

Estimating Community Health Impacts of Increased Residential Biomass Combustion Emissions in Pennsylvania

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Overview

- Motivation
- Process of health impact assessment
- Application to increased residential biomass combustion in PA
- Limitations and uncertainties
- Conclusion

Motivation

- Recent attention to transition to renewable energy economy
 - Greenhouse gas emissions associated with burning fossil fuels
 - Limited reserves will eventually lead to scarcity
- Burning biomass or biomass products offers a source of renewable energy
 - Energy output needs to be larger than *total* energy inputs, including fossil fuel inputs to chemical fertilizers, transportation of resources, etc.

Motivation

- Locally-produced resources more energy efficient
- PA has the agricultural capacity to produce large amounts of biomass
- But what impact would increased biomass combustion emissions have on public health?
 - Composition of biomass combustion emissions and their toxicological properties discussed previously
 - Focus on PM_{2.5}

Framework for health impact assessment

- Standard methodology for PM air pollution discussed by Martuzzi et al. (2003)

- Formula:

$$E = (dRR)(C)(B)(P)$$

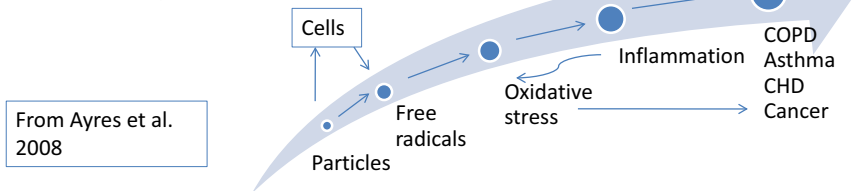
- E is the attributable number of cases for a given health outcome
- dRR is the relative risk of a given health outcome associated with a per unit change in exposure level
- C is the difference in exposure level (same units as dRR)
- B is the rate observed in the population
- P is the population size exposed to C

Objective

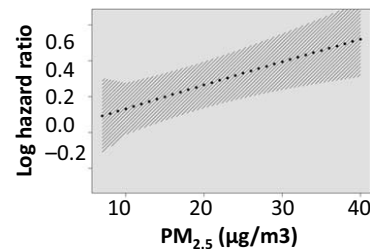
- Identify uncertainties in health impact assessment process
- How many cases would be caused if concentrations were increased by X?
 - Y greater cases observed *if* the association observed in epidemiologic studies is entirely causal and *if and when* the rates observed in the reference population are achieved following increase in exposure

Selected assumptions

- Causality between PM exposure and effect



- Linearity between baseline and elevated dose
 - Supporting evidence accumulating (Schwartz et al. 2008, Pope et al. 2009)
- Reversibility and latency
 - Acute wood-burning PM effects within 2-4 days (Ito et al. 2006); chronic PM_{2.5} mortality effects within 1 year (Puett et al. 2009)



Example application to PA

- What information is available?
 - Population size (P) from US Census
 - Current age-adjusted rates (B) from PA Dept. of Health (EpiQMS; EPHTN) for selected outcomes
 - Difference in exposure (C) from emissions estimates and exposure modeling
 - Relative risks (dRR) from epidemiologic literature on several biomass combustion-related health outcomes, acute and chronic
 - Health outcomes for dRR and B must match

Population at risk (P)

- From US Census
 - PA total population: ~12.4 million persons
 - Assume an additional 30% of PA households begin to operate residential wood combustion, so 3.73 million persons at risk

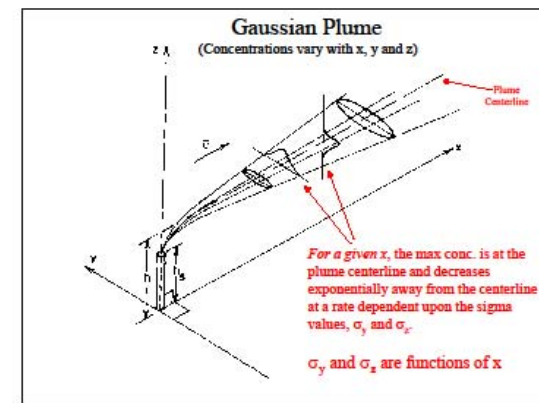
Observed prevailing rate (B)

- Rate in the population exposed to current levels
- Outcomes
 - Acute
 - Cardiovascular deaths per year
 - Asthma hospitalizations per year
 - Chronic
 - Total pneumonia/influenza deaths per year
 - Fatal coronary heart disease (CHD) deaths per year
 - State level (PA) age-adjusted rates for 2006

