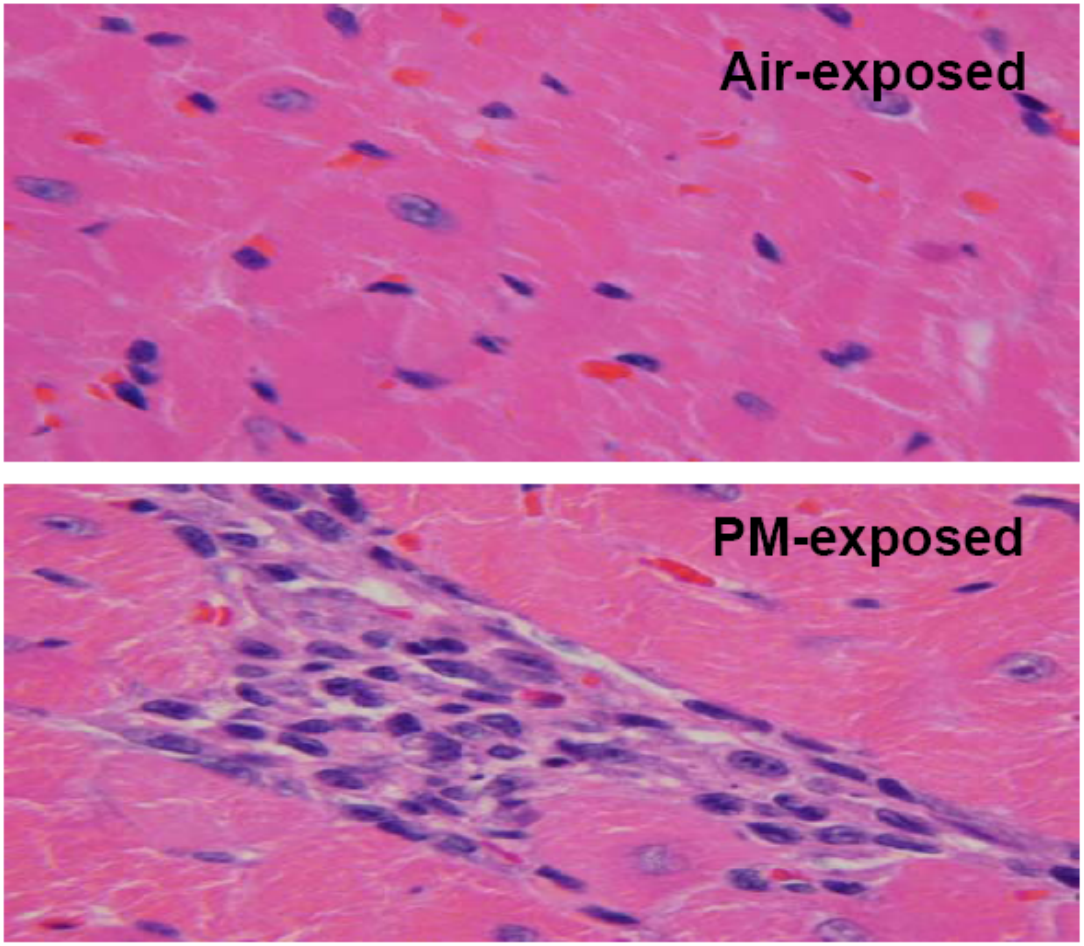
- Several theories exist here… likely more than one mechanism

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

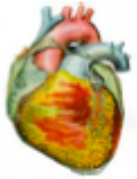


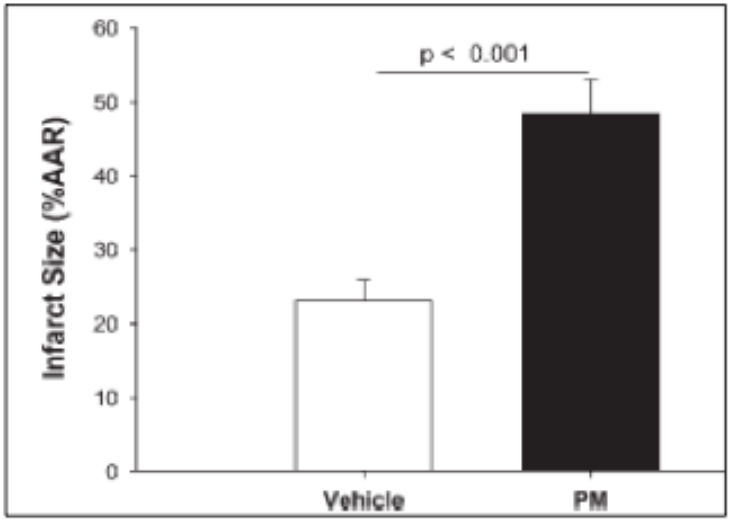
Rats exposed to ambient PM one day per week for 16 weeks

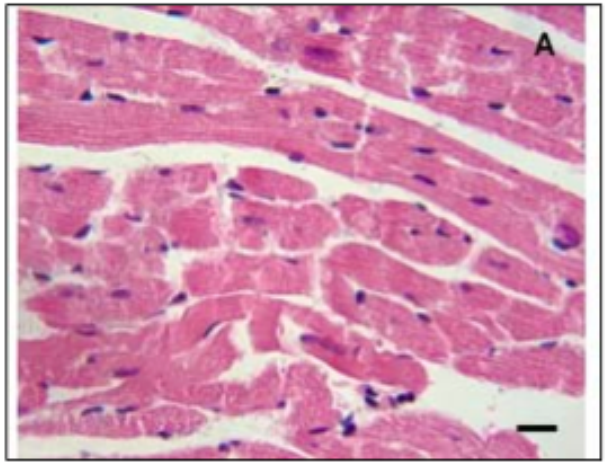
Kodavanti et al., 2003

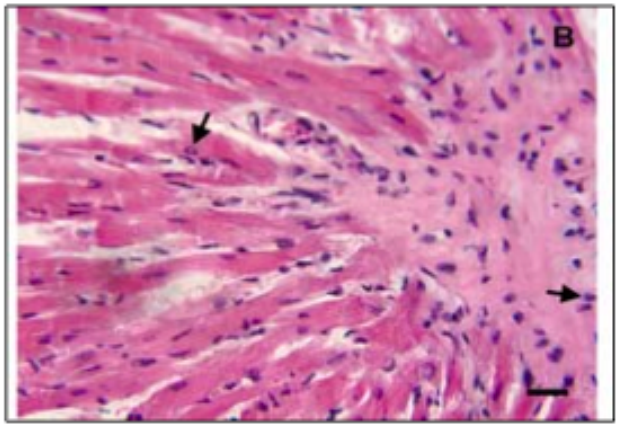
PM causes injury to cardiac cells

Ambient PM



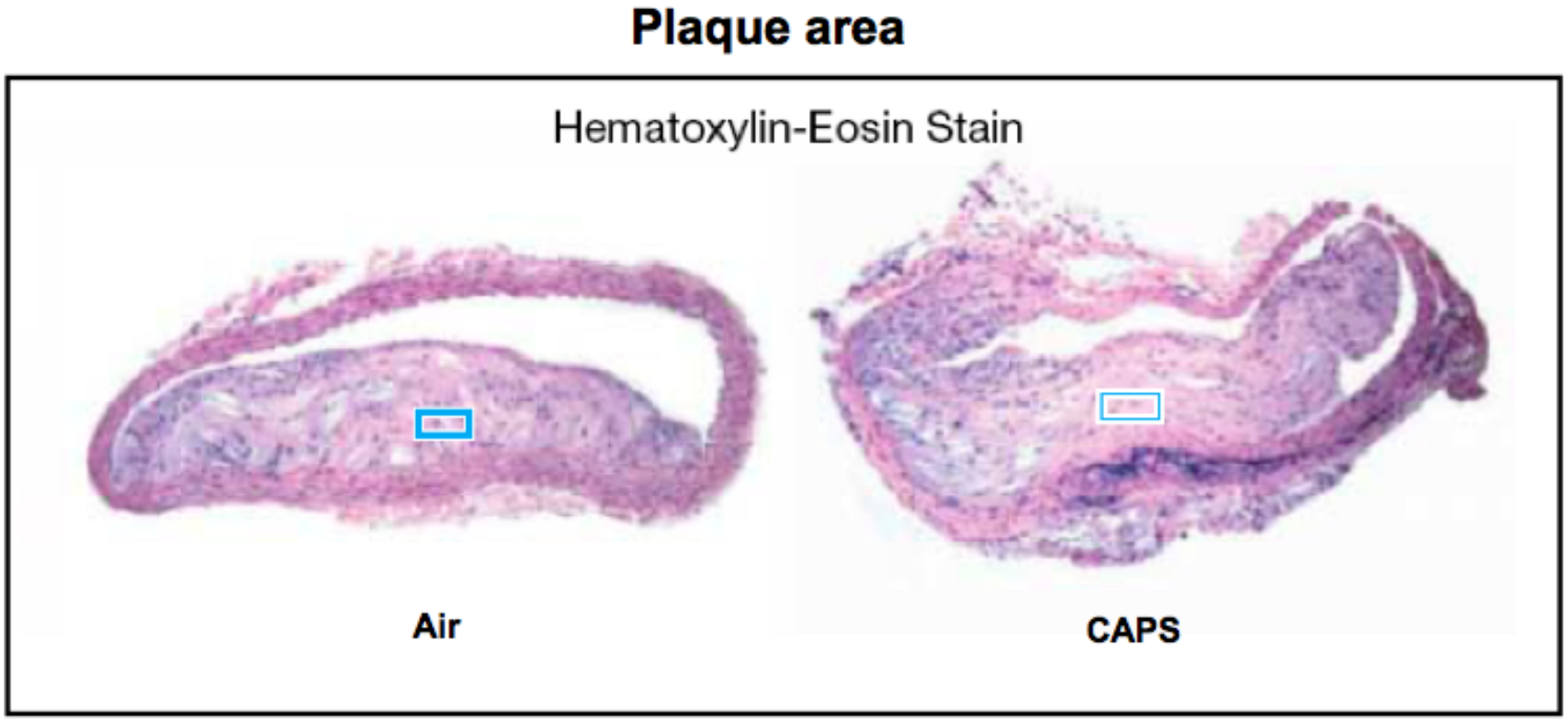






Cozzi et al., 2007

PM hardens arteries

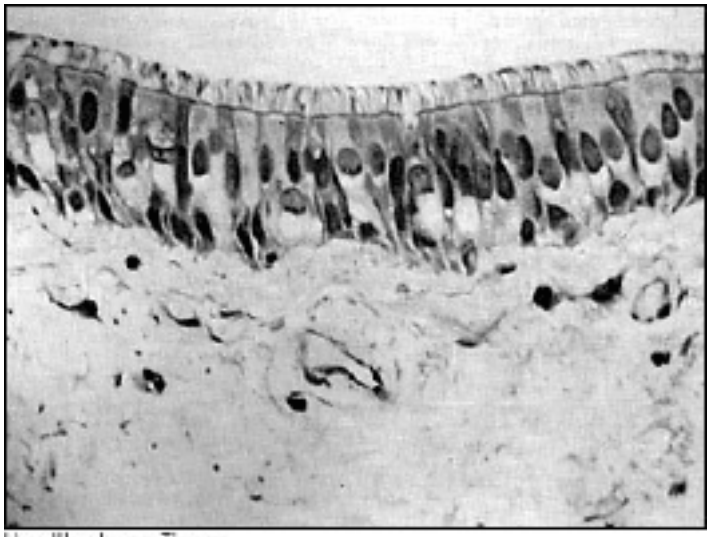


ApoE mice exposed for 6 hrs/day, 5 days/wk x 6 months to CAPS (85 μg/m³ average)
Mean levels only 15.2 μg/m³

