Health Effects of Wood Combustion Aerosols: A Review

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Abstract

The study surveys investigations on health effects of particulate matter (PM) from biomass combustion. While specific health effects from wood combustion particles remain difficult to identify by epidemiological methods, adverse effects such as oxidative stress, inflammation, cytotoxicity, and genotoxicity are reported from in vivo and in vitro tests with exposure to wood smoke or biomass particles. Further, the combustion type is identified as an important factor which can influence specific health effects. While incomplete or low oxygen combustion is found as a signature characteristic for increased health impact, an assessment on the specific influence of the particle size cannot be asserted from this review. Since current investigations mainly focus on wood smoke, the total effect of biomass flue gases in real-life conditions cannot be accurately assessed due to limited information on the potential of secondary organic aerosols (SOA) and health effects from these species.

Keywords: Biomass combustion, wood combustion, particulate matter, health effects, oxidative stress, inflammation, cytotoxicity, genotoxicity, epidemiology

1 Introduction

Smoke from biomass combustion is identified as an important cause of premature deaths being more important than Malaria and Tuberculosis [IEA 2010].

2 Sources of PM from wood combustion

Wood combustion exhibits three different types of PM. Depending on the combustion regime, oxygen availability, and temperature, salts are formed at near-complete combustion conditions, soot is formed in high temperature zones with lack of oxygen, and Condensable Organic Compounds (COC) are formed at low temperature combustion. While wood combustion in technical appliances is important in European countries, open field burning, three-stone-cooking fires and simple cooking devices are widely applied in developing countries.

3 Methods to determine health effects

The present report provides a survey on investigations on health effects related to biomass combustion with focus on studies performed since 2000. Although health effects can be expected from different groups of pollutants, particulate matter (PM) is assumed to play the dominant role together with organic compounds. To enable a comprehensive interpretation of results from different studies, the methods are being categorized in different groups as follows:
1. **In vitro exposure studies**
   – on animal cells
   – on human cells
2. **In vivo exposure studies**
   – on animals
   – on humans
3. **Epidemiologic studies**
   – available only in humans

4 Results

4.1 In vitro exposure

Different in vitro studies exhibit effects on inflammation, cytotoxicity, e.g. [Kocbach et al. 2008]. Increasing health effects were found for particles from incomplete combustion with increasing cytotoxicity and genotoxicity from salt to soot to COC [Klippel & Nussbaumer 2006]. Recent investigations were performed on particles from different combustion devices such as old log wood boiler, old wood stove, modern log wood boiler, pellet boiler, and wood chip boiler by [Kelz et al. 2010]. The results confirm increased toxicity of particles from simple combustion devices operated at high concentrations of carbon monoxide and hydrocarbons. [Jalava et al. 2010] compared particles from normal conditions and smoldering conditions, which also confirmed higher apoptotic, inflammatory, and cytotoxic responses for combustion operated with low oxygen.

4.2 In vivo exposure

In vivo exposure studies are performed with humans or animals in the following type of investigations:

<table>
<thead>
<tr>
<th></th>
<th>Humans</th>
<th>Animals</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Questionnaire Responses</td>
<td>-</td>
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</tr>
<tr>
<td>5. Genotoxicity</td>
<td>+</td>
<td>+</td>
</tr>
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</table>

Examples:
1. Questionnaire responses after wood smoke exposures did not reveal significant effects [Riddervold et al 2011; Sällsten et al. 2006].
2. Inflammation effects by Serum Amyloid A (SAA), a rapid responder to acute inflammation, were found by [Baaregard et al. 2006].
3. [Park et al. 2004] found increased oxidative stress after wood smoke exposure in sheep lungs.
4. Different effects on respiratory function were found by [Kou & Lai 1994].
5. [Danielsen et al. 2008] report results on genotoxicity in blood cells and urine.
4.3 Epidemiology

Epidemiology investigations cover indoor cooking with biomass [Baumgartner et al 2011; Regalado et al 2006] and wildfire events [Künzli et al 2006].