Exposure increment (C)

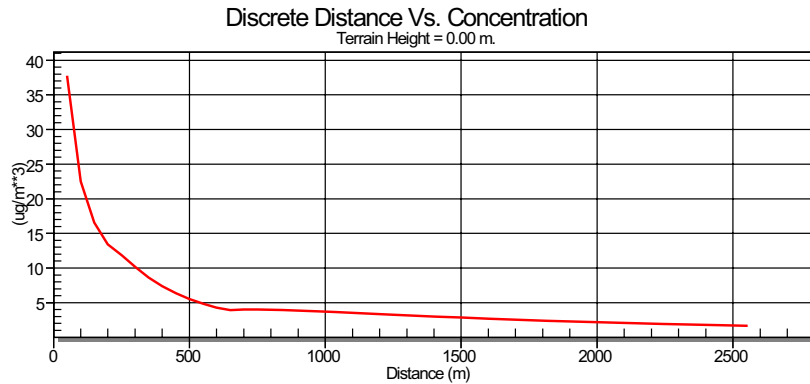
- Typical emissions rate of 52 g PM_{2.5}/hr
 - Using 5H emission factor of 17.3 (g/kg) and 3 kg/hr burn rate from USEPA Residential Wood Combustion report (Houck and Tiegs 1998, p. 20)
- Assumed use:
 - 12 hours/day, 5 months/year
- Emissions in tons/year = 1.05 E-1; 1.44 E-2 g/sec
- Gaussian point-source plume dispersion model
 - Dispersion parameters from screening version of USEPA ISC (SCREEN3)
 - Standard combination of meteorological inputs used
 - Grid of all receptor points within 3 km of point-source (98% decay)
- Increase in outdoor concentrations due to a new point-source at residence
 - Mean within 36 km² impacted area: 0.056 µg/m³

Gaussian dispersion

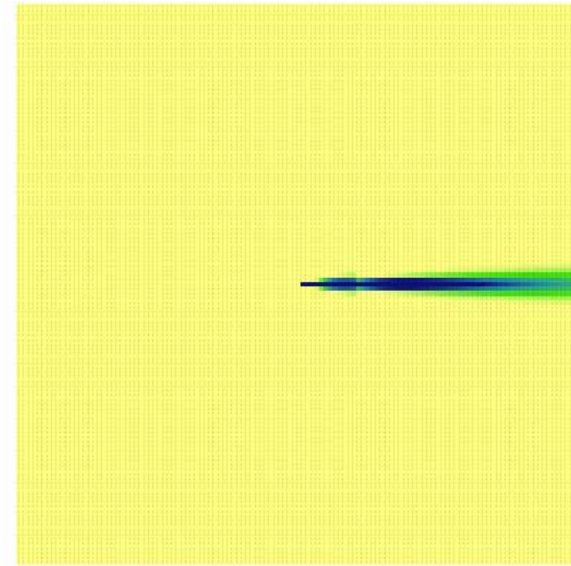


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Decay function (downwind)



Gaussian dispersion on grid (36 km²)



Relative risk (dRR)

- Select 'key study' for each outcome
- Use tracer or biomass source contribution where possible
- Acute
 - Daily cardiovascular mortality and $\text{PM}_{2.5}$ K in CA (Ostro et al. 2007)
 - Asthma hospitalizations and $\text{PM}_{2.5}$ in Seattle, WA (Sheppard et al. 1999)
- Chronic
 - Pneumonia/influenza deaths and annual $\text{PM}_{2.5}$ in the US (Pope et al. 2004)
 - Fatal coronary heart disease (CHD) and annual $\text{PM}_{2.5}$ in the northeast and midwest US (Puett et al. 2009)

Epidemiologic results from key studies

- Reported relative risks converted to dRR per $\mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$
- Ostro et al. 2007: 5.3% increase in CVD deaths
- Sheppard et al. 1999: 0.34% increase in asthma hospitalizations
- Pope et al. 2004 (1999-2000, driven by never smokers): 2.0% increase in pneumonia/influenza deaths
- Puett et al. 2009: 10.2% increase in fatal CHD deaths

