Biomass smoke and Health

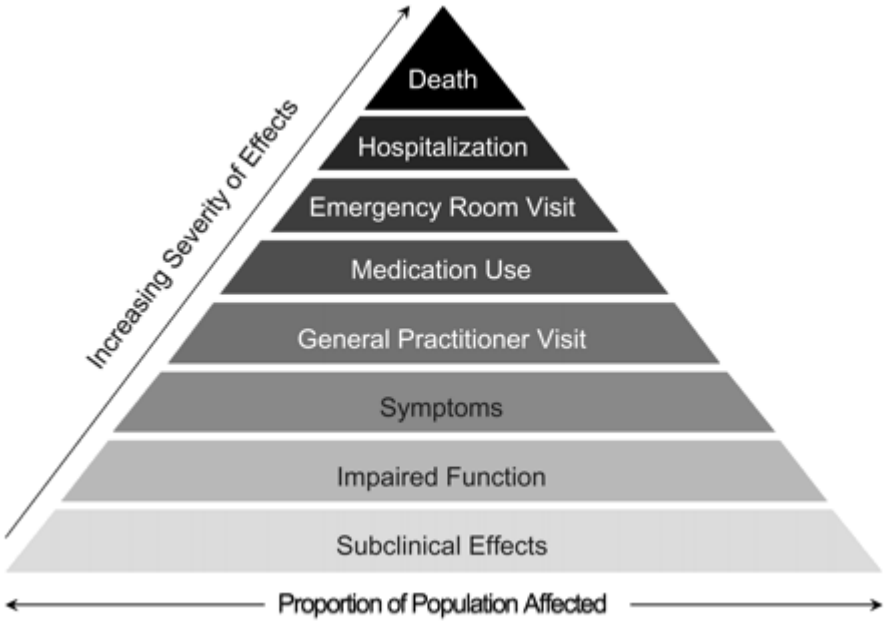
Michael Brauer

Webinar
October 24, 2013

a place of mind
THE UNIVERSITY OF BRITISH COLUMBIA

Air pollution and health

- Air pollution (individual) **risk** is small…but large **exposed population = large population risk**
  - Smoking: Larger risk, smaller exposed population
- On **days** with worse air quality, more people die*
- In **more polluted cities**, people die earlier than in less polluted cities…
- …and, in the **most polluted areas** of cities, there is an increased risk of dying



Larrieu et al. Am J Epidemiol, 2009

*out-of-hospital, >65 yrs

Wood biomass fuels in context

- Inexpensive, secure fuel
  - Increasing/fluctuating costs & taxes for fossil fuels
- Promoted as renewable, GHG-neutral fuel
- Relatively unregulated source
- Impact on winter air quality coinciding with stagnation
- Exposure proximity, high “intake fraction”

POLICYFORUM

13 MARCH 2009 VOL 323 SCIENCE www.sciencemag.org

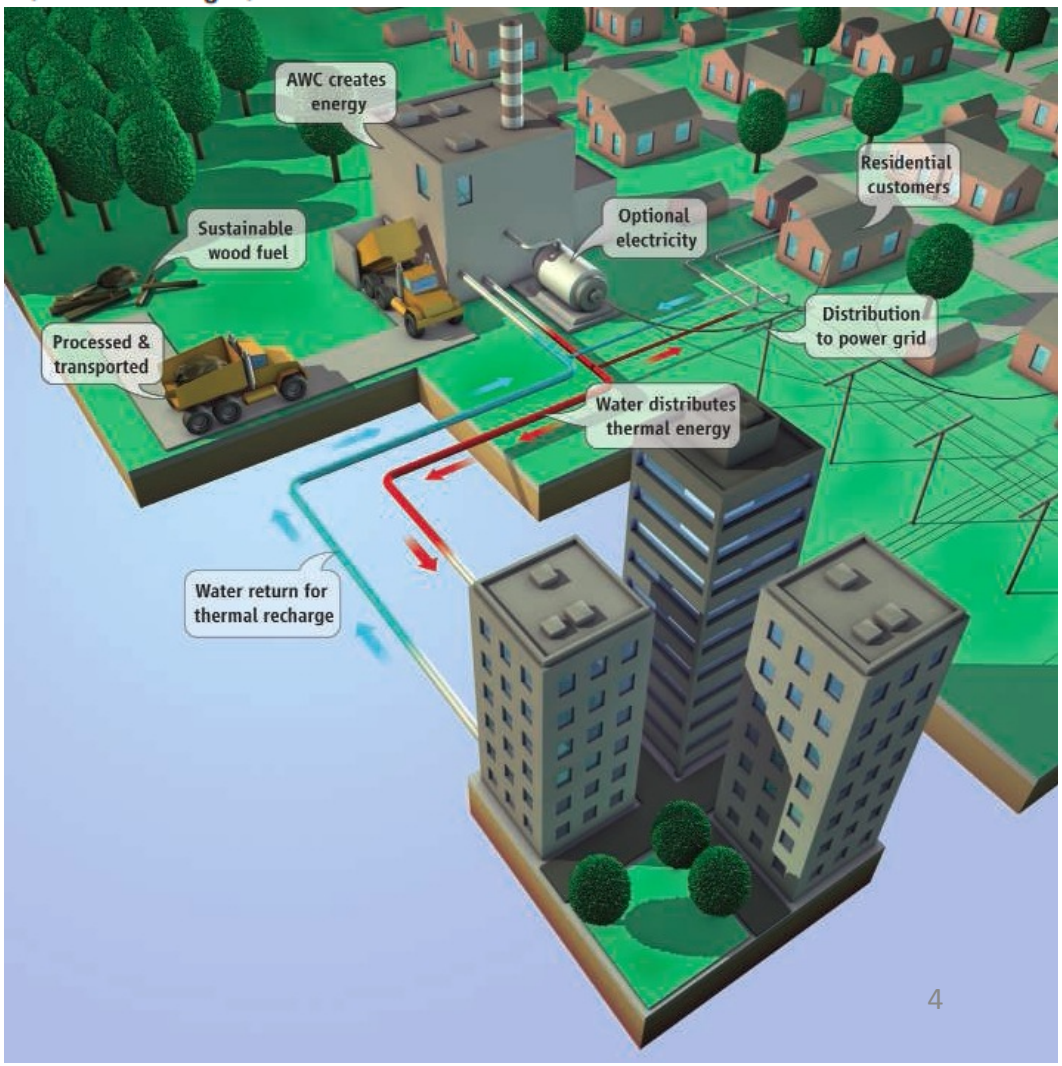
RESOURCE POLICY

Wood Energy in America

Daniel deB. Richter Jr.,[1*] Dylan H. Jenkins,[2] John T. Karakash,[3] Josiah Knight,[4] Lew R. McCreery,[5] Kasimir P. Nemestothy[6]

Issues raised in response:

- Is biomass really inexpensive?
- Sufficient supply?
- Emissions of current technologies > emissions of oil, natural gas
  - need cleanest fuels/emissions controls when burned in populated areas and with distributed sources
- Carbon neutrality
  - Stock replacement
  - Black Carbon



