An overview of health impacts from biomass combustion

“Bioenergy Emissions and Health Impacts”
Short Course

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Overview

• Brief characterization of biomass combustion emissions (BCE)
• Summary of epidemiologic data for particulate matter (PM) acute and chronic health effects
Pollutant emissions

• Result from partially-oxidized fuel
• Emission rates depend on combustion conditions
  – Combustion device, fuel type (gross wood, pellets, chips, etc.), fuel moisture content, combustion air, etc.
• Much of existing health-related BCE literature:
  – Focuses on residential wood burning as well as wildland and prescribed fire
  – Limited comparability of emission rates due to:
    • Lack of available standards
    • Validation of analyte detection
Air pollutants in wood smoke/BCE

• Gases and particles (including condensates of particle precursor gases; in BCE, alkali salts such as KOH or KCl enriched)

• Gases
  – CO, NO$_x$
  – Many respiratory irritants: aldehydes, phenols, VOCs, others

• Particles
  – Differentiated by size, which influences relevance to health
    • Composition also important
  – EC, OC: benzo[a]pyrene, other PAHs and oxygenated organics (tar)

• Some of these ‘criteria’ air pollutants
  – PM$_{2.5}$ considered best single indicator of health effects of combustion emissions

From Naeher et al. 2007
Size distribution of biomass smoke particles

Figure 2.2. Size distribution of woodsmoke and dungsmost particles. Measurements taken in the East–West Center simulated village house as reported in Smith et al. (1984b). (Figure prepared by Premlata Menon.)
More recently

Particle size distribution

Figure 2. Steady-state ultra-fine particle number size distributions for emissions from 0.5 mmBtu pellet boiler, 0.5 mmBtu chip boiler, and 1.7 mmBtu pellet boiler (Chandrasekaran, et al., 2011).

From E. Burkhard, NYSERDA
Toxic compounds in ‘biomass fuel smoke’ from incomplete combustion

• Hydrocarbons
  – 25+ saturated hydrocarbons such as $n$-hexane
  – 40+ unsaturated hydrocarbons such as 1,3 butadiene
  – 28+ mono-aromatics such as benzene & styrene
  – 20+ polycyclic aromatics such as benzo(a)pyrene

• Oxygenated organics
  – 20+ aldehydes including formaldehyde & acrolein
  – 25+ alcohols and acids such as methanol
  – 33+ phenols such as catechol & cresol
  – Many quinones such as hydroquinone
  – Semi-quinone-type and other radicals

From Naehler et al. 2007
Deposition
One possible mechanism illustrated

- **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)
BCE exposures in other settings

• From biomass cookstoves
  – CO an obvious concern, also PM$_{2.5}$
  – Transition metal content low (Fullerton et al. 2009)
Potential health effects of air pollutants in BCE

- Asphyxia (due to CO)
- Airway irritation, pulmonary inflammation and oxidative stress, lowered pulmonary function ($\text{FEV}_1$)
- Immunotoxic effects (increased URI/LRI)
- Mutagenic/carcinogenic/teratogenic effects
- Neurotoxicity
- Others: allergic responses, cardiovascular effects due to vasoconstriction

“Fine particles ... efficiently evade the mucociliary defense system and are deposited in the peripheral airways, where they may exert toxic effects”

From Naeher et al. 2007
From Urch et al. 2004
Broad statement on the respiratory effects of BCE

– “The subacute inflammatory reactions caused by recurrent exposure to irritant and cilia-toxic and mucus-coagulating emissions make the trachea, bronchi, and bronchioles, especially in infants, susceptible to infection, which may manifest itself as acute infective bronchitis, bronchiolitis, or pneumonia.” – de Koning et al. 1985

– Open question: How likely are these effects at the levels emitted by current ICI technology?
Indoor pollution concentrations from typical woodfired cookstove during cooking