Sun et al. 2005

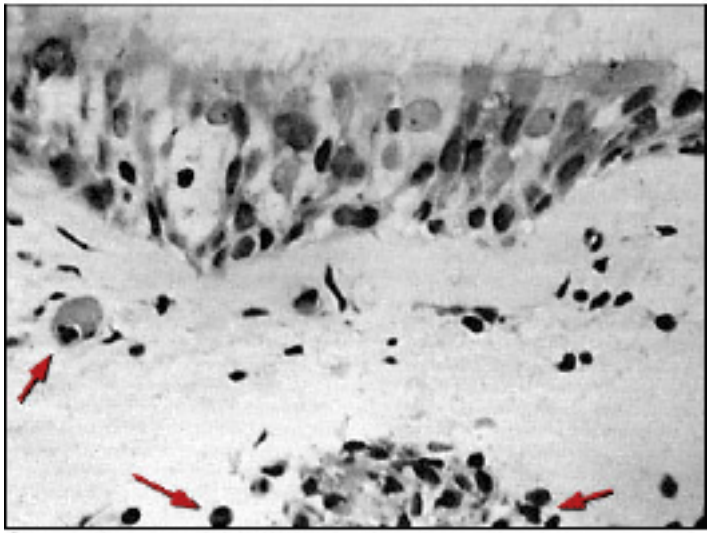
Ozone damages lung tissue

- Tiny cilia that clear the lungs from mucus appear along the top of the image to the right (healthy lung tissue)



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



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, 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

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

  - RR > 1.0 = association
  - RR >> 1.0 = strong association

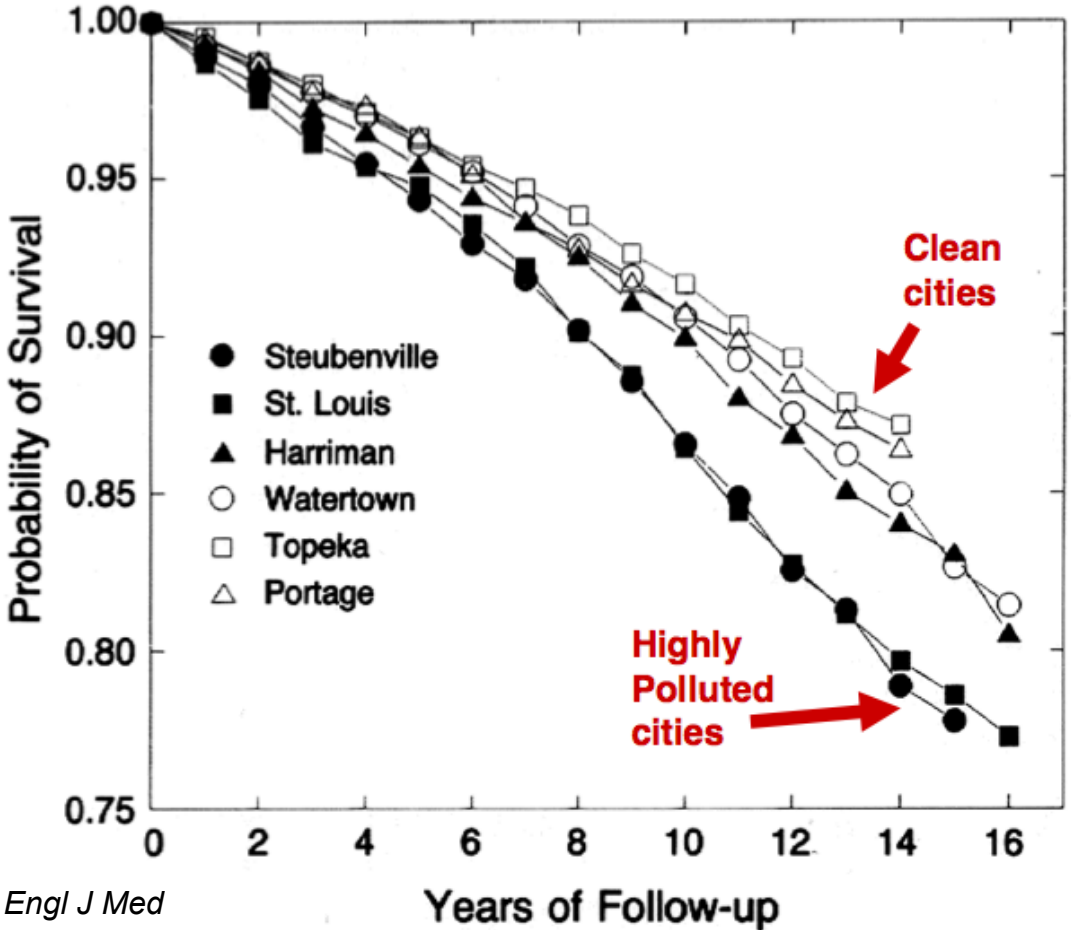
- Odds ratio = OR (often ~RR)

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

  - OR > 1.0 = association
  - OR >> 1.0 = strong association

Health effects of outdoor PM: Epidemiology

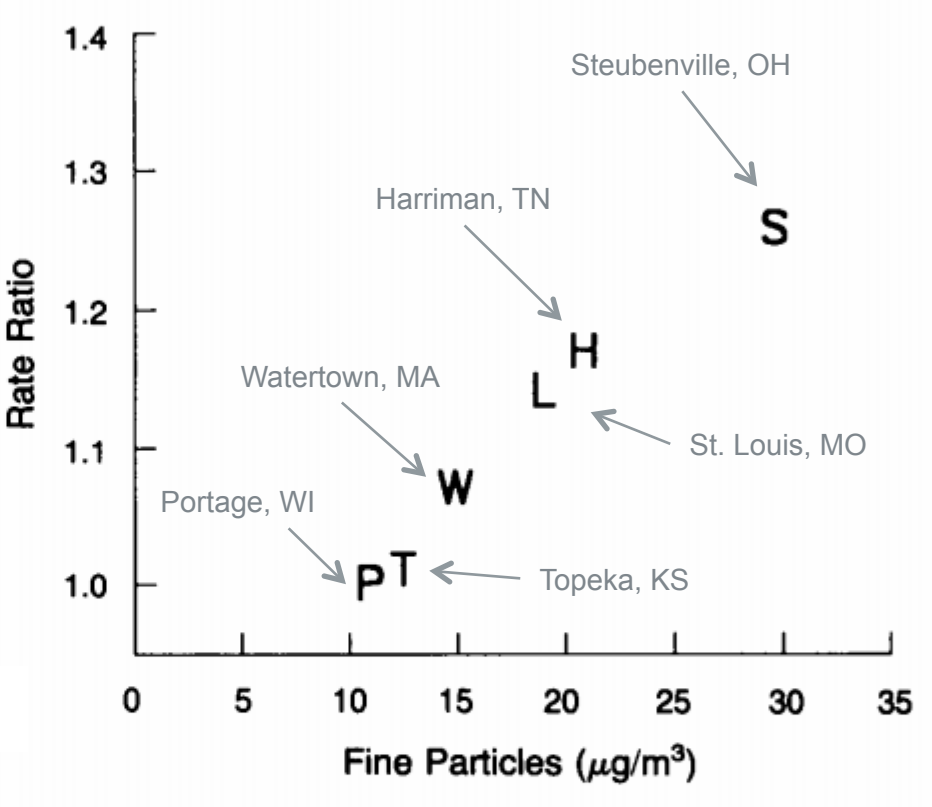
- Early high impact study: The Harvard Six Cities Study
  - Long-term air pollution linked to shortened life expectancy
  - 15 year prospective study of 8000+ adults in six US cities



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 $PM_{2.5}$

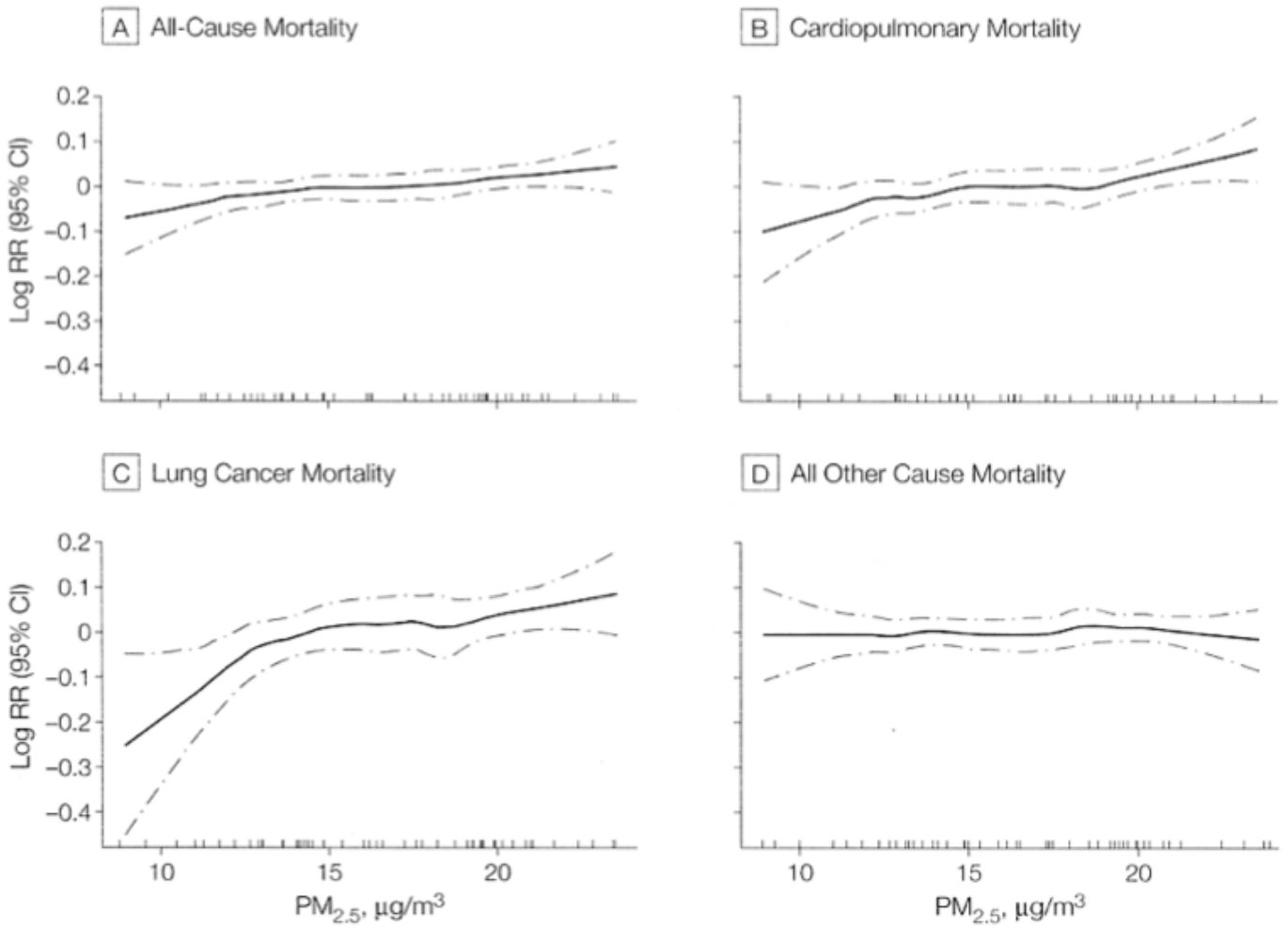


Mean $PM_{2.5}$ concentration measured outdoors in six cities over several years in the 1980s