Wood biomass fuels in context

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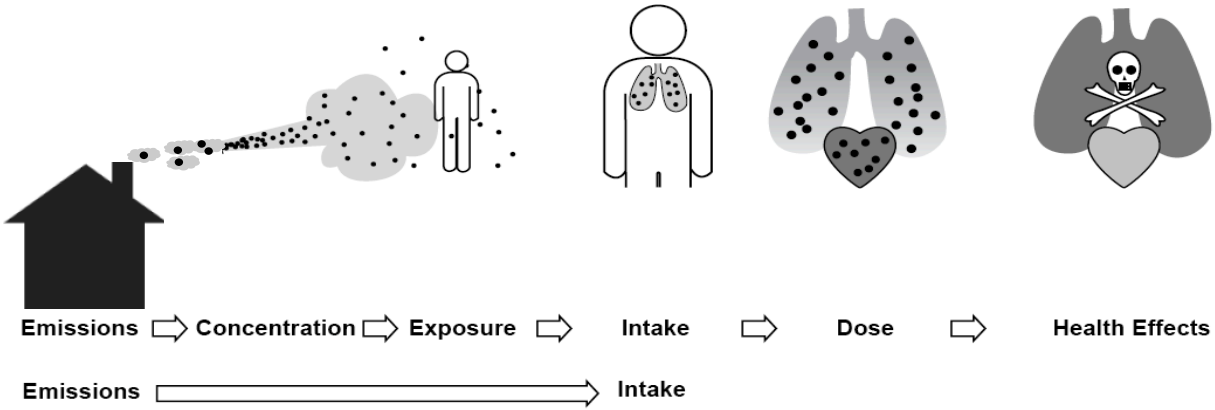


Ries F, Marshall J, Brauer M. Intake fraction of urban wood smoke. Environ Sci Technol, 2009 Jul 1;43(13):4701-6; Brauer M, Miller P, Allen G, Rector L. Modeling Pollution from Residential Wood Combustion. EM Magazine (Air and Waste Management Association). May, 2010. pp 24-28

Biomass smoke and health: evidence

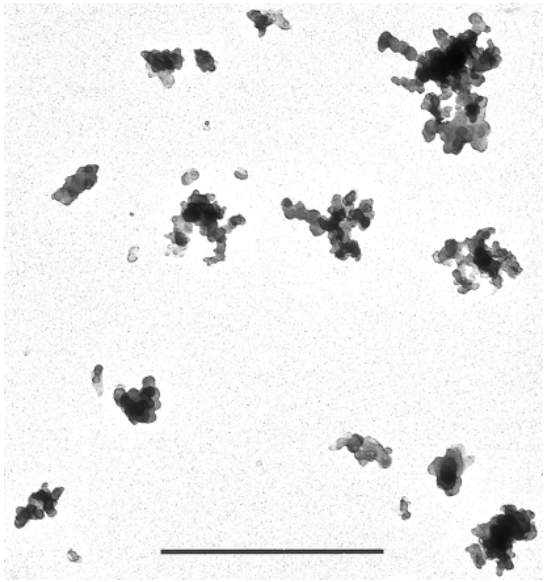
- Constituents/Composition ($PM_{2.5}$, aldehydes, PAHs)
- Toxicology
- High concentration, chronic exposures – developing countries
- High concentration acute/sub-chronic exposures – wildland firefighters
- Firesmoke, agricultural burning
- Controlled human exposures
- Residential woodsmoke epidemiology

- NOTE: very little direct research on health impacts of ICI-scale combustion

Do woodsmoke particles pose different levels of risk from other particles?

- **Respiratory disease: No**
- **Cardiovascular disease: ?**

Woodsmoke Particles



Bar = 1 µm = 1/1000 of 1mm

Inhalation Toxicology, 19:67–106, 2007
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DOI: 10.1080/08958370600985875

Woodsmoke Health Effects: A Review

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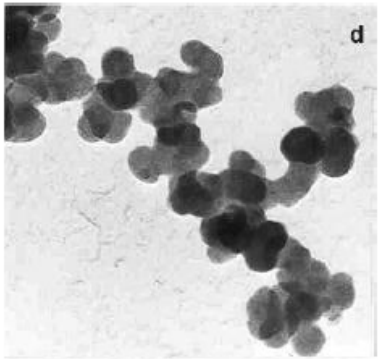
PAH content: **WS >Traffic PM**

Inflammatory potential: **WS ≈ Traffic PM**

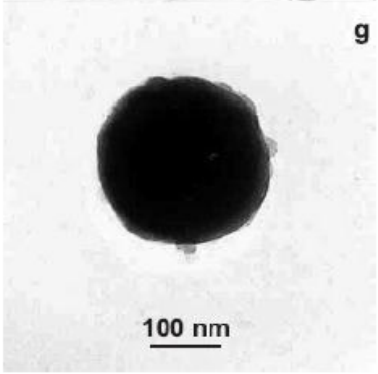
Kocbach et al. Toxicology. 2008, 247(2-3):123-132

Naeher LP, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, Koenig JQ, Smith KR. Woodsmoke health effects: A review. Inhal Toxicol. 2007 Jan;19(1):67-106.

PM composition



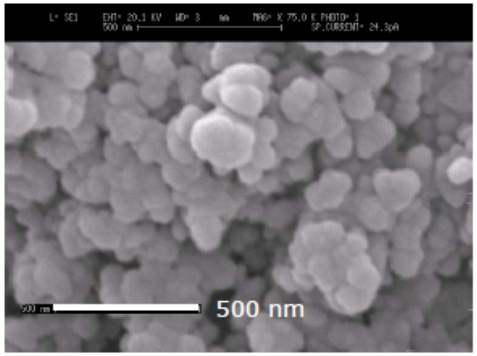
Wood smoke soot



Wood smoke organic particles (low-temp combustion)

"conventional"

from *Kocbach et al, Science of the Total Environment, 2005*)



"Good" wood pellet combustion PM (alkali salt particles)

"advanced"

from *Boman et al, Energy and Fuels 2011;25:(1):307-314*

Combustion source	Emissions (mg/MJ)	Composition
Open fireplace	160 – 910 (0.38-2.2 lb/mmBTU)	
Conventional woodstove	50 – 2100 (0.38-2.2 lb/mmBTU)	
Conventional log boilers	50 – 2000 (50 – 250)	
‘Modern” woodstoves log/chip boilers	34 – 330 5 – 450	
Pellet stoves/boilers	10 – 50 (0.04 – 0.21 lb/mmBTU)	

adapted from: Kocbach Bølling et al. 2009

Animal/Cellular Toxicology

Inflammation: Medium Temp > High Temp

Low oxygen > High oxygen

 > 

Soluble inorganic ash particles:

- inflammation in cell culture
- no inflammation in animal inhalation studies
-  soluble and cleared from lungs