[Regalado et al. 2006] measured PM$_{10}$ exposure as function of the cooking system and found high exposure for biomass cooking (i.e. 1 to 2.5 mg/m$^3$), both with and without flue.

[Milijevic et al. 2010] investigated the effect of combustion temperature on Reactive Oxygen Species (ROS) in a wood stove and found that low combustion temperature leads to high ROS, while above 650°C, ROS concentration were low.

Further epidemiological investigations evaluated respiratory and other effects. Significant effects with odds ratios OD up to 2.5 were found for eye irritation [Künzli et al 2006] and cancer [Pintos et al 1998; Sapkota et al 2007].

5 Conclusions

There is a relevant number of in vitro and in vivo investigations on wood PM.

The majority of the tests reveal adverse health effects which are statistically significant for:

1. Cytotoxicity
2. Oxidative stress
3. Inflammation
4. Physiological responses
5. Genotoxicity and carcinogeneity.

Trends for increasing health relevance are:
- Salt < soot < condensable organic compounds
- decreasing combustion temperature
- lack of oxygen
- worse combustion design.

Consequently, incomplete combustion PM is most relevant for health and high combustion quality is crucial.

Epidemiology on wood PM is limited, however confirms health effects by eye irritation and cancer. In addition to sampled PM, Secondary Organic Aerosols from wood smoke need to be considered. Information on health effects considering SOA is lacking.

Acknowledgments

- Federal Office for the Environment (BAFU)
- Prof. Dr. Barbara Rothen-Rutishauser, University of Fribourg
6 References


Health Effects of Wood Combustion Aerosols

A Literature Review on behalf of the Swiss Federal Office for Environment

Kelvin Fong
Thomas Nussbaumer

Verenum Research
SWITZERLAND

16th ETH-Conference on Combustion Generated Nanoparticles
1. Introduction
2. Sources of PM from wood combustion
3. Methods to determine health effects
4. Results
   4.1 In vitro exposure
   4.2 In vivo exposure
   4.3 Epidemiology
5. Conclusions
Global Situation:
Premature annual deaths from biomass smoke

Situation in Switzerland

3700 deaths/year due to $\text{PM}_{10}$ of which

16% comes from wood combustion

[BAFU 2005] and [UVEK 2006]
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Sources of PM from wood combustion

3 types of primary aerosols:
- Solid Particles & Condensables
  - Salts
  - Soot
  - COC

Gas phase emissions:
- VOC
- CO
- CO₂
- H₂O

SOA

[Schmid]
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Methods to determine health effects

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Inflammation (a, b, c) and Cytotoxicity (d) after 40 hours at 40 µg/cm²

[Kocbach et al 2008]
Cytotoxicity on lung cells from Chinese hamster

[Graph showing cell survival (%)]

- Diesel soot
- PM from badly operated wood stove
- PM from automatic boiler

[Verenum]

[Klippel & Nussbaumer, 10th ETH Nanoparticle Conference 2006]
Genotoxicity by chromosome aberration

![Graph showing cell survival vs. particle concentration in cell medium for different sources of nanoparticle exposure, with Diesel soot and wood stove with bad firing conditions highlighted.]

[Klippel & Nussbaumer, 10th ETH Nanoparticle Conference 2006]
Comparison of $\text{PM}_1$ from different combustion types