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

Health effects of outdoor PM: Epidemiology

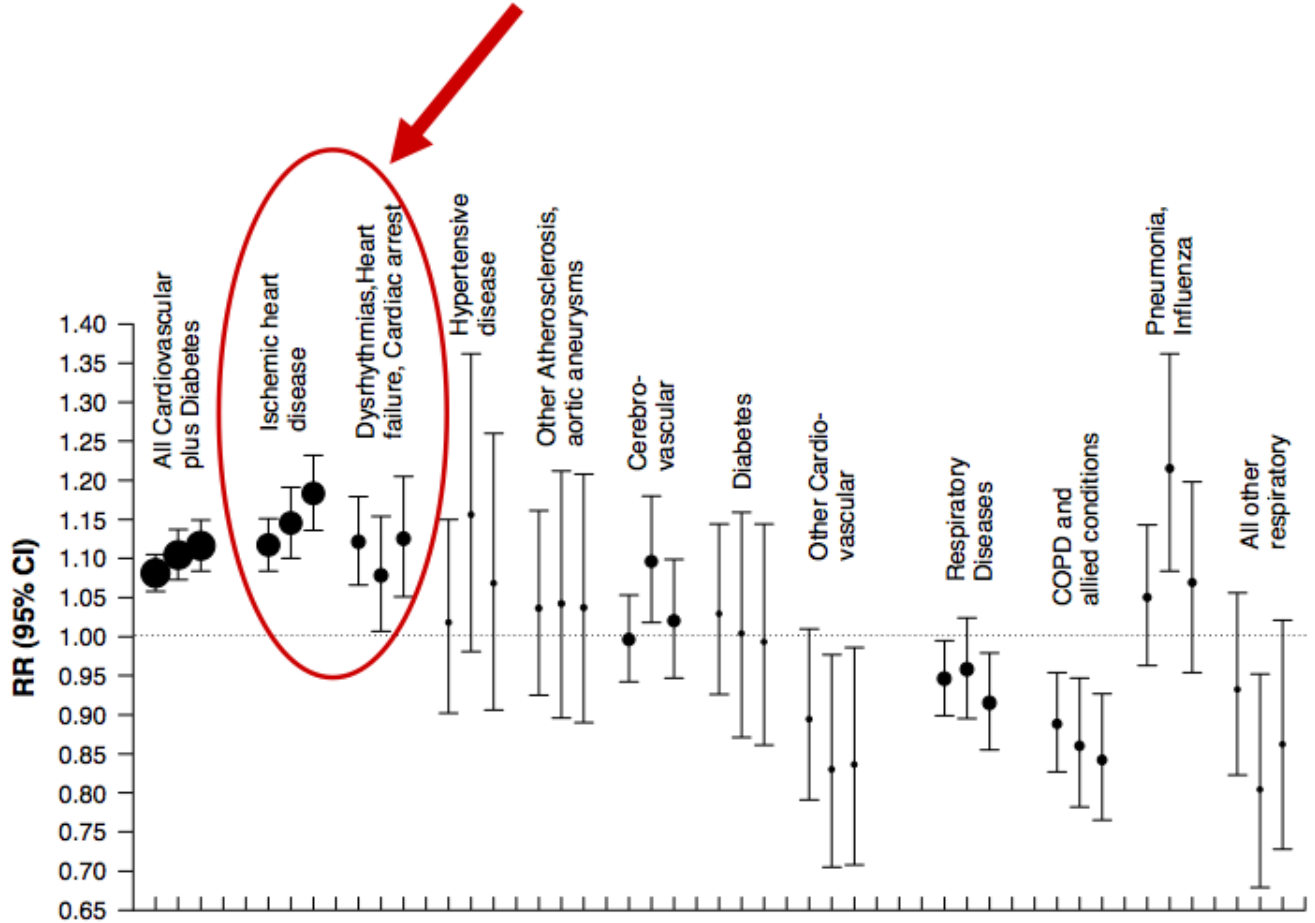
- ACS cohort: over 1 million people
  - Increased $PM_{2.5}$ → increased risk of death



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

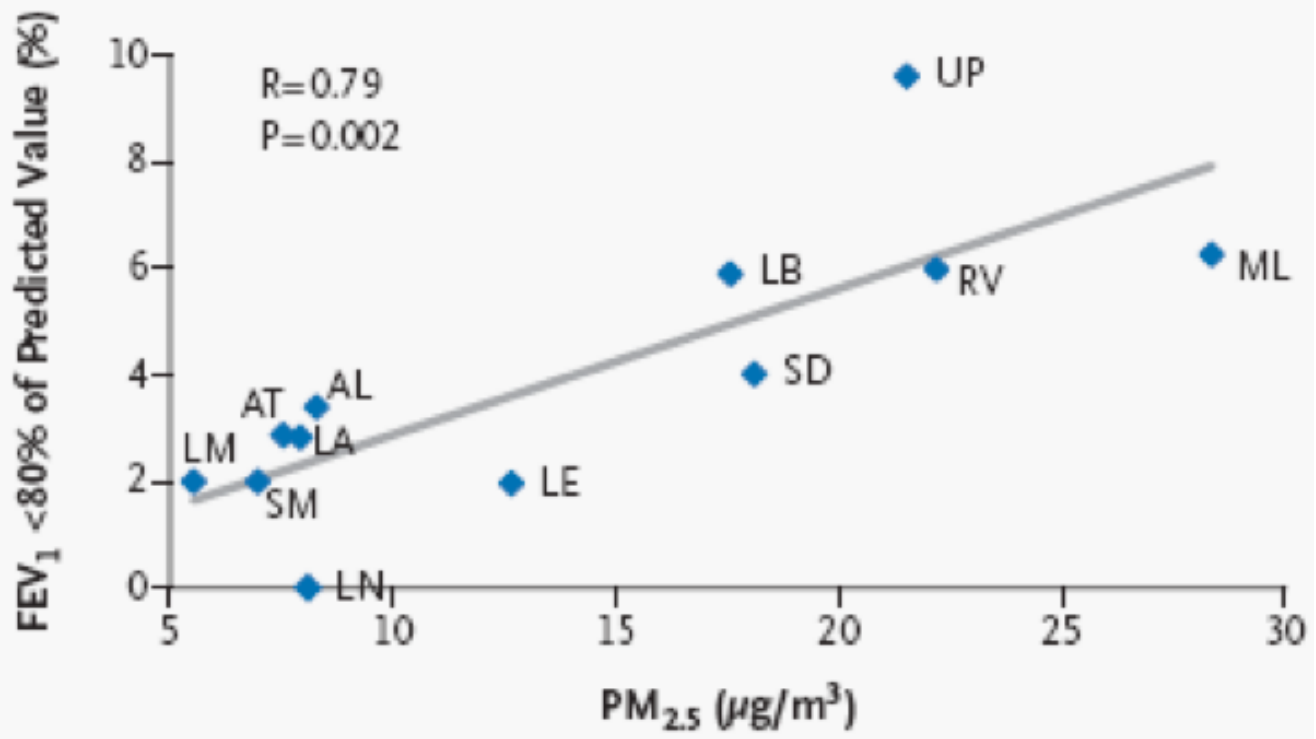


Pope et al., **2004** *Circulation*

Outdoor PM and lung growth

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

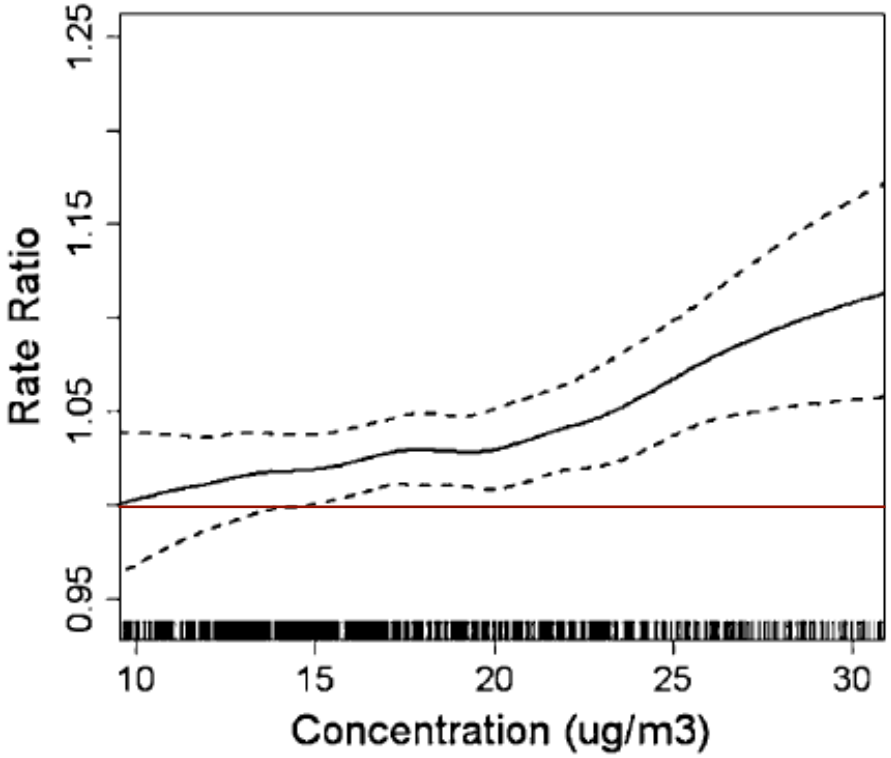
FEV_1 = forced expiratory volume in 1 second
- Volume of air you can exhale in 1 sec



