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 km$^2$ impacted area: 0.056 µg/m$^3$

Gaussian dispersion

From Guenther class notes, LETE
Decay function (downwind)

Relative risk (dRR)

- Select ‘key study’ for each outcome
- Use tracer or biomass source contribution where possible
- Acute
  - Daily cardiovascular mortality and PM$_{2.5}$ K in CA (Ostro et al. 2007)
  - Asthma hospitalizations and PM$_{2.5}$ in Seattle, WA (Sheppard et al. 1999)
- Chronic
  - Pneumonia/influenza deaths and annual PM$_{2.5}$ in the US (Pope et al. 2004)
  - Fatal coronary heart disease (CHD) and annual 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 µg/m$_3$ 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

\[ E = (dRR)(C)(B)(P) \]

<table>
<thead>
<tr>
<th>Outcome</th>
<th>dRR</th>
<th>C (µg/m$^3$)</th>
<th>B (per 1E5)</th>
<th>P (millions)</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVD deaths</td>
<td>1.053</td>
<td></td>
<td>302.5</td>
<td></td>
<td>643</td>
</tr>
<tr>
<td>Asthma hospitalizations</td>
<td>1.0034</td>
<td>0.056</td>
<td>951.0</td>
<td></td>
<td>1,985</td>
</tr>
<tr>
<td>Pneumonia/influenza deaths</td>
<td>1.02</td>
<td>18.2</td>
<td>3.73</td>
<td></td>
<td>39</td>
</tr>
<tr>
<td>CHD deaths</td>
<td>1.102</td>
<td></td>
<td>154.8</td>
<td></td>
<td>352</td>
</tr>
</tbody>
</table>

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

- \( dRR \) (per \( \mu g/m^3 \)) = \( (RR - 1) / \Delta C \)

- \( PM_{2.5} \) K to \( PM_{2.5} \) total mass ratio of 25.5 from Sarnat et al. 2008 used to convert \( PM_{2.5} \) 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)