Good combustion:
- Pellet boiler
- Wood chip boiler

10 x CO and VOC:
- Wood stoves
- Modern log wood boiler
- Tiled stove

100 x CO and VOC:
- Old log wood boiler

<table>
<thead>
<tr>
<th>Combustion system</th>
<th>Test run</th>
<th>$\text{O}_2$ vol% d.b.</th>
<th>CO mg/MJ</th>
<th>OGC mg/MJ</th>
<th>$\text{PM}_1$ mg/MJ</th>
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<td>PE-m</td>
<td>1</td>
<td>12.6</td>
<td>47.1</td>
<td>2.5</td>
<td>6.2</td>
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<td>12.5</td>
<td>45.4</td>
<td>1.7</td>
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<td>WC-m</td>
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<td>168.1</td>
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<td>12.1</td>
<td>182.2</td>
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<td>13.6</td>
</tr>
<tr>
<td>LW-m</td>
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<td>8.6</td>
<td>700.4</td>
<td>78.7</td>
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<tr>
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<td>793.1</td>
<td>62.4</td>
<td>17.6</td>
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<td>LW-o</td>
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<td>12,632.3</td>
<td>1,143.8</td>
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<td>8,969.4</td>
<td>650.8</td>
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<td>ST-m</td>
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<td>ST-o</td>
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<td>11.1</td>
<td>2,084.6</td>
<td>185.7</td>
<td>55.5</td>
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<td>TST-m</td>
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<td>15.4</td>
<td>1,207.3</td>
<td>52.4</td>
<td>31.3</td>
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<td>15.3</td>
<td>1,007.5</td>
<td>69.2</td>
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</table>

PE = Pellet
WC = Wood chips
LW = Log wood boiler
ST = Stove
TST = Tiled stove
-m = modern
-o = old

[Kelz et al. 2010]
Cytotoxicity by cell membrane permeability exposed to PM$_1$ samples

PE  = Pellet
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[Kelz et al. 2010]
Apoptotic, inflammatory, and cytotoxic responses for Normal Conditions (NC) versus Smoldering Conditions (SC) and for two size fractions of PM

<table>
<thead>
<tr>
<th></th>
<th>Apoptosis</th>
<th>MIP-2</th>
<th>TNFα</th>
<th>Cytotoxicity</th>
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<tr>
<td><strong>per mg PM</strong></td>
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<tr>
<td>NC</td>
<td>PM&lt;sub&gt;1–0.2&lt;/sub&gt;</td>
<td>4.8</td>
<td>2.4</td>
<td>2.0</td>
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<tr>
<td></td>
<td>PM&lt;sub&gt;0.2&lt;/sub&gt;</td>
<td>1</td>
<td>1</td>
<td>1</td>
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<tr>
<td>SC</td>
<td>PM&lt;sub&gt;1–0.2&lt;/sub&gt;</td>
<td>6.7</td>
<td>5.7</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>PM&lt;sub&gt;0.2&lt;/sub&gt;</td>
<td>6.7</td>
<td>2.0</td>
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<td><strong>per MJ</strong></td>
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<td>NC</td>
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<tr>
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<td>1</td>
<td>1</td>
<td>1</td>
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<tr>
<td>SC</td>
<td>PM&lt;sub&gt;1–0.2&lt;/sub&gt;</td>
<td>33</td>
<td>28</td>
<td>7.9</td>
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<tr>
<td></td>
<td>PM&lt;sub&gt;0.2&lt;/sub&gt;</td>
<td>15</td>
<td>4.3</td>
<td>2.3</td>
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[Jalava et al. 2010]
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# In vivo exposure studies

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<td>5. Genotoxicity</td>
<td>+</td>
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</tr>
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</table>
1. Questionnaire responses in human exposure studies:

Experiment Exposure Chamber used for Human Exposures

Exposure to 250 μg/m³ for 4 h:
No significant effect in questionnaire responses

[Riddervold et al. 2011; Sällsten et al. 2006]
2. Inflammation in human exposure studies: Differences in Serum Amyloid A (SAA)* concentrations before and after exposure of humans to clean air to and wood smoke.