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

Outdoor PM and asthma

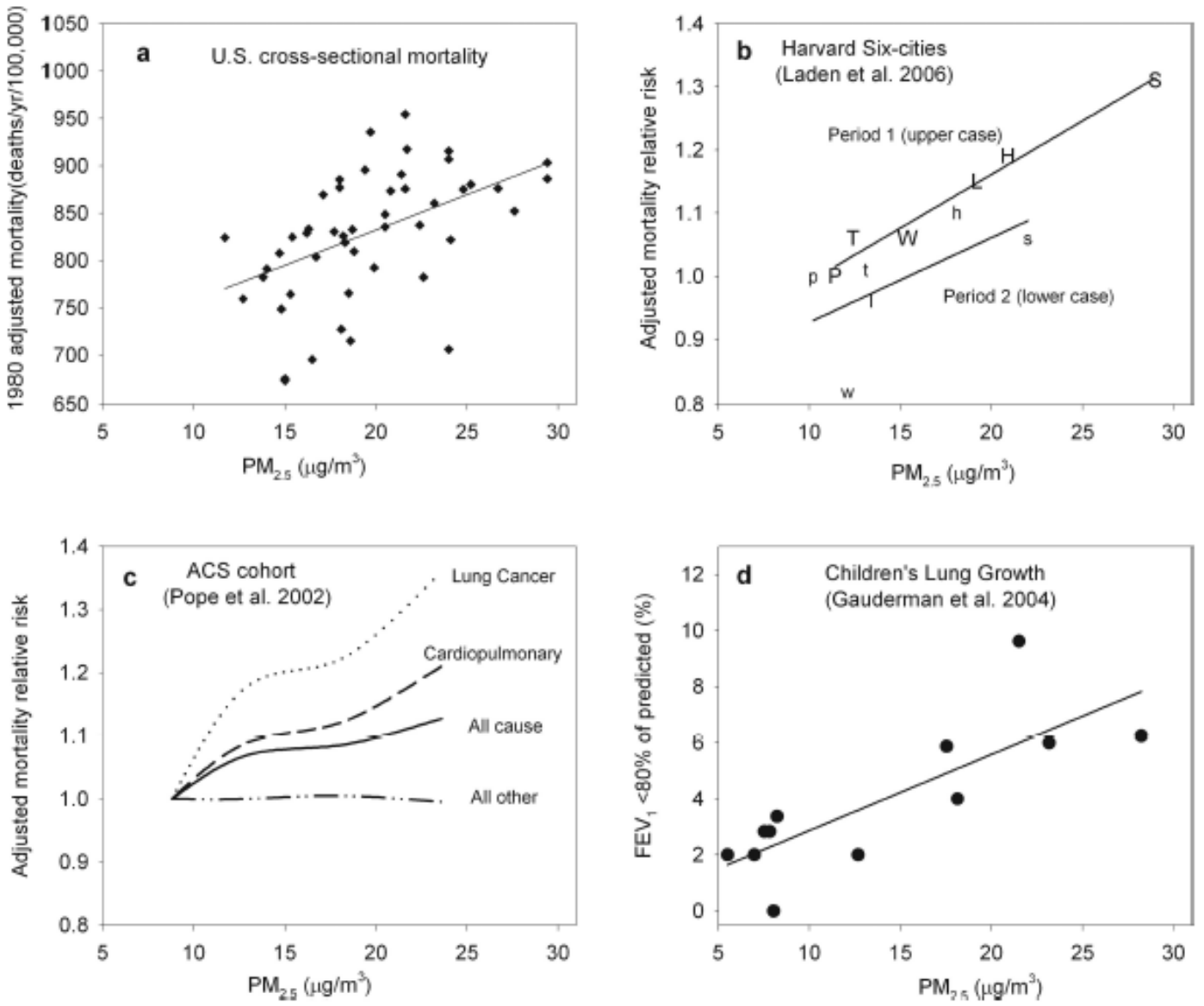
Ambient $PM_{2.5}$ and ER visits for pediatric asthma



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*

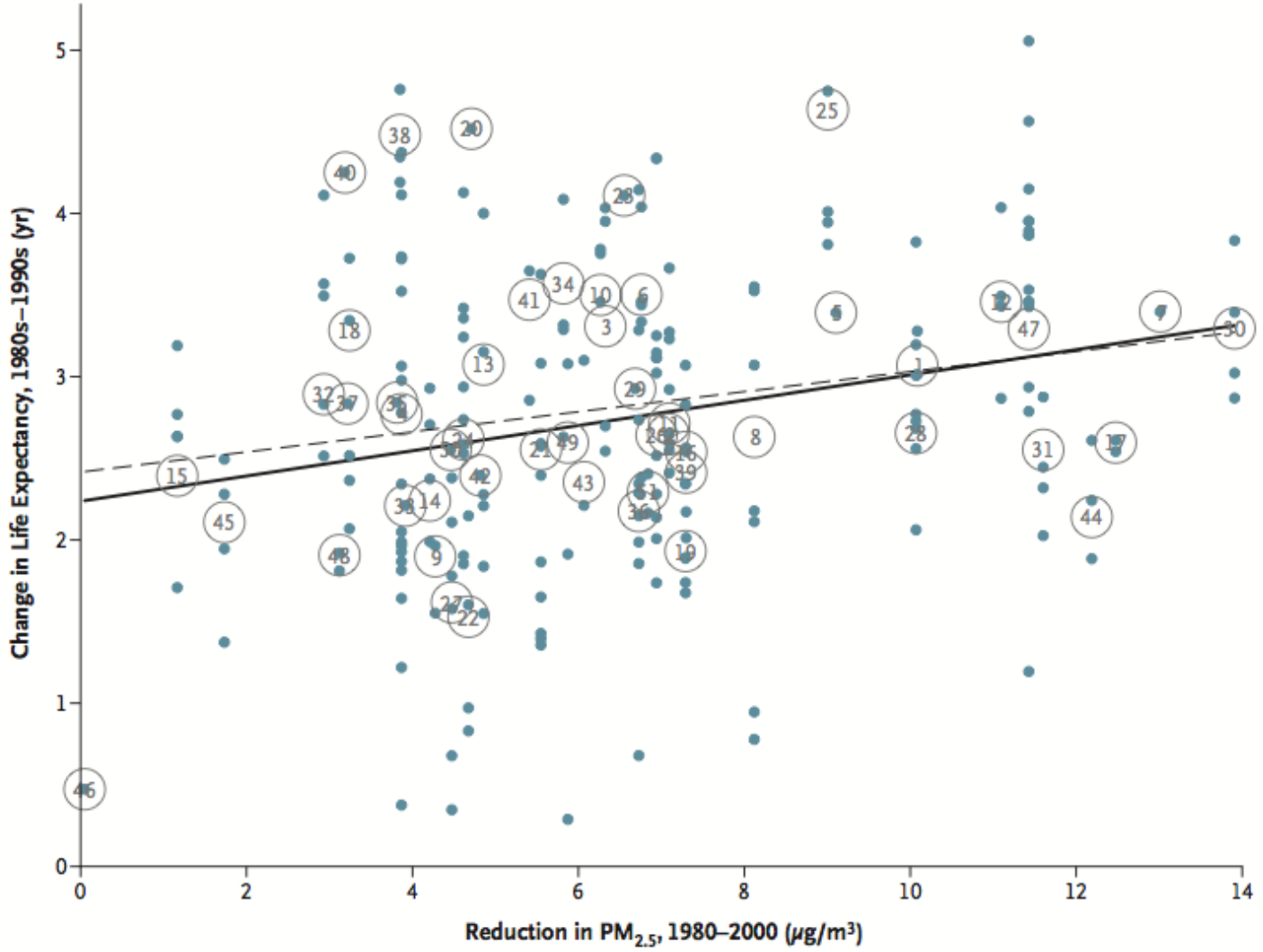
More $PM_{2.5}$ risk relationships



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*

- 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

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

$PM_{2.5}$ 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*

WHERE IS PM MOST IMPORTANT IN THE US?

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 $\mu g/m^3$ (primary) 150 $\mu g/m^3$ (secondary)	Not to be exceeded more than once per yr
		Annual	75 $\mu g/m^3$ (primary)	Annual geometric mean
1987 (52 FR 24634)	PM_{10}	24 h	150 $\mu g/m^3$	Not to be exceeded more than once per yr on average over a 3-yr period
		Annual	50 $\mu g/m^3$	Annual arithmetic mean, averaged over 3 yr
1997 (62 FR 38652)	$PM_{2.5}$	24 h	65 $\mu g/m^3$	98th percentile, averaged over 3 yr
		Annual	15 $\mu g/m^3$	Annual arithmetic mean, averaged over 3 yr[1]
	PM_{10}	24 h	150 $\mu g/m^3$	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 $\mu g/m^3$	Annual arithmetic mean, averaged over 3 yr
2006 (71 FR 61144)	$PM_{2.5}$	24 h	35 $\mu g/m^3$	98th percentile, averaged over 3 yr
		Annual	15 $\mu g/m^3$	Annual arithmetic mean, averaged over 3 yr[2]
	PM_{10}	24 h	150 $\mu g/m^3$	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