Cell cytoxicity:

 > Diesel >  > 

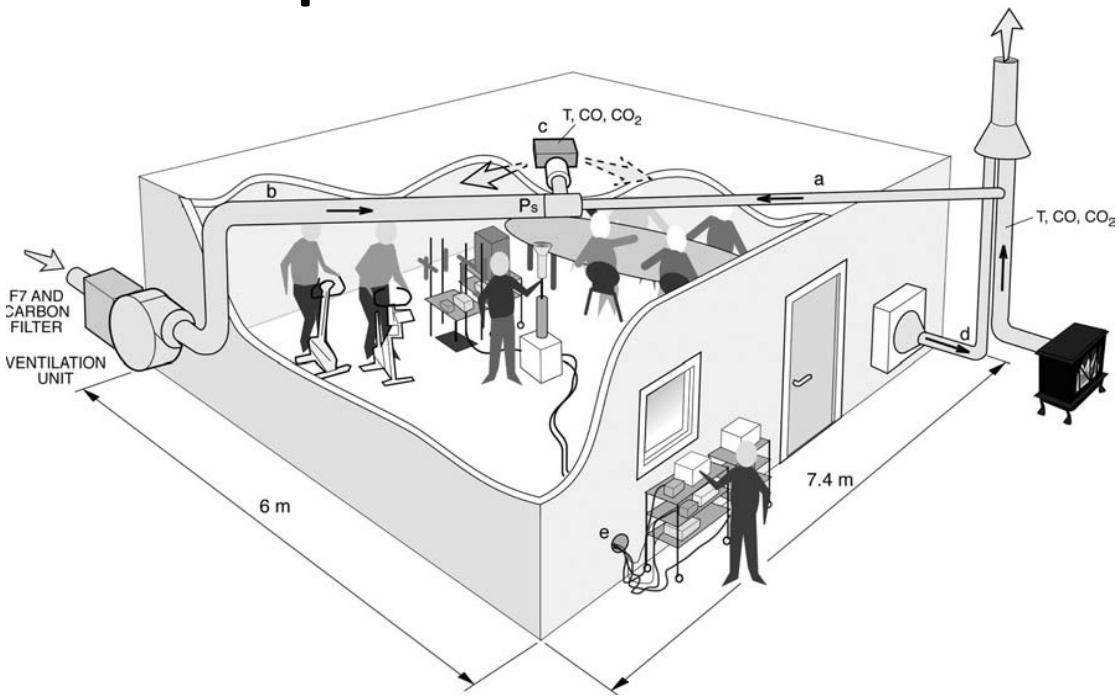
adapted from: Kocbach Bølling et al. 2009

Combustion source	Emissions (mg/MJ)	Composition
Open fireplace	MORE TOXIC	
Conventional woodstove		
Conventional log boilers		
‘Modern” woodstoves log/chip boilers		
Pellet stoves/boilers	LESS TOXIC	

adapted from: Kocbach Bølling et al. 2009

Controlled human exposure studies

- **Subjects exposed to realistic (high) concentrations (~250 $\mu g/m^3$) of woodsmoke for 4 hrs**
  - **Increases in measures of inflammation, oxidative stress post-exposure compared to clean air**



- **Pellet stove incomplete combustion**
  - **No inflammation**
  - **Early adaptive protective response**

Sallsten, G et al. Experimental wood smoke exposure in humans. *Inhal. Toxicol*. 18(11):855–864.; Barregard L et al. Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation. Inhal Toxicol. 2006 Oct;18(11):845-53.; Danielsen PH et al. Oxidatively damaged DNA and its repair after experimental exposure to wood smoke in healthy humans.. Mutat Res. 2008 Jul 3;642(1-2):37-42.; Barregard L et al. Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress.. Occup Environ Med. 2008 May;65(5):319-24.

Sehlstedt, M., R. Dove, et al. (2010). "Antioxidant airway responses following experimental exposure to wood smoke in man." Particle and Fibre Toxicology 7(1): 21.

Biomass smoke epidemiology

" …..epidemiologic studies of indoor and community exposure to biomass smoke indicate a generally consistent relationship between exposure and increased **respiratory symptoms, increased risk of respiratory illness, including hospital admissions and emergency room visits, and decreased lung function**. Several studies suggest that **asthmatics** are a particularly susceptible subpopulation with respect to smoke exposure…The effects of community exposure to biomass air pollution (from wildfires) on **mortality** have not been sufficiently studied to support general conclusions."

Woodsmoke health effects: a review. Naeher LP, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, Koenig JQ, Smith KR. Inhal Toxicol. 2007 Jan;19(1):67-106. Review.

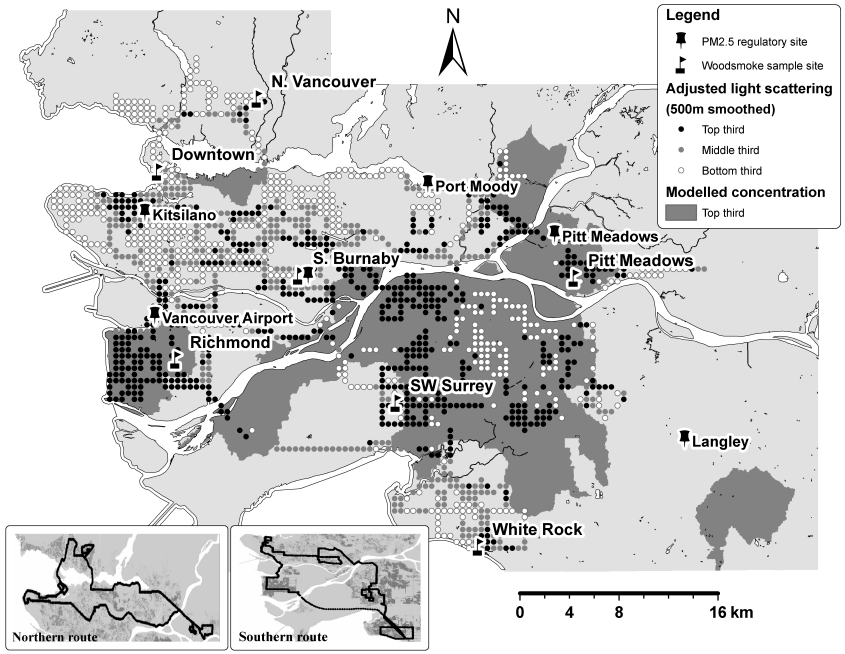
Adapted, from Fay Johnston

Biomass combustion and CVD

Country	Study	Exposure	Outcome	Result
New Zealand	McGowan (2002)	Outdoor PM_{10} (90% from biomass in winter)	CVD admissions	+
New Zealand	Barnett (2006)	Outdoor PM_{10}	CVD admissions	-
Chile	Sanhueza (2009)	Outdoor PM_{10} (90% from biomass in winter)	CVD admissions CVD mortality	+ +
Canada	Allen (2011)	Indoor $PM_{2.5}$ intervention study	Markers of inflam & endothelial function	+
India	Ray (2006)	Biomass vs non biomass fuel use	Markers of thrombosis risk	+
Turkey	Emiroglu (2010)	Biomass vs non biomass fuel users	Ventricular dysfunction	+
Guatemala	McCracken (2007, 2011)	Biomass smoke chimney intervention	Blood pressure, ST-segment depression	+