Wood: 1.0 kg Per Hour in 15 ACH 40 m³ kitchen

- Carbon Monoxide: 150 mg/m³ (10 mg/m³)
- Particles: 3.3 mg/m³ (0.1 mg/m³)
- Benzene: 0.8 mg/m³ (0.002 mg/m³)
- 1,3-Butadiene: 0.15 mg/m³ (0.1 mg/m³)
- Formaldehyde: 0.7 mg/m³ (0.1 mg/m³)

Indoor levels

Typical standards to protect health

International Agency for Research on Cancer (IARC) Group I Carcinogens

From Smith 2007
PM 2.5 emissions input basis

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From E. Burkhard, NYSERDA
USEPA National ambient air quality standards (NAAQS)

- Identify air pollutants anticipated to endanger public health
- Issue air quality criteria which accurately reflect latest scientific knowledge of kind and extent of all identifiable effects on public health or welfare
- Define primary standards based on health impacts allowing an adequate margin of safety
- Review every five years

From Dockery 2012
PM$_{2.5}$ NAAQS

- Current standard
  - 24-hr 35 µg/m$^3$; Annual 15 µg/m$^3$
- 2010 CASAC recommendation
  - Annual from 15 to 11-13 µg/m$^3$
- This may impact proposed new sources by reducing the ‘room-to-pollute’
  - “Largest estimated benefits of all federal regulations attributable to a reduction in a single air pollutant: PM$_{2.5}$”

2007 OMB Report to Congress on the Benefits and Costs of Federal Regulations and Unfunded Mandates on State, Local, and Tribal Entities

PM$_{2.5}$ Air Quality Analyses Memo, July 22, 2010
PM$_{2.5}$ Annual Standard
15 $\mu$g/m$^3$

DESIGNATED PM-2.5 NONATTAINMENT AREAS (39)
UNDER CLEAN AIR ACT AMENDMENTS OF 1990
AS OF AUGUST 30, 2011
1997 Standard
PM$_{2.5}$ Annual Standard

13 $\mu$g/m$^3$

VIOLATIONS BASED ON 2001 - 2003 PM2.5 DATA USING MSAs

Source: Based upon U.S. EPA data interpreted by A.S.L. & Associates, Helena, MT

10/2004
New BCE-source policy issues

• Siting
  – Measure ‘background’ PM$_{2.5}$ concentration
  – Subtracting this from NAAQS gives ‘room-to-pollute’

• Should we instead incentivize minimizing emissions?
  – Minimum technology standards?
  – Continuous emissions monitoring (e.g., oxygen sensors in effluent)?
  – Automatic switchover to conventional fuel?
Acute PM$_{2.5}$ air quality standards

• Health impacts recognized as early as 1952 London smog incident
  – Five-fold increase in death rates noted during an air pollution episode lasting four days
• Many time-series studies document effects of acute PM$_{2.5}$ exposure on mortality, respiratory, and cardiovascular outcomes
  – APHEA (2000): 0.6% increase in total mortality per 10 units elevation in PM$_{10}$ concentrations
  – NMMAPS (Dominici et al. 2002): 0.21% increase in total mortality per 10 units elevation in PM$_{10}$ concentrations
Annual PM$_{2.5}$ air quality standards

- Elevated health risks of exposure to ambient PM$_{2.5}$, even after controlling for individual risk factors such as smoking and SEP
  - PM$_{2.5}$ associated with mortality (1.26 (95%CI: 1.08-1.47), H vs. L)
  - Elevated risks for lung cancer and cardiopulmonary disease but not other causes considered together
  - Lower risks for other air pollutants, sulfates similar
  - Confirmed Six Cities findings in a large cohort, dispersed across US
  - Exposures estimated as averages across metropolitan area
  - Each 10-µg/m$^3$ elevation in PM$_{2.5}$ associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality. Coarse particles and TSP were not consistently associated with mortality
  - Among cardiovascular effects, ischemic heart disease, dysrhythmias, heart failure, and cardiac arrest strongest. Fine PM was associated with 8% to 18% increases in mortality risk for cardiovascular causes
Other studies