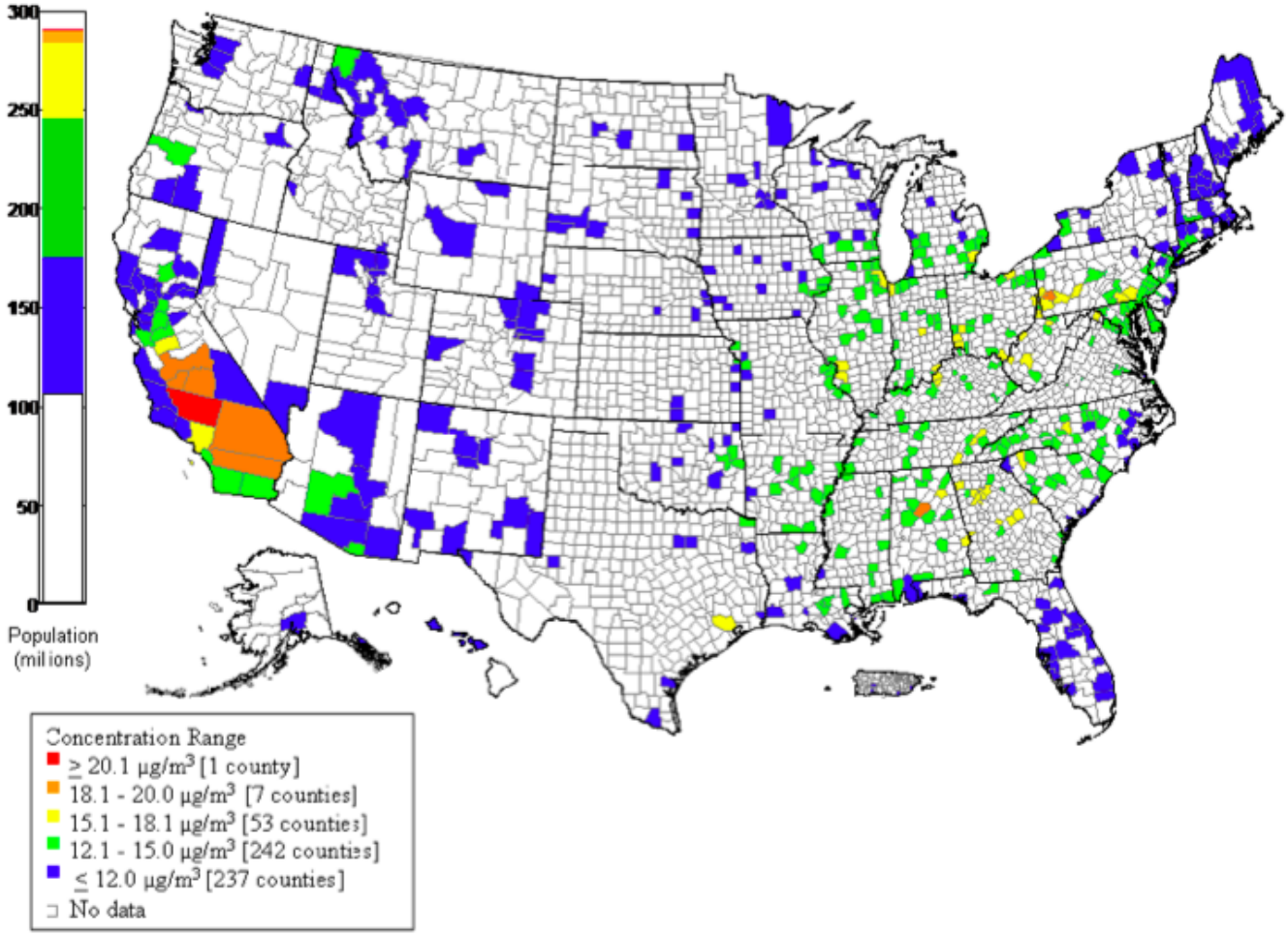
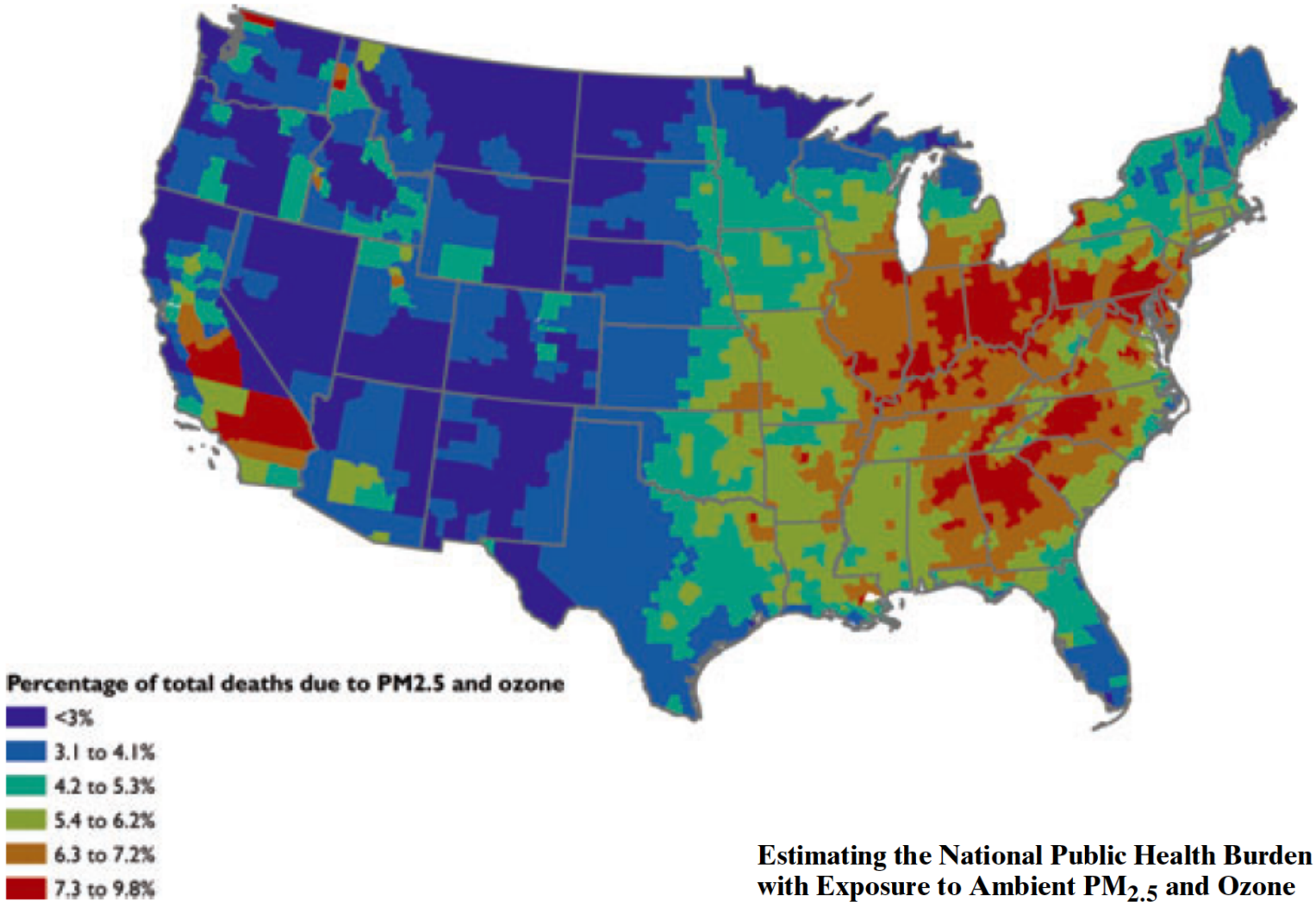


Figure 3-9. **Three-yr avg 24-h $PM_{2.5}$ concentration by county derived from FRM or FRM-like data, 2005-2007. The population bar shows the number of people residing within counties that reported county-wide average concentrations within the specified ranges.**

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*

EPA Integrated Science Assessment for PM

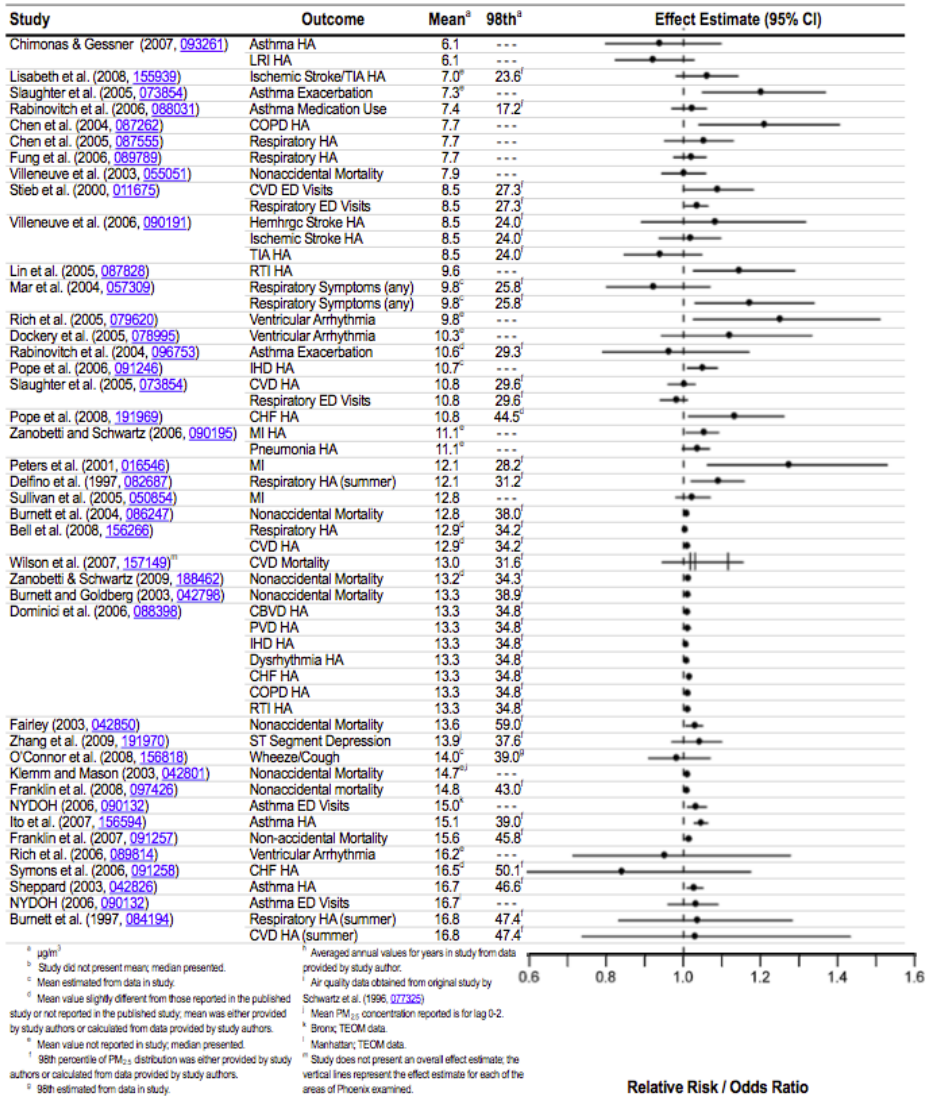
Table 2-1. Summary of causal determinations for short-term exposure to $PM_{2.5}$.

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



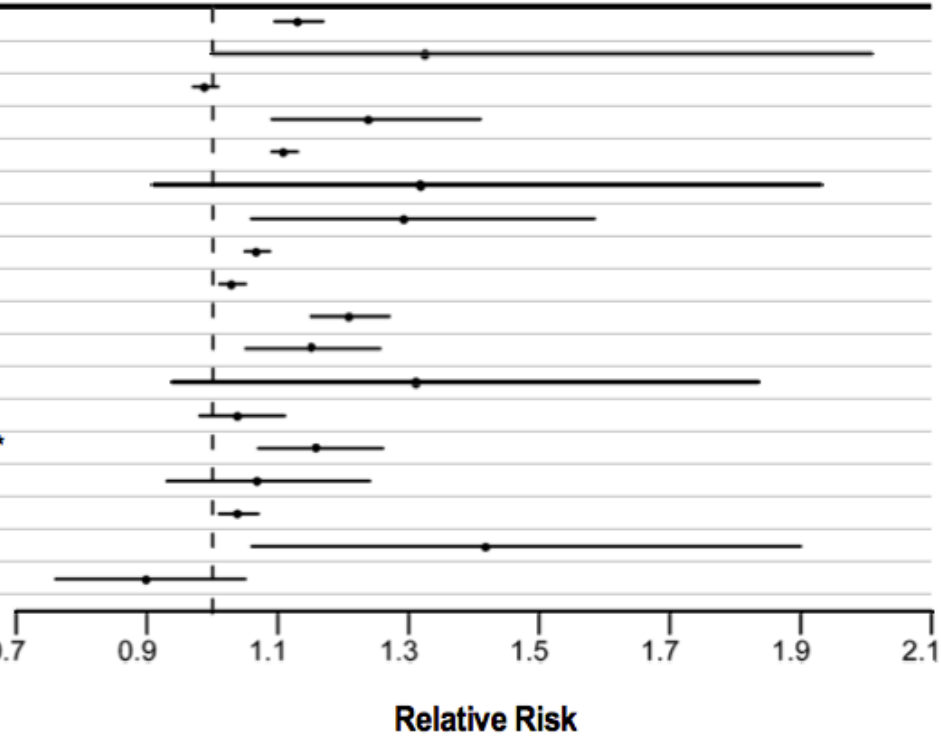
Study	Outcome	Mean[a]	98th[a]
Chimonas & Gessner (2007, 093261)	Asthma HA	6.1	---
	LRI HA	6.1	---
Lisabeth et al. (2008, 155939)	Ischemic Stroke/TIA HA	7.0[e]	23.6[f]
Slaughter et al. (2005, 073854)	Asthma Exacerbation	7.3[e]	---
Rabinovitch et al. (2006, 088031)	Asthma Medication Use	7.4	17.2[f]
Chen 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[g]
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[i]	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	---
Burnett et al. (1997, 084194)	Respiratory HA (summer)	16.8	47.4[f]
	CVD HA (summer)	16.8	47.4[f]

[a] µg/m^3
[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/m^3) 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/m^3.