Woodsmoke & multiple health measures

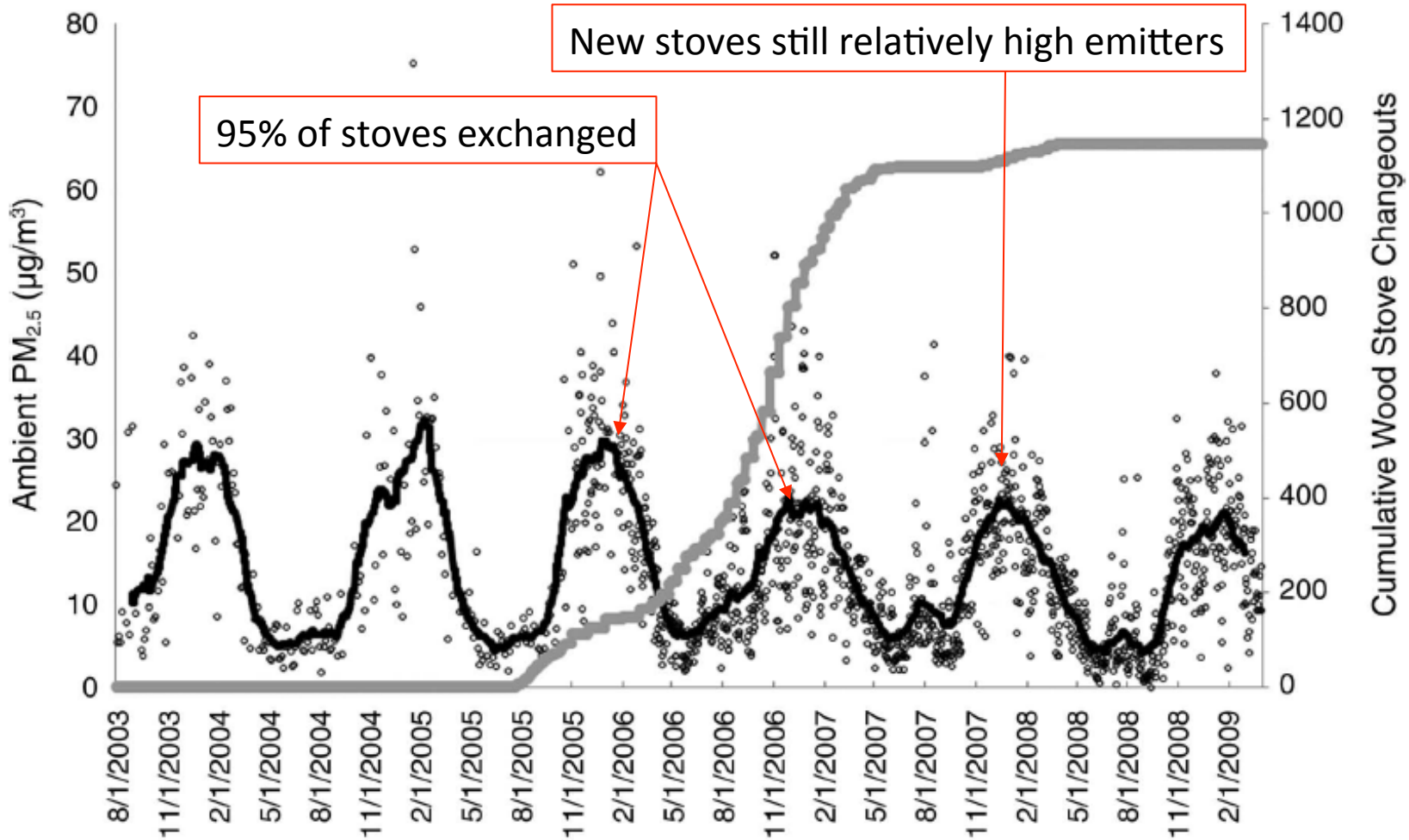
- 32% increase in middle ear infections**
  - Top reason for children < 2 yrs to see physician, be prescribed antibiotics
- 8% increase in bronchiolitis*
  - Top reason for hospitalization of children < 1 yr
- 15% increase in COPD hospitalization*
- 15% increase in SGA birth*
- No associations with:
  - pre-term birth#
  - asthma *incidence*#
  - cardiovascular, COPD mortality#



** > traffic pollution, *~traffic, #<traffic

MacIntyre EA et al., Exposure to residential air pollution and otitis media during the first two years of life. Epidemiology. 2011 Jan;22(1):81-9.; Karr CJ et al.,Influence of ambient air pollutant sources on clinical encounters for infant bronchiolitis. Am J Resp Crit Care Med, 2009, 180(10):995-1001.; Clark NA et al.,Effect of early life exposure to air pollution on development of childhood asthma. Environ Health Perspect 2010, 188(2): 118:284-290; Gan W et al., Associations of Ambient Air Pollution with Chronic Obstructive Pulmonary Disease Hospitalization and Mortality. Am J Resp Crit Care Med. 2013. 187(7):721-7. ; Gehring et al., Epidemiology 2013, in press.