• ACS (Jerrett et al. 2005)
  – PM$_{2.5}$ associated with all-cause mortality (RR 1.11 (95%CI: 0.99-1.25)) in Los Angeles, CA, USA
  – Risks of IHD and lung cancer also elevated (RR 1.25 (95%CI: 0.99-1.59), 1.20 (95%CI: 0.79-1.82))
  – Within-city effects nearly 3 times larger than between-city

• WHI (Miller et al. 2007)
  – Higher risks for PM$_{2.5}$ than previous studies: cardiovascular event (RR 1.24 (95%CI: 1.09-1.41)), cardiovascular death (RR 1.76 (95%CI: 1.25-2.47))
  – Also higher within-city effects; only in women
  – Used nearest year 2000 PM$_{2.5}$ monitor data as exposure surrogate
Validity concerns

• Exposure issues
  – Ambient data from one time period as exposure surrogates
    • Nearest monitor
    • Community-average
    • Poor monitoring coverage over space and time, especially for PM$_{2.5}$
  – Within-city gradients in exposure
    • PM varies within and between cities
    • Larger health effects for within community comparisons than between (Jerrett et al. 2005, Miller et al. 2007)
  – Limited to areas nearby monitors
  – Regional spatial patterns in outdoor concentrations change over time
Spatio-temporal model with monthly smooth spatial terms and GIS and meteorological covariates combined provided greater spatial resolution at regional and local scales.
Epidemiological results: PM$_{2.5}$

- Hazard ratios (95% CI’s) for each 10 µg/m$^3$ change in 12-month average modeled PM$_{2.5}$

<table>
<thead>
<tr>
<th>Category</th>
<th>Cases</th>
<th>Crude models</th>
<th>Fully-adjusted single pollutant</th>
<th>Fully-adjusted multi-pollutant</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause mortality</td>
<td>3,785</td>
<td>1.45 (1.19-1.78)</td>
<td>1.26 (1.02-1.54)</td>
<td>1.29 (1.03-1.62)</td>
</tr>
<tr>
<td>First CHD</td>
<td>1,348</td>
<td>1.19 (0.85-1.65)</td>
<td>1.11 (0.79-1.55)</td>
<td>1.10 (0.76-1.60)</td>
</tr>
<tr>
<td>Fatal CHD</td>
<td>379</td>
<td>2.29 (1.26-4.18)</td>
<td>2.02 (1.07-3.78)</td>
<td>2.13 (1.07-4.26)</td>
</tr>
<tr>
<td>Nonfatal CHD</td>
<td>854</td>
<td>0.76 (0.50-1.15)</td>
<td>0.73 (0.48-1.12)</td>
<td>0.71 (0.44-1.13)</td>
</tr>
</tbody>
</table>

Fully-adjusted: Crude + smoking status, family history of MI, BMI, hypercholesterolemia, diabetes, hypertension, median family income and median house value in census tract of residence, and physical activity

- Lower risks for nearest monitor than model-predicted PM$_{2.5}$: all-cause (1.35 (1.08-1.69)) and fatal CHD (1.47 (0.73-2.99))

From Puett et al. 2009
Health benefits

Life Expectancy vs PM$_{2.5}$
1980-2000

EPA required to periodically review NAAQS

Health benefits below current NAAQS

Pope, Ezzati, Dockery (NEJM 2009)
Recent epidemiologic findings for chronic PM$_{2.5}$ exposure

- Expanded exposure models to continental US
- Chronic exposure to PM$_{2.5}$ may also have effects on cognitive decline (Weuve et al. 2012)
Conclusions

• Many constituents of BCE impact public health
  – Large body of epidemiologic evidence for PM$_{2.5}$ health impacts
    • Improved exposure estimates indicate higher mortality risks from PM$_{2.5}$
      – Observed in several studies
      – Lends confidence to a causal relationship
    • Toxicological data and that from controlled human exposures offer support, discussed in detail later
  – Also for other toxic compounds- benzene, PAHs, CO, formaldehyde, benzo[a]pyrene, etc.
Thank you