EPA Integrated Science Assessment for PM

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
\+ $\mu g/m^3$

Figure 2-2. Summary of effect estimates (per 10 $\mu g/m^3$) 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

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

- 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

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

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

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

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

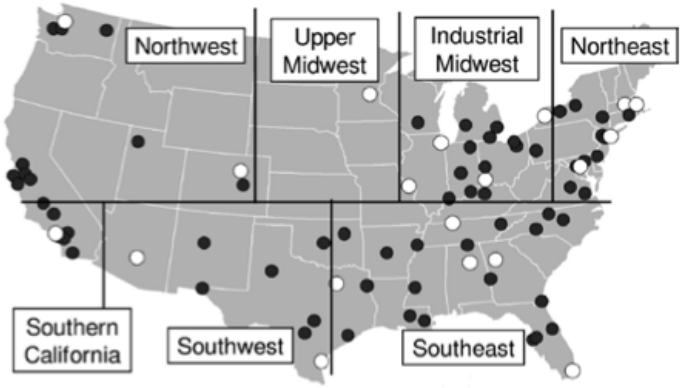


FIGURE 1. Location within the seven U.S. regions of the 19 cities from the NMMAPS with detailed building infiltration rates (open circles) that were used in the original analysis (Figure 2) and the 64 NMMAPS cities with less well-characterized building infiltration rates (closed circles) that were added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

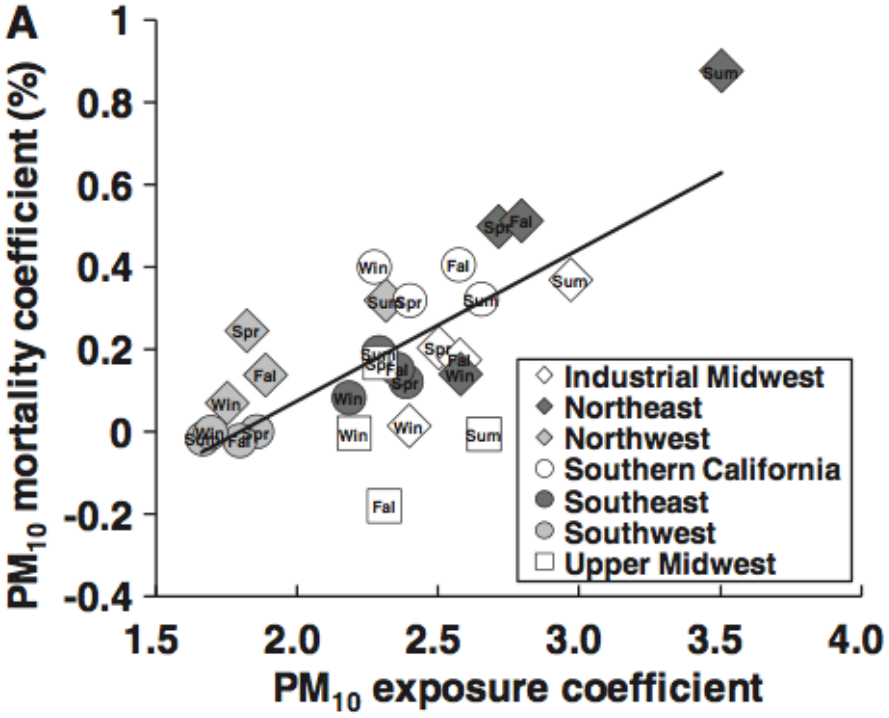
Chen et al., **2012** *Epidemiology*

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*

OTHER INDOOR AIR EPIDEMIOLOGY STUDIES

Gas stoves

Association between gas cooking and respiratory disease in children

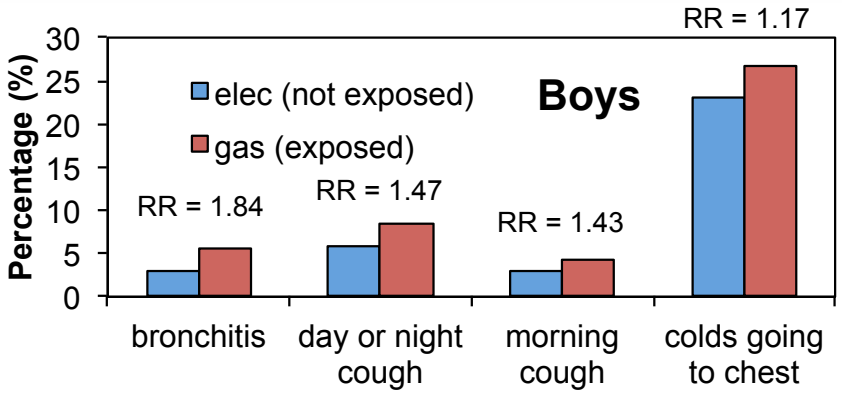
Melia et al., *British Medical Journal* **1977**, 2, 149-152

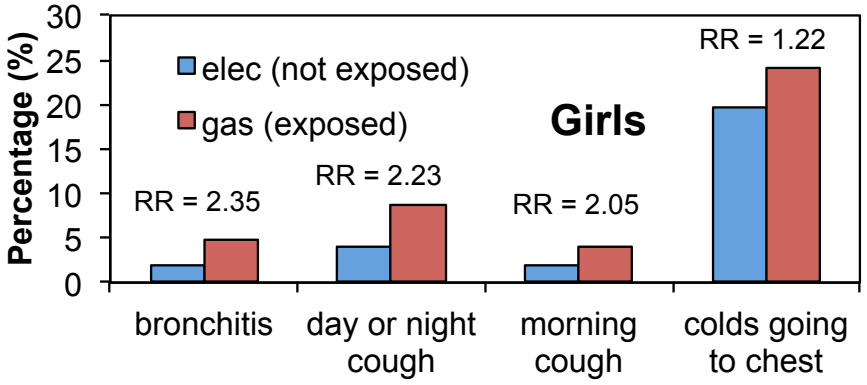
- Four year longitudinal study of the prevalence of respiratory symptoms and disease in almost 6000 6-11 year old school children
  - Children from homes in which gas was used for cooking were found to have more cough, “colds going to the chest,” and bronchitis than children from homes where electricity 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 samples
  - 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 NO2
    - What does that mean?

Respiratory Symptom	% of Children	Gas Stove Exposure		Bedroom NO_2	
		OR*	95% CI	OR*	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.

Gas stoves

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

- 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

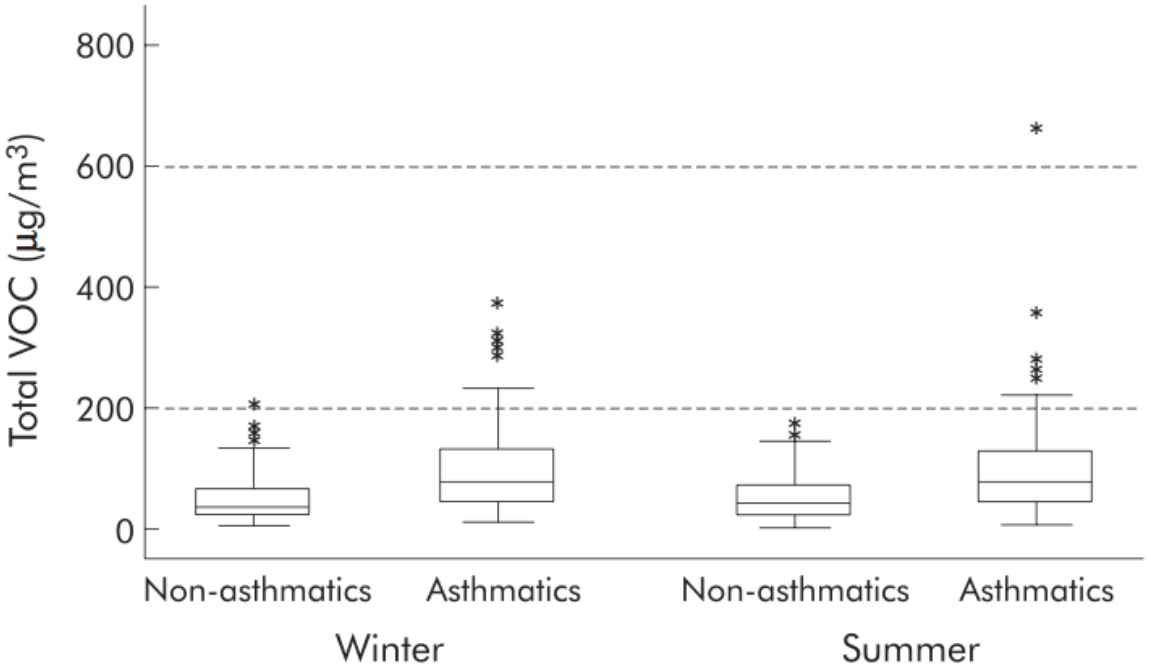


Figure 1 Seasonal differences in exposure levels to total volatile organic compounds (VOCs, µg/m^3) for asthmatic and non-asthmatic children.

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

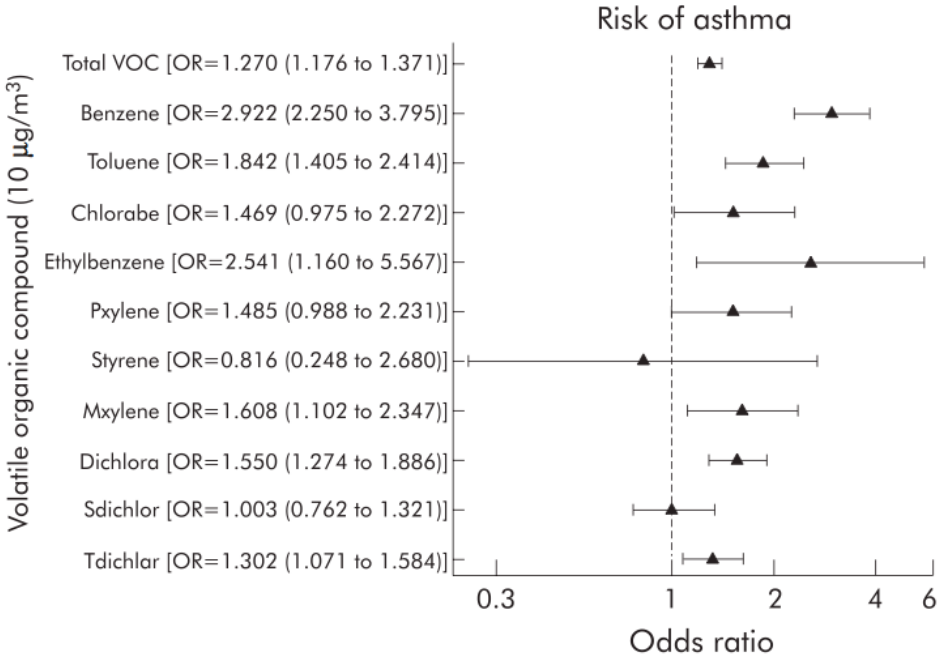


Figure 3 Adjusted odds ratio with ±95% confidence intervals for the risk of asthma with each 10 mg increase in exposure to VOCs.