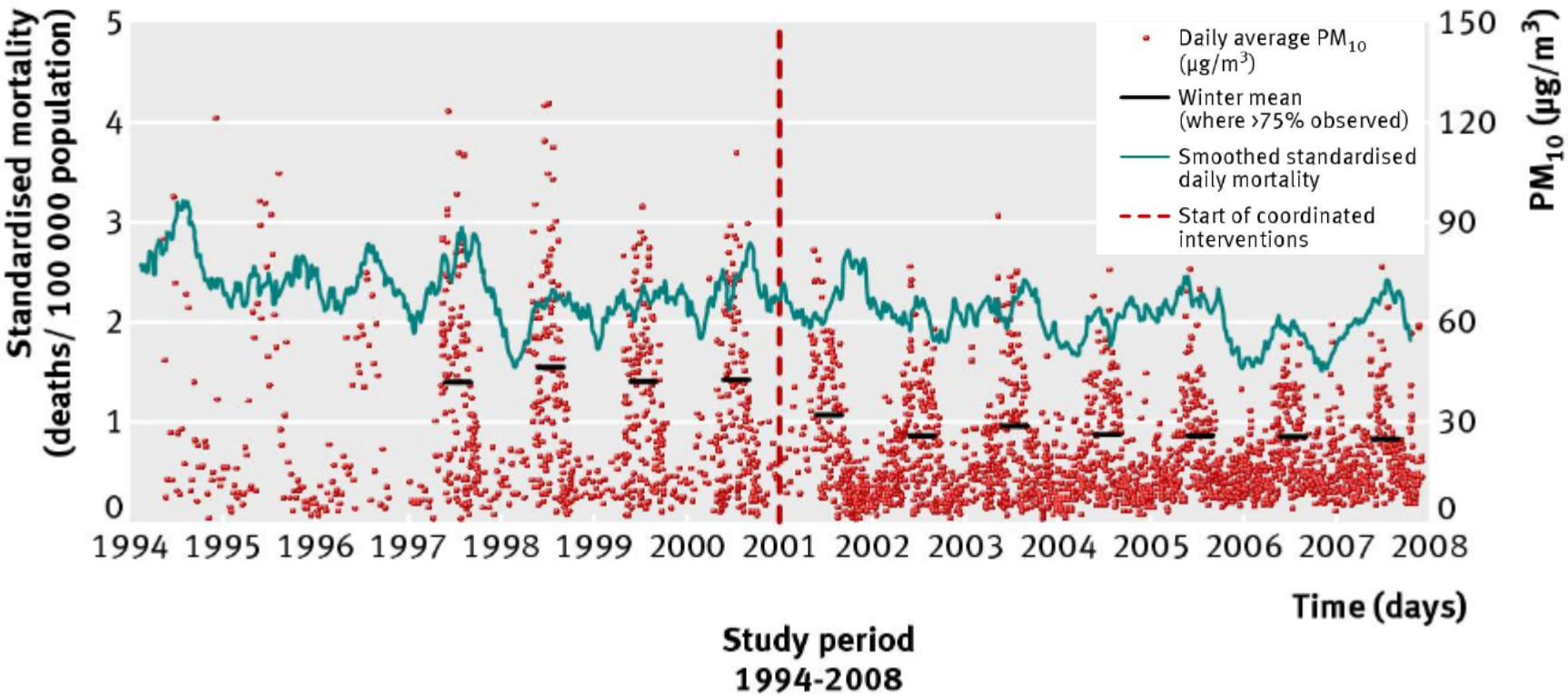
Libby, Montana stove exchange



- ~30% reduction in winter $PM_{2.5}$
- ↓ in childhood wheeze, itchy eyes, sore throat, cold, bronchitis, influenza, throat infections
- School absence associations inconsistent

A rural community intervention targeting biomass combustion sources: effects on air quality and reporting of children's respiratory outcomes. **Noonan CW**, Ward TJ, Navidi W, Sheppard L. Occup Environ Med. 2012

Tasmania woodstove → electricity

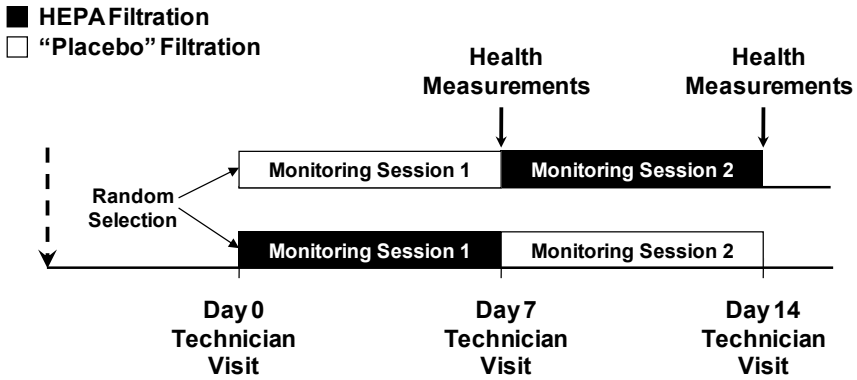


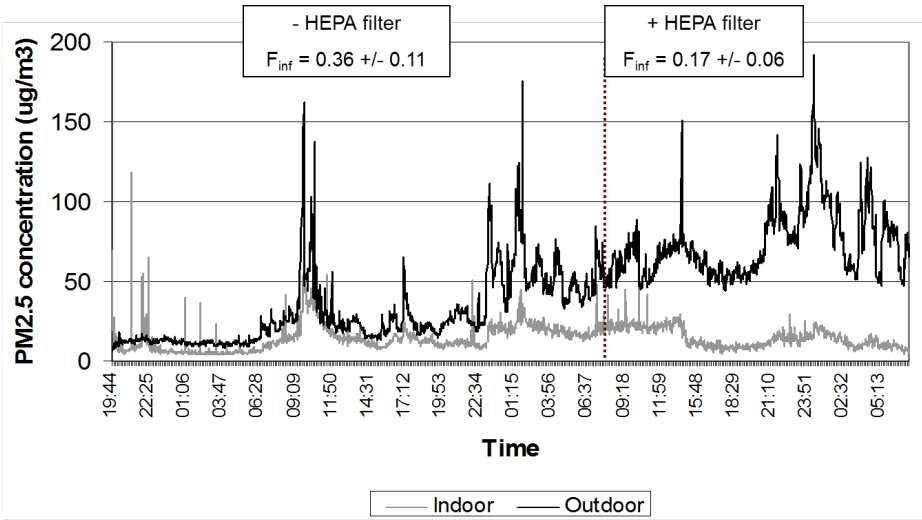
- ~39% reduction in winter PM_{10}
- ↓ winter cardiovascular (-19.6%) and respiratory (-27.9%) mortality
- Similar decreases not observed in control community

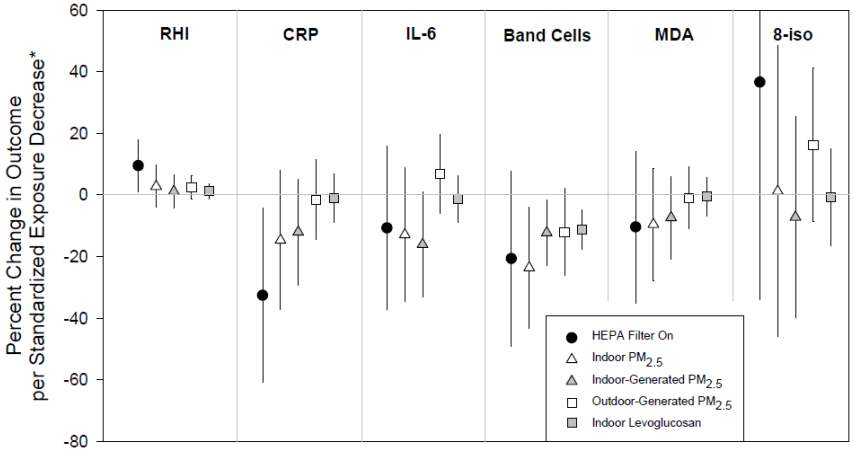
Evaluation of interventions to reduce air pollution from biomass smoke on mortality in Launceston, Australia: retrospective analysis of daily mortality, 1994-2007. Johnston FH, Hanigan IC, Henderson SB, Morgan GG. BMJ. 2013

Air filtration

- Portable HEPA filters 60% ↓ in indoor $PM_{2.5}$
- ↑ endothelial function, ↓ inflammatory markers







