

# Public Comment

for November 12, 2024

Community Advisory Committee  
Meeting

## 2 - Non-Agenda Item Public Comment

**From:** [Wendy Ring](#)  
**To:** [Public Comment](#)  
**Subject:** Comment for upcoming CAC meeting  
**Date:** Monday, September 30, 2024 6:54:27 PM

---

I was very happy to join the Biomass Technical Advisory Committee. I was not invited by RCEA as a health professional but came onto the committee as the designated representative of 350 Humboldt. The three important things I gained from the information provided by HSC were:

1) **HSC cheated on toxic emissions tests** by burning wood with a moisture content less than half the moisture content of their usual fuel. The wetter the fuel, the higher the emissions, so results with dry fuel are not representative of what workers and the community are breathing and, if the air district accepts them, may let the company get away without doing a health risk assessment.

(When I went to look back at the fuel moisture content during tests for compliance with the Clean Air Act, I found HSC had done the same thing and the air district accepted the results).

2) **The biomass plant burned only mill waste** over the past year. Some was from other local mills and some was trucked in from Mendocino Redwood's mill in Mendocino.

3) HSC knows that **the new multiclones** it is installing will not decrease pollution emissions, as the CAC was told. Their purpose is to decrease shutdowns for maintenance and allow all the boilers to operate more hours, which **will increase pollution**. The air district's draft permit to install the multiclones states that running all 3 boilers simultaneously 24 hours a day, 365 days a year would **nearly double VOCs, triple NOx and SOx, and increase pm2.5 and CO emissions fivefold**. The EPA is currently reviewing this.

The Notices of Violation which HSC provided to RCEA are not all the NOV's for that time period. The air district is delaying the issuance of a large number of NOV's saying that they will be issued along with HSC's Full Compliance Evaluation. The FCE is a federal requirement and it is past due. The air district has been working on it for 3 years now. If there were no significant compliance issues, it would probably not take that long.

The Humboldt Coalition for Clean Energy submitted a comment protesting the air district's approval of any emissions increase from HSC, and suggesting either limits to keep operating time the same or installation of best available pollution controls.

The EPA has been investigating the air district's permitting practices with regard to HSC for the past 7 months. EPA staff told me they plan to issue their conclusion in a few weeks as to whether or not the plant has been operating without a permit.

Wendy Ring

*Stories of climate action from the bottom up  
with [Cool Solutions Podcast](#)*

## 2 - Non-Agenda Item Public Comment

**From:** [Walter Paniak](#)  
**To:** [Lori Taketa](#)  
**Subject:** European Study reference anthropogenic biomass pollution  
**Date:** Wednesday, October 16, 2024 3:10:20 PM  
**Attachments:** 1577full.pdf

---

The copy and pasted quoted paragraph below is a summary of the longer article in the attachment below.

Please post this note and attachment as a public comment and forward it to the appropriate parties for discussions and consideration.

Most European countries have a common medical record data base. Finland, for example, can do a census of medical record versus a sample of a limited numbers of records across several data bases from different sources. The level of certainty is greater from a large and organized sample.

“Conclusions Biomass combustion is widespread, is increasing, and makes an important contribution to ambient PM2.5, especially in winter, in the developed world. Epidemiological studies strongly suggest that there are adverse health effects related to short-term as well as long-term exposure to biomass smoke in the developed world. Intervention studies performed, to date, suggest beneficial health effects of reducing exposure to biomass smoke. We recommend that emissions from biomass combustion should be kept to a minimum to protect public health. As the evidence from studies in the developed world is still limited, further studies are necessary to more precisely quantify the adverse health effects of biomass combustion. This should include comparative studies to document similarities and differences between effects of combustion products from biomass and fossil fuels.”