Exposure-response curve

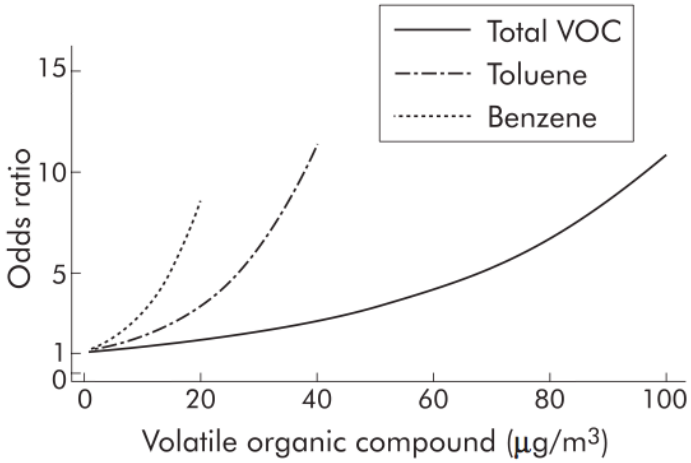


Figure 2 Asthma in young children associated with exposure to indoor volatile organic compounds (µg/m^3): odds ratios adjusted for age, sex, atopy, socioeconomic status, smoking indoors, air conditioning, house dust mites, and gas appliances.

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

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

Use of cleaning products

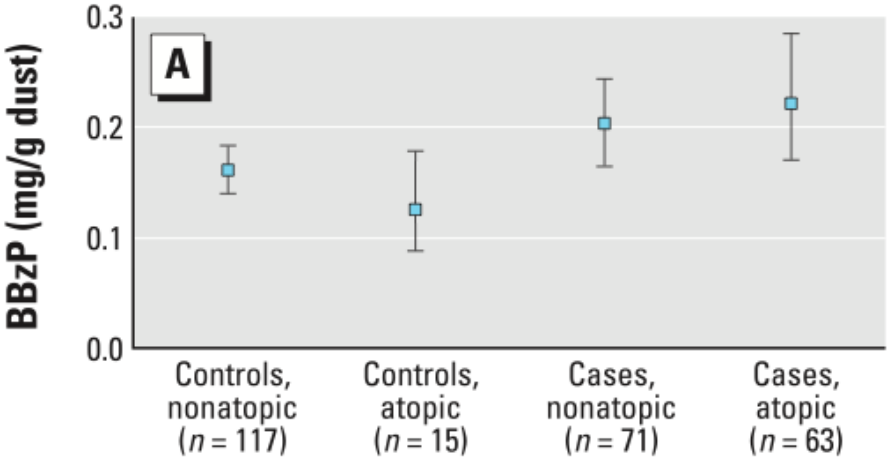
- Identified 3503 people in 10 countries who do the cleaning in their homes and who were free of asthma at the beginning of the study
- Frequency of use of 15 types of cleaning products was obtained by interview
- Tracked incidence of asthma
- Use of cleaning sprays at least weekly (42% of participants) 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

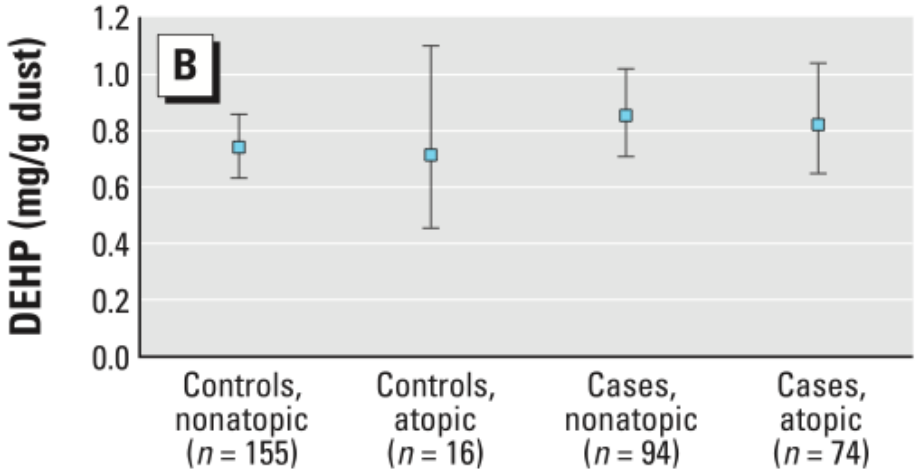
What about SVOCs?

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

			P-value	
Type of buildings	Cases	Controls	*t*-test	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?

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

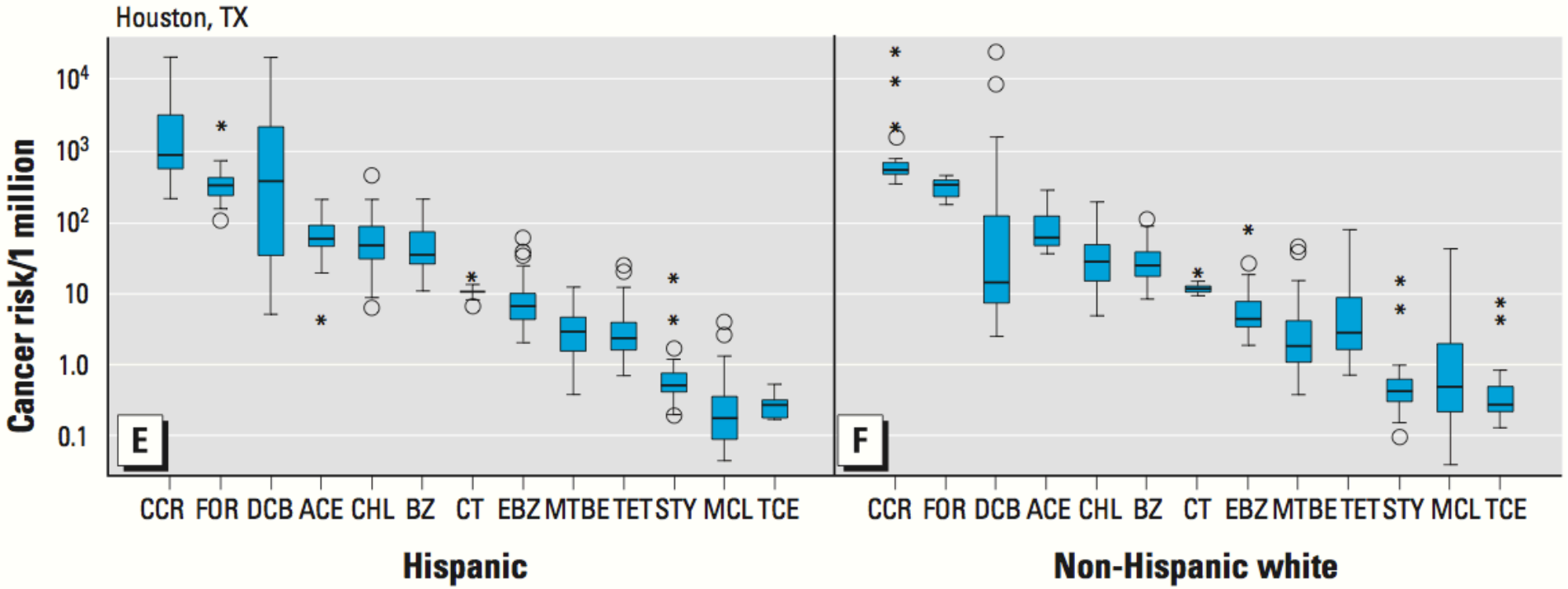
Chemical	Category	Potency factor oral route $(mg/kg\text{-}day)^{-1}$	Potency factor inhalation route $(mg/kg\text{-}day)^{-1}$
Arsenic	A	1.75	50
Benzene	A	2.9×10^{-2}	2.9×10^{-2}
Benzol(a)pyrene	B2	11.5	6.11
Cadmium	B1	—	6.1
Carbon tetrachloride	B2	0.13	—
Chloroform	B2	6.1×10^{-3}	8.1×10^{-2}
Chromium 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.



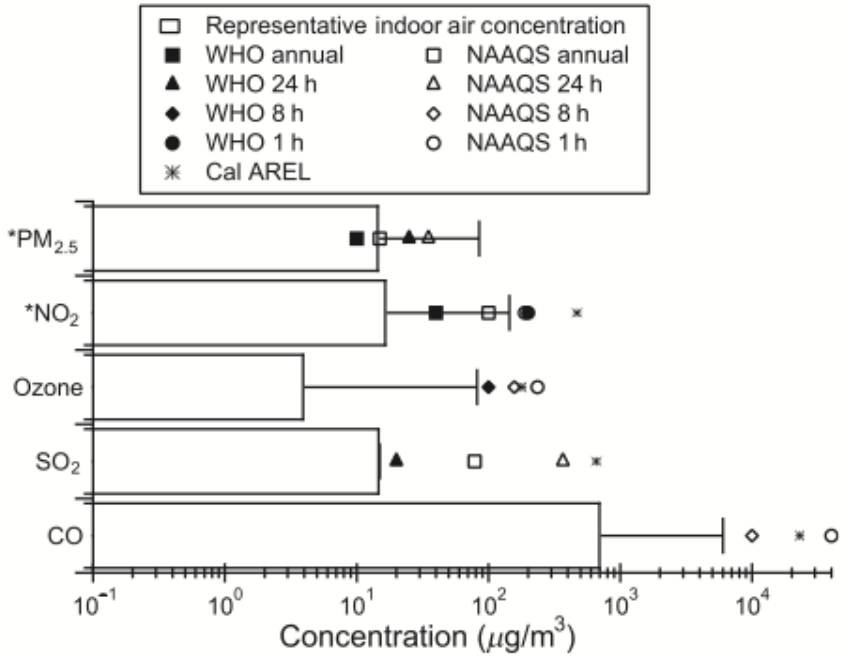
CONCLUSIONS: Hispanics appear to be disproportionately affected by certain HAPs from indoor and outdoor sources. Policies that aim to reduce risk from exposure to HAPs for the entire population and population subgroups should consider indoor air pollution.