11.2 → 4.6 ug/m^3 $PM_{2.5}$
127 → 33 ng/m^3 levoglucosan

An air filter intervention study of endothelial function among healthy adults in a woodsmoke-impacted community. Allen RW, Carlsten C, Karlen B, Leckie S, van Eeden S, Vedal S, Wong I, Brauer M. Am J Respir Crit Care Med. 2011

Towards healthier wood heat & energy

- Location matters!!
  - Distributed sources – high intake fraction
- Health impacts
  - Respiratory disease (+++)
  - Systemic inflammation (+)
  - Cardiovascular disease (+/-)
  - Pregnancy outcomes (?)
- Burn in most controlled manner → derive max energy
  - Wildfires → prescribed fires → Advanced Residential Combustion → Advanced Cogeneration
- Advanced technology
  - Lower mass emissions
  - Different composition → suggestions of lower toxicity

(Some) Key Knowledge Gaps

- Cardiovascular (mortality) impacts?
- Health impacts of short-term peak exposures
- Magnitude of population exposure
- Epidemiological and exposure studies of "advanced" combustion technologies

Thank you!

More questions?

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EXTRA SLIDES

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans **VOLUME 95: INDOOR AIR POLLUTION FROM HOUSEHOLD COOKING AND HEATING: SOLID-FUEL USE AND HIGH-TEMPERATURE FRYING Lyon, France: 10-17 October 2006**

Combustion of biomass

- There is *limited evidence* in humans for the carcinogenicity of household combustion of biomass fuel (primarily wood). Household combustion of biomass fuel (primarily wood) is associated with cancer of the lung.
- There is *limited evidence* in experimental animals for the carcinogenicity of emissions from combustion of wood.
- There is *sufficient evidence* in experimental animals for the carcinogenicity of wood smoke extracts.

- **Overall evaluation:** Indoor emissions from household combustion of biomass fuel (primarily wood) are *probably carcinogenic to humans (Group 2A*).

Pellet stove exposure

- N=19 healthy adults
- 3 hrs (w/exercise) $PM_{2.5}$ = 224 μg/m^3
- ”Incomplete” combustion: emissions dominated by organic carbon
- Mild symptoms. No impact on lung function, FENO
- •Bronchoscopy 24 hrs post-exposure
  - GSH ↑ in BAL
  - No impact on cell counts, airway inflammatory markers
- No inflammation/cardiovascular effects
- Early adaptive protective response.

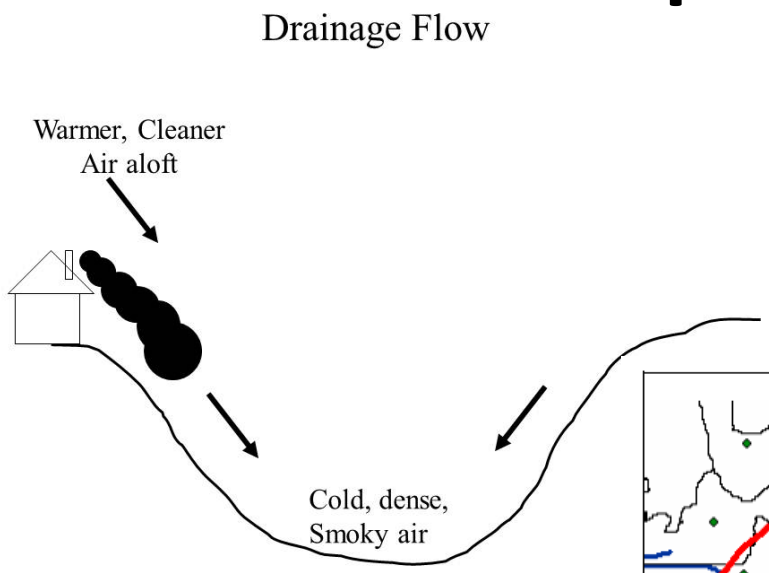
Sehlstedt, M., R. Dove, et al. (2010). "Antioxidant airway responses following experimental exposure to wood smoke in man." Particle and Fibre Toxicology 7(1): 21.

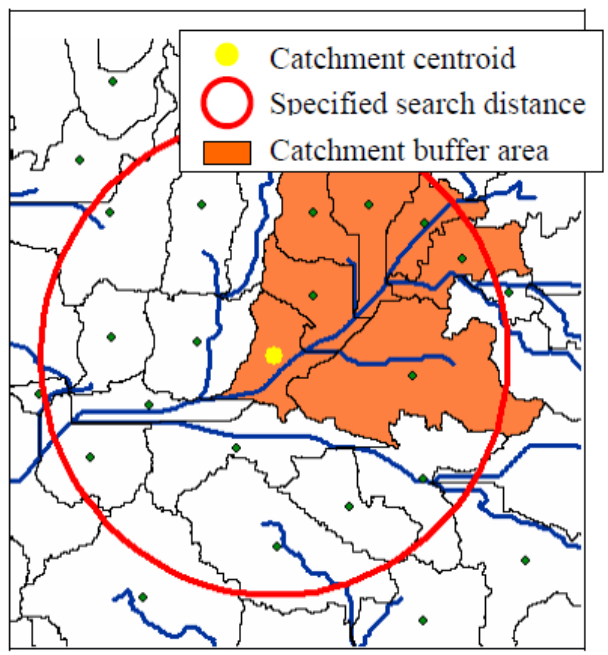
Cardiovascular effects

- Community wildfire exposure (+/-):
  - mixed results for CVD association (mortality, cardiac arrest)
  - high bushfire exposures in Australia: (+)
- Wildland firefighters (+):
  - systemic inflammation
- Controlled exposures: (+/-):systemic inflammation

Swiston JR et al. Acute Pulmonary and Systemic Inflammatory Response Induced by Exposure to wood smoke. European Respiratory Journal, 2008; 32: 129-
Sallsten, G et al. Experimental wood smoke exposure in humans. *Inhal. Toxicol*. 18(11):855–864.; Barregard L et al. Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation. Inhal Toxicol. 2006 Oct;18(11):845-53.; Barregard L et al. Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress.. Occup Environ Med. 2008 May;65(5):319-24;
Allen RW, Carlsten C, Karlen B, Leckie S, van Eeden S, Vedal S, Wong I, Brauer M. An Air Filter Intervention Study of Oxidative Stress, Endothelial Dysfunction, and Inflammation Among Healthy Adults in a Woodsmoke-Impacted Community. American Journal of Respiratory and Critical Care Medicine, 2011. 183, 1222-1230.