Epidemiologic results that support, but don't match

- Sarnat et al. 2008: PM_{2.5} from biomass burning in Atlanta, GA and CVD ED visits: RR 2.7% (95%CI 1.7-3.7) per IQR; dRR of 2.8%
 - Increased respiratory ED visits not detected
- Ostro et al. 2009: PM_{2.5} K and respiratory hospital admissions in children: RR 4% (95%CI, 0.3-7.7) per IQR; dRR of 13%
- Suggestive evidence of increased total non-accidental mortality (Ito et al. 2006; Mar et al. 2006)

Calculation of excess attributable cases

$$E = (dRR)(C)(B)(P)$$

Outcome	dRR	C (µg/m ³)	B (per 1E5)	P (millions)	E
CVD deaths	1.053	0.056	302.5	3.73	643
Asthma hospitalizations	1.0034		951.0		1,985
Pneumonia/ influenza deaths	1.02		18.2		39
CHD deaths	1.102		154.8		352

Conclusions

- If 30% of PA households began to burn wood in small combustion appliances, at the assumed emission rate and dispersion characteristics, the expected number of additional cases per year would be:
 - 643 additional CVD deaths
 - 1,985 additional asthma hospitalizations
 - 39 additional pneumonia/influenza deaths
 - 352 additional CHD deaths

Limitations

- Uncertainties in:
 - Emission rates
 - Exposure modeling
 - All susceptible subpopulations captured?
 - Ostro et al. 2009: PM_{2.5} associated with respiratory hospital admissions in children (dRR: 1.13)
 - Respiratory hospital admissions data by age not currently available for PA but may be important
 - Generalizability of epidemiologic results
 - Only recent US studies used

Future work

- Explore effects of uncertainties
 - Confidence limits and error propagation
 - Monte-carlo analysis?
- Expand current approach
 - To the county level to identify 'hot-spots' due to large numbers cases
 - To include other pollutants: CO, benzene, acrolein, PAHs, etc.
 - To other source types with differing emission characteristics

Questions

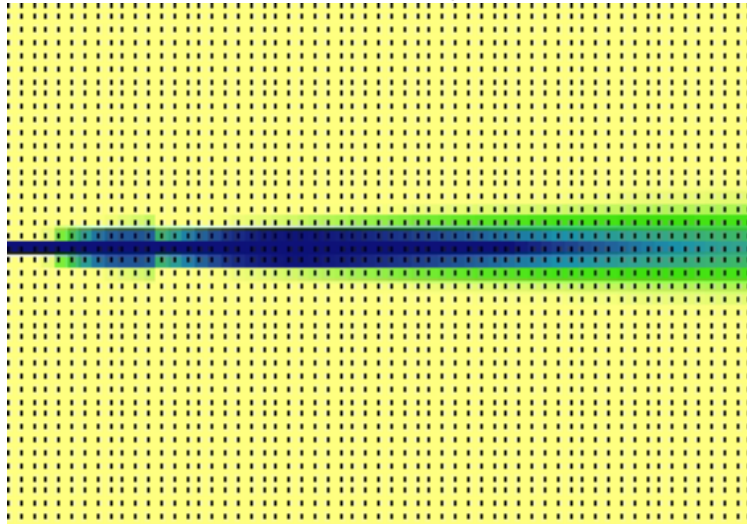
USEPA SCREEN3 model point-source inputs

- Emission rate = 0.0144 g/sec
- Stack height = 3 m
- Stack inside diameter = 0.5 m
- Stack gas exit velocity = 2 m/s
- Stack gas temperature = 394.3 K (250 deg F)
- Ambient temperature = 293 K (67.7 deg F)
- Receptor height above ground = 2 m
- Urban/rural option = Rural

Questions about assessing PM_{2.5} health effects

- Is PM_{2.5} mass appropriate indicator of biomass combustion emissions?
 - If so, is a linear dose-response appropriate?
 - If not, what is appropriate threshold?
- What are relevant health endpoints?
 - Does this adequately protect susceptible subpopulations such as asthmatics/children?

Gaussian dispersion on grid (36 km²) close-up



Formula for converting to dRR

- $dRR \text{ (per } \mu\text{g/m}^3) = (RR - 1) / \Delta C$
- $PM_{2.5} \text{ K to } PM_{2.5} \text{ total mass ratio of 25.5 from Sarnat et al. 2008 used to convert } PM_{2.5} \text{ K to } PM_{2.5}$

Caveat

- The attributable number of cases, E, may not be fully realized, though, due because of the assumption of full causality
- If, instead, the exposure-disease association takes place through intermediate steps, wherein addition of one factor in the chain results in only a limited increase in the following factor, E may overestimate (adapted from (Martuzzi et al. 2003))