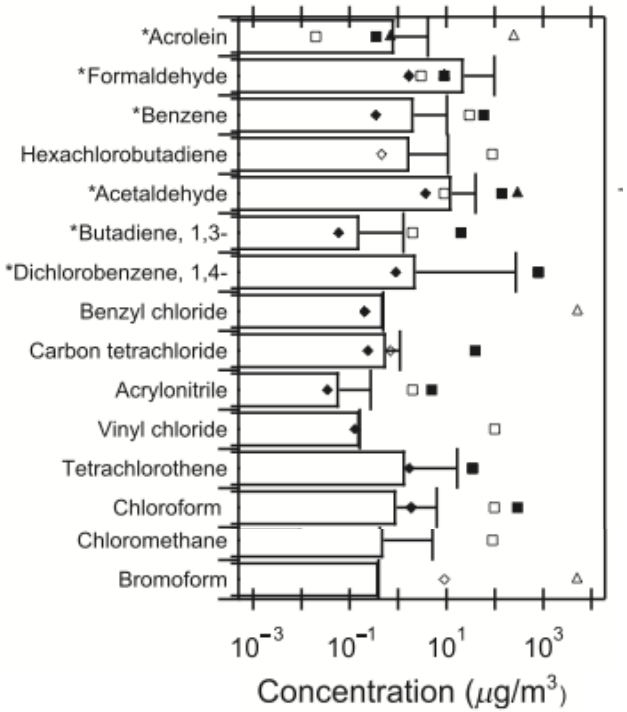
Hazard assessment of chemical air contaminants measured in residences

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

Table 1 Publications with chronic exposure-relevant concentrations

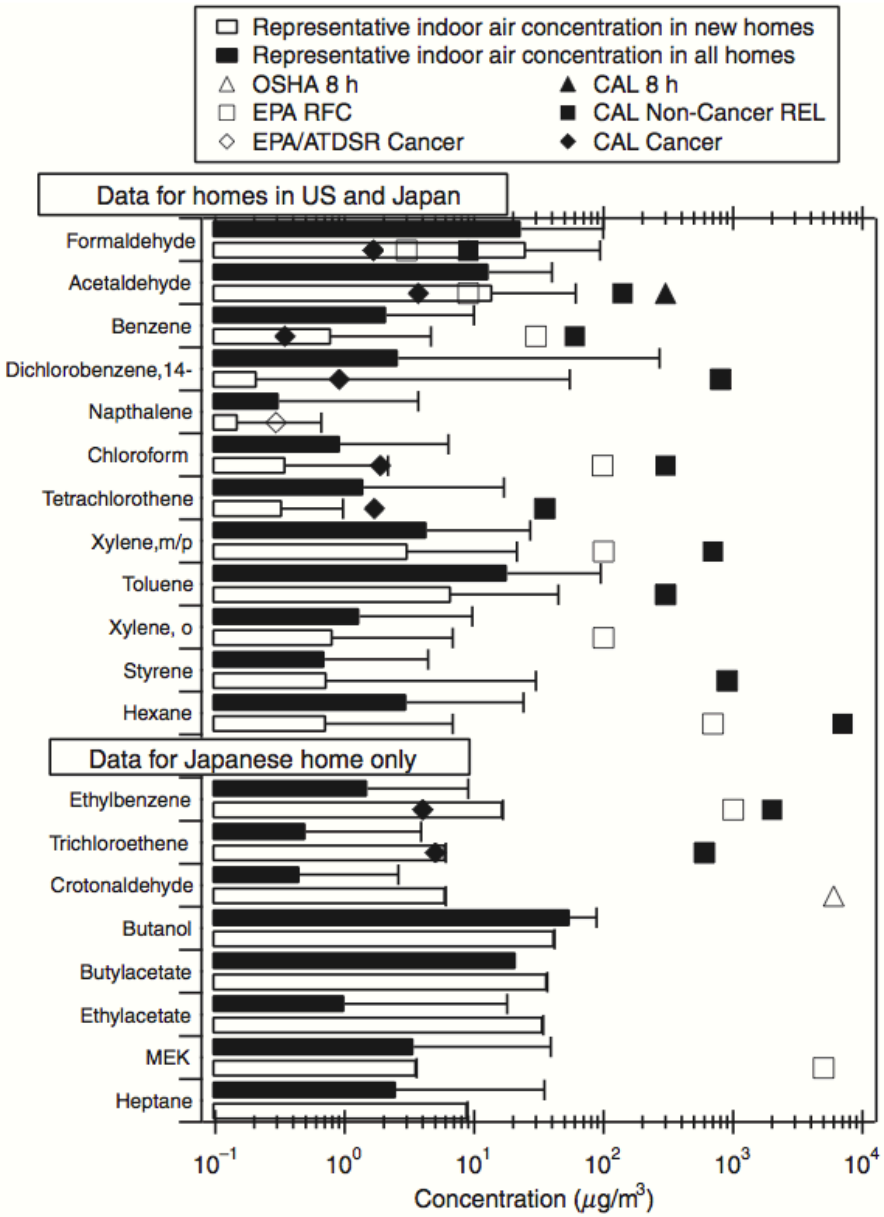
	Study	Sample Period	Location: city, country or US State	US homes	New homes	Criteria pollutants	VOCs	Aldehyde	SVOCs	Metals	Number of samples
1	Topp et al. (2004)	2 weeks	Hamburg/Erfurt, Germany			N	X				2524
2	Park and Ikeda (2006)	24 h	Japan		X		X	X			2151
3	Geyh et al. (2000)	6 months	Upland, CA, USA	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**

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

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

Intake-incidence-DALY approach

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

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

Intake-DALY approach

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

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

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

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**

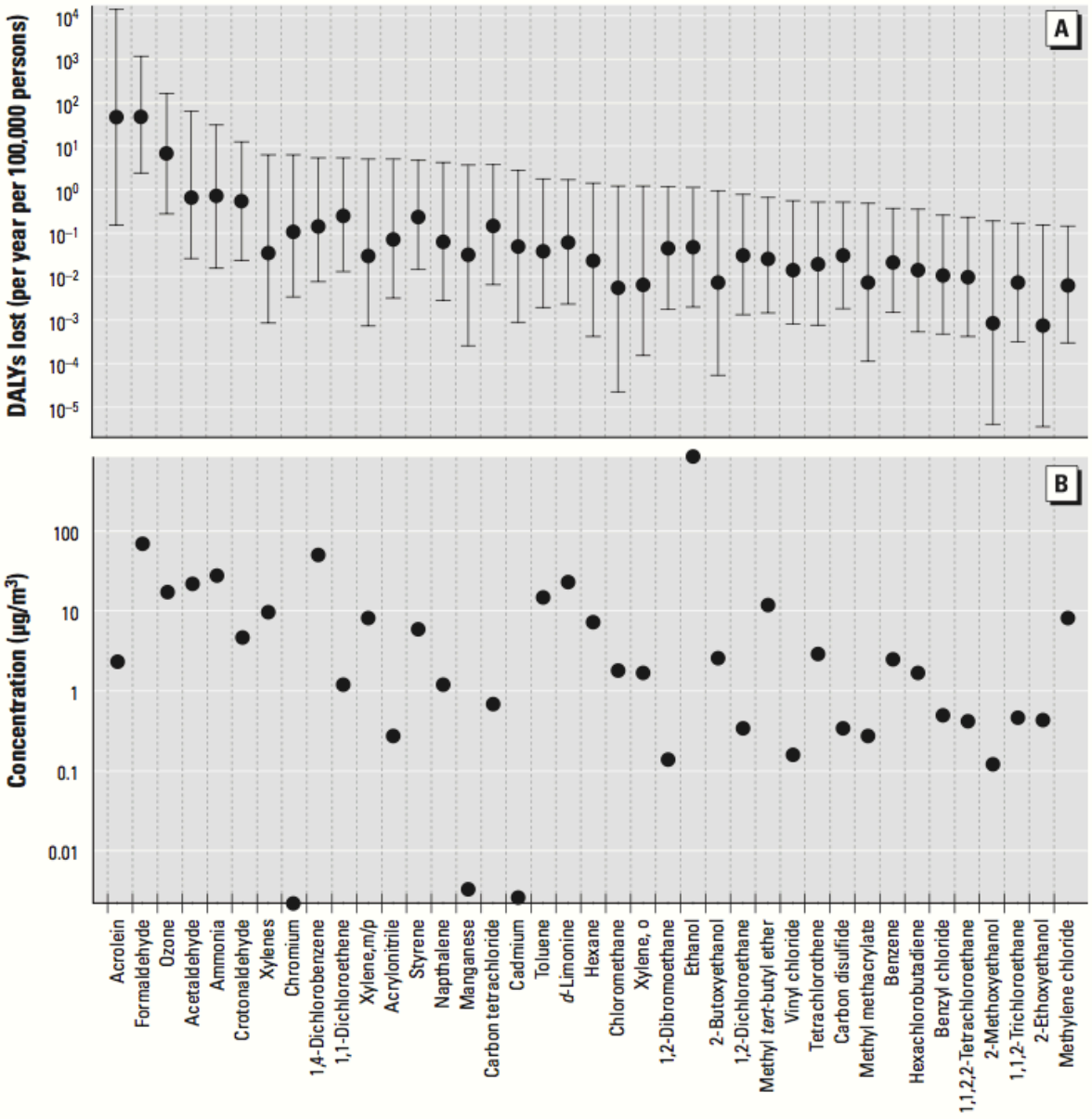
Table 2. Criteria pollutant C-R function outcomes and DALYs lost per incidence.

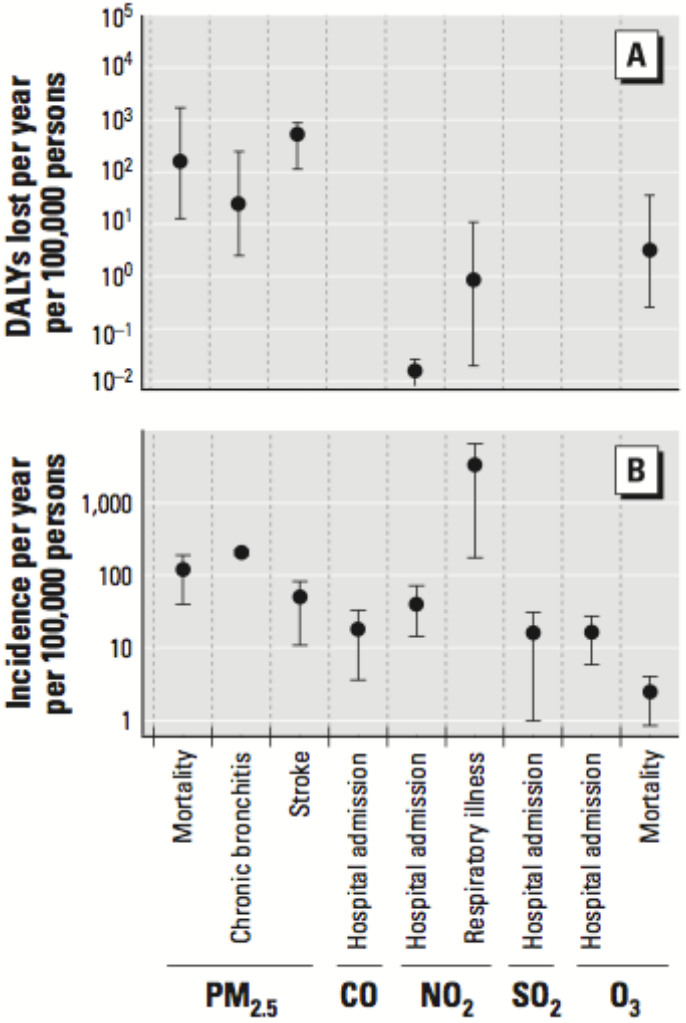
Pollutant	Outcome	β-Coefficient (95% CI)	y_0	DALYs lost per incidence (95% CI)
$PM_{2.5}$	Total mortality (Pope et al. 2002)	0.058 (0.002, 0.010)	7.4×10^{-3}	1.4 (0.14, 14) (Pope 2007; Pope et al. 2002, 2009)
	Chronic bronchitis (Abbey et al. 1995)	0.091 (0.078, 0.105)	0.4×10^{-3}	1.2 (0.12, 12) (Lvovsky 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**





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

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