Spatial extent





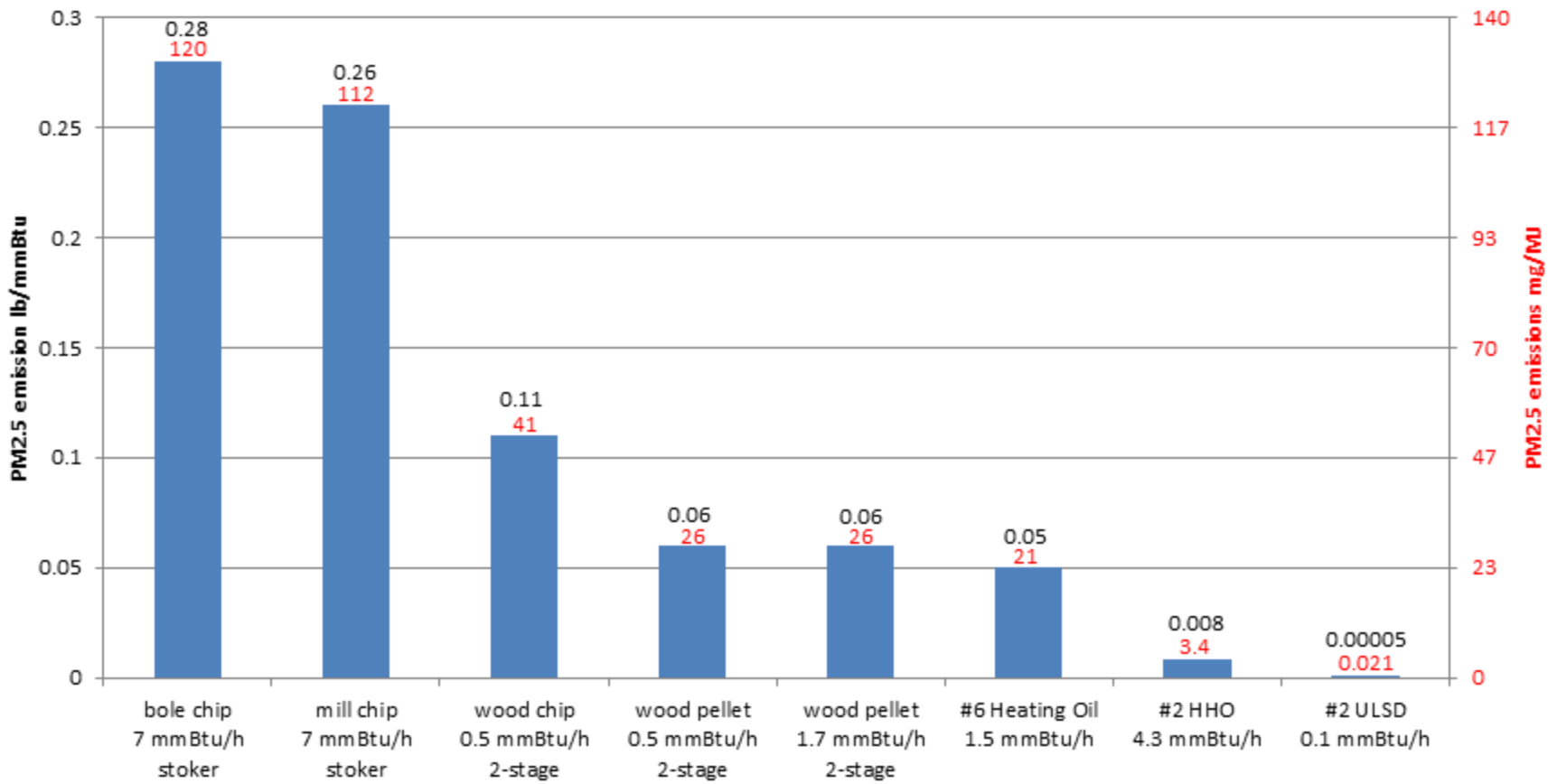
For typical drainage wind speed (1 m/s) maintained over a 3 hour period, upslope influence ~ **10 km**

Catchment modeling[1,2] suggests upslope influence of **4 – 8 km**

Semivariogram analysis[3] suggests spatial extent of **2.7 km**

[1]Larson T, Su J, Baribeau A-M, Buzzelli M, Setton E, Brauer M. A Spatial Model of Urban Winter Woodsmoke Concentrations. Environmental Science and Technology. 2007; 41 (7): 2429 -2436.
[2]Su JG, Allen GA, Miller PJ, Brauer M. Spatial modeling of residential woodsmoke across a non-urban upstate New York region. Air Quality, Atmosphere and Health, 2011 http://dx.doi.org/10.1007/s11869-011-0148-1
[3]Lightowlers, C et al. Determining the spatial scale for analysing mobile measurements of air pollution. Atmospheric Environment 42 (2008)

PM 2.5 emissions input basis



Acknowledgements
Lisa Rector, NESCAUM
Phil Hopke, Clarkson University
Tom Butcher, BNL

nyserda
Energy. Innovation. Solutions.