*An inflammatory marker. Rapid responder to acute inflammation, mainly expressed in the liver.

hours after 4 hours of exposure to clean air and wood smoke (279 and 243 μg/m³)
3. Oxidative stress in animal exposure studies
Myeloperoxidase activity in sheep lung lobes after smoke exposure

Mild exposure = 175 s
Moderate = 350 s
Severe = 560 s

[Park et al. 2004]
4. Physiological responses by animal exposure studies with rats
Mean initial respiratory responses after inhalation of smoke

Apneic duration = period of no breathing between breaths: Effect: x 2

Ratio between tidal volume ($V_t$) after exposure divided by $V_t$ before exposure: Effect: x 3

[Image: Graphs showing effects of smoke concentration on apneic duration and tidal volume ratio]

[Kou & Lai 1994]
5. Genotoxicity
by effects on mRNA levels
in human blood cells (PBMC) and markers of genotoxicity
following 4 hours of wood smoke exposure

<table>
<thead>
<tr>
<th></th>
<th>Time after exposure to filtered air</th>
<th>Time after exposure to wood smoke</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>3h</td>
<td>20h</td>
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<tr>
<td><strong>PBMC</strong></td>
<td></td>
<td></td>
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<tr>
<td>hH01 (\times 10^{-6})</td>
<td>14.2 ± 6.45</td>
<td>19.0 ± 8.61&lt;sup&gt;1, b&lt;/sup&gt;</td>
</tr>
<tr>
<td>hNUDT1 (\times 10^{-6})</td>
<td>0.41 ± 0.09</td>
<td>0.63 ± 0.34&lt;sup&gt;a, b&lt;/sup&gt;</td>
</tr>
<tr>
<td>hOGG1 (\times 10^{-6})</td>
<td>1.07 ± 0.28</td>
<td>1.05 ± 0.35&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>hOGG1 activity&lt;sup&gt;c&lt;/sup&gt;</td>
<td>5.44 ± 4.78</td>
<td>6.57 ± 3.53</td>
</tr>
<tr>
<td>FPG sites (per 10&lt;sup&gt;6&lt;/sup&gt; bp)</td>
<td>0.25 ± 0.14</td>
<td>0.25 ± 0.098</td>
</tr>
<tr>
<td>SB (per 10&lt;sup&gt;6&lt;/sup&gt; bp)</td>
<td>0.071 ± 0.053</td>
<td>0.085 ± 0.043</td>
</tr>
<tr>
<td><strong>Urine</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8-OxodG (nmol/h)&lt;sup&gt;g&lt;/sup&gt;</td>
<td>-</td>
<td>0.73 ± 0.19&lt;sup&gt;h&lt;/sup&gt;</td>
</tr>
<tr>
<td>8-OxoGua (nmol/h)&lt;sup&gt;g&lt;/sup&gt;</td>
<td>-</td>
<td>4.24 ± 2.31&lt;sup&gt;h&lt;/sup&gt;</td>
</tr>
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[Danielsen et al. 2008]
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Indoor $\text{PM}_{10}$ exposure for different cooking systems

[Regalado et al. 2006]
Reactive Oxygen Species (ROS) in emissions for log wood combustion

\[ r = -0.876 \]

[Milijevic et al. 2010]
Epidemiology

Exposure:
1. Indoor cooking with biomass\(^1\)
2. Wildfires\(^2\)

Effects:
- Respiratory: Wheezing, Acute Respiratory Infections
- Others: Physician Visits, Cancer, Eye Irritation
- From no effect to OD* ~2.5 for some
  (aero-digestive tract cancers\(^3\))

\(^*\)OD = Odds ratio: Describes how much more likely was one event vs. another

\(^1\): [Baumgartner et al 2011; Regalado et al 2006]
\(^2\): [Künzli et al 2006]
\(^3\): [Pintos et al 1998; Sapkota et al 2007]
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The majority of tests reveal adverse health effects, which are statistically significant for:

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4. Physiological responses
5. Genotoxicity and carcinogenicity.
Trends for **increasing health relevance** are:

- condensable organic compounds > soot > salt
- decreasing combustion temperature
- lack of oxygen
- worse combustion design

**Incomplete combustion** PM is most relevant for health hence high combustion quality is crucial
Epidemiology on wood PM is limited, however confirms health effects by eye irritation and cancer

In addition to sampled PM, Secondary Organic Aerosols from need to be considered

Information on health effects considering SOA is lacking
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Prof. Dr. Barbara Rothen
The End