Source: Ellen Burkhard

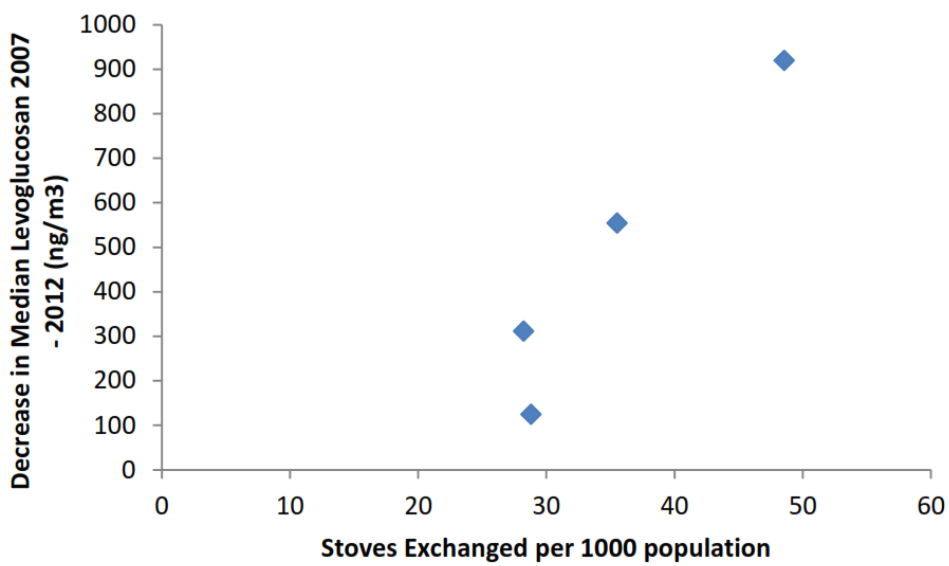
Adapted, from Fay Johnston

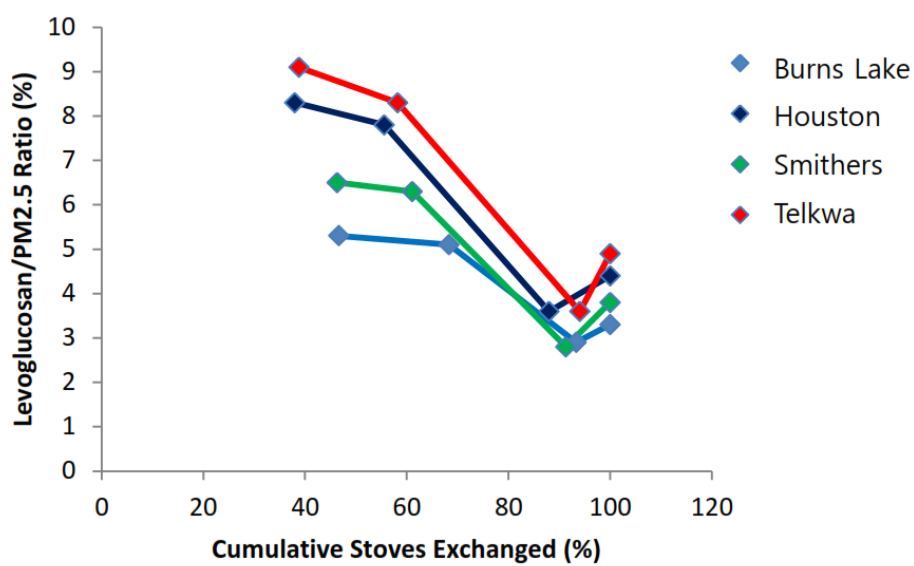
Firesmoke and CVD?

Country	Study	Exposure	Outcome	Result
Australia	LFS project	Bushfire smoke (PM_{10} $PM_{2.5}$, event)	CVD admissions CVD mortality	- inconclusive
Australia	Morgan 2010	Bushfire smoke (PM_{10})	CVD admissions IHD admissions	- inconclusive
Australia	Dennekamp 2010	Bushfire smoke (PM_{10} $PM_{2.5}$, event)	Out of hospital cardiac arrest	+
Brazil	Arbex 2010	Sugar cane burning (TSP harvest vs non-harvest periods)	Admissions for hypertension	+
Canada	Henderson 2011	Forest fire smoke	CVD physician and hospital visits	-
Canada	Moore 2006	Forest fire smoke episode	CVD physician billing	-
Malaysia	Mott 2005	Forest fire smoke episode	CVD admissions	-
Malaysia	Sastry 2002	PM_{10},/visibility	Daily (all-cause/CVD) mortality	+
USA	Delfino 2009	Forest fire smoke ($PM_{2.5}$)	CVD admissions	inconclusive
Canada	Swiston (2007)	Fire fighters: smoke exposure during a shift	Markers of systemic inflammation	+

Cautionary tales – the impact of the economic context

(Bulkley Valley) **W**oodstove **E**xchange **ST**udy





	Spherical organic carbon particles	Soot (elemental carbon aggregates)	Inorganic ash particles
Schematic drawing			
Diameter measured by electron microscopy*	50-600 nm [52, 53]	20-50 nm [52, 73]	50-125 nm [97]
Mobility diameter	100-300 nm [68-70]	50-300 nm [68, 76]	50-125 nm [69, 98, 99]
Internal turbostratic microstructure	No [61]	Yes / No [81-83]	No
Solubility (H_2O)	Depends on ageing [61]	Insoluble	Soluble
Main chemical characteristic	Organic carbon [62, 64, 67] (Most abundant organic compounds: metoxyphenols and monosaccaride anhydrides) [57-60]	Elemental carbon with variable amounts of organics condensed on the surface [12, 62, 81] (Most abundant organic compounds: hydrocarbons and polycyclic aromatic hydrocarbons) [84, 85]	Alkali salts (mainly KCl and K_2SO_4 with small amounts of trace elements (e.g. Zn)) [78, 92]
Combustion conditions	Low-temperature, incomplete combustion [11, 52-56]	High-temperature, incomplete combustion [52]	High-temperature, complete combustion [120]
Possible sources	Air starved combustion or start-up phase of batch wise combustion in conventional stoves, open fireplaces [58,62,64,67]	Combustion in conventional stoves, open fireplaces, boilers for wood, wood chips and pellets [14, 52, 75-79]	Combustion in pellets stoves, boilers for wood, wood chips and pellets [69, 120]

Kocbach Bølling et al. 2009

Experimentally Determined Human Respiratory Tract Deposition of Airborne Particles at a Busy Street

Environ. Sci. Technol. **2009**, *43*, 4659–4664

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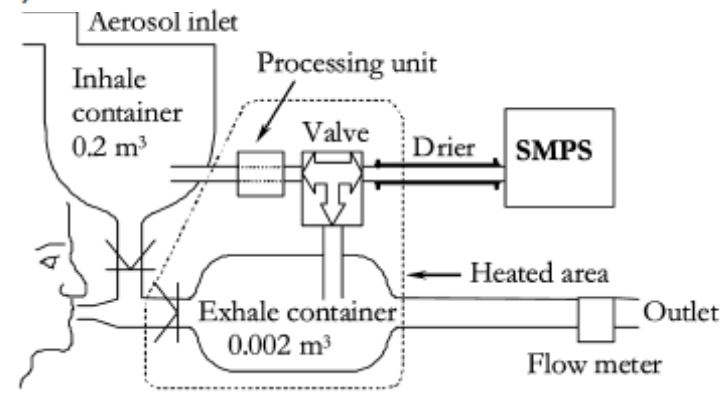

TABLE 2. Total Deposition Fractions ($TDF_{measured}$) and Deposited Dose (In 1-Hour, Inhaled Total Particle Mass Concentration of 100 $\mu g/m^3$) of 12–580 nm Particles from the Curbside of a Busy Street, from Traffic Exhaust and, for Comparison, from Biomass Combustion (Mean Values for All Subjects)[a]

	total deposition fractions ($TDF_{measured}$)			dose/h (if 100 $\mu g/m3$)		
	number	surface area	mass	number ($\times 10^9$)	surface area (mm^2)	mass (μg)
curbside	0.60 ± 0.04	0.29 ± 0.04	0.23 ± 0.05	80 ± 15	610 ± 160	10.8 ± 3.4
traffic exhaust (hydrophobic)	0.68 ± 0.08	0.35 ± 0.03	0.28 ± 0.03	103 ± 15	770 ± 100	12.6 ± 1.8
biomass combustion	0.22 ± 0.07	0.23 ± 0.07	0.24 ± 0.07	6.5 ± 4.5	280 ± 91	11.7 ± 3.7

[a] The traffic exhaust particles are assumed to be the "hydrophobic" fraction of the curbside particles as measured with the H-TDMA. As for the curbside and biomass combustion particles the mass concentration of the traffic exhaust particles is normalized to 100 $\mu g/m^3$. The total deposition fraction and dose for biomass combustion particles was measured in a previous study using the same RESPI setup, but with 10 other subjects (*12*).

- **For equal mass: 16X lower (by number), 3x lower (by surface area) dose/hr, biomass particles relative to traffic exhaust PM**
- (lower deposition probability and lower number/surface area concentration per unit mass)
- **Biomass particles largely water soluble**

…may impact toxicity