Walt Paniak  
Arcata resident



# Health impacts of anthropogenic biomass burning in the developed world

Torben Sigsgaard<sup>1</sup>, Bertil Forsberg<sup>2</sup>, Isabella Annesi-Maesano<sup>3,4</sup>, Anders Blomberg<sup>5</sup>, Anette Bølling<sup>6</sup>, Christoffer Boman<sup>7</sup>, Jakob Bønløkke<sup>1</sup>, Michael Brauer<sup>8</sup>, Nigel Bruce<sup>9</sup>, Marie-Eve Héroux<sup>10</sup>, Maija-Riitta Hirvonen<sup>11</sup>, Frank Kelly<sup>12</sup>, Nino Künzli<sup>13,14</sup>, Bo Lundbäck<sup>15</sup>, Hanns Moshhammer<sup>16</sup>, Curtis Noonan<sup>17</sup>, Joachim Pagels<sup>18</sup>, Gerd Sallsten<sup>19</sup>, Jean-Paul Sculier<sup>20</sup> and Bert Brunekreef<sup>21,22</sup>

**Affiliations:** <sup>1</sup>University of Aarhus, Institute of Public Health, Aarhus, Denmark. <sup>2</sup>Dept of Public Health and Clinical Medicine/Environmental Medicine, Umeå University, Umeå, Sweden. <sup>3</sup>INSERM UMR-S 1136, Institute Pierre Louis of Epidemiology and Public Health, Epidemiology of Allergic and Respiratory Diseases, Paris, France. <sup>4</sup>UPMC, UMR-S 1136, Institute Pierre Louis of Epidemiology and Public Health, Epidemiology of Allergic and Respiratory Diseases, Paris, France. <sup>5</sup>Dept of Public Health and Clinical Medicine/Medicine, Umeå University, Umeå, Sweden. <sup>6</sup>Norwegian Institute of Public Health, Division of Environmental Medicine, Dept of Air Pollution and Noise, Oslo, Norway. <sup>7</sup>Thermochemical Energy Conversion Laboratory, Dept of Applied Physics and Electronics, Umeå University, Umeå, Sweden. <sup>8</sup>University of British Columbia, School of Population and Public Health, Vancouver, BC, Canada. <sup>9</sup>WHO, Geneva, Switzerland. <sup>10</sup>WHO Regional Office for Europe, Bonn, Germany. <sup>11</sup>University of Eastern Finland, Kuopio, Finland. <sup>12</sup>King's College London, London, UK. <sup>13</sup>Swiss Tropical and Public Health Institute, Basel, Switzerland. <sup>14</sup>University of Basel, Basel, Switzerland. <sup>15</sup>Krefting Research Centre, Institute of Medicine, University of Gothenburg, Gothenburg, Sweden. <sup>16</sup>Medical University of Vienna, Institute of Environmental Health, Vienna, Austria. <sup>17</sup>The University of Montana, Center for Environmental Health Sciences, Missoula, MT, USA. <sup>18</sup>Lund University, Ergonomics and Aerosol Technology, Lund, Sweden. <sup>19</sup>Division of Occupational and Environmental Medicine, Institute of Medicine, University of Gothenburg, Gothenburg, Sweden. <sup>20</sup>University Jules Bordet, Brussels, Belgium. <sup>21</sup>Utrecht University, Institute for Risk Assessment Sciences, Utrecht, The Netherlands. <sup>22</sup>Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht, The Netherlands.

**Correspondence:** Torben Sigsgaard, Aarhus University, Dept of Public Health, Section for Environment, Occupation and Health, Bartholin Allé 2 DK-8000 Aarhus C, Denmark. E-mail: ts@iph.au.dk

**ABSTRACT** Climate change policies have stimulated a shift towards renewable energy sources such as biomass. The economic crisis of 2008 has also increased the practice of household biomass burning as it is often cheaper than using oil, gas or electricity for heating. As a result, household biomass combustion is becoming an important source of air pollutants in the European Union.

This position paper discusses the contribution of biomass combustion to pollution levels in Europe, and the emerging evidence on the adverse health effects of biomass combustion products.

Epidemiological studies in the developed world have documented associations between indoor and outdoor exposure to biomass combustion products and a range of adverse health effects. A conservative estimate of the current contribution of biomass smoke to premature mortality in Europe amounts to at least 40 000 deaths per year.

We conclude that emissions from current biomass combustion products negatively affect respiratory and, possibly, cardiovascular health in Europe. Biomass combustion emissions, in contrast to emissions from most other sources of air pollution, are increasing. More needs to be done to further document the health effects of biomass combustion in Europe, and to reduce emissions of harmful biomass combustion products to protect public health.



@ERSpublications

**Biomass combustion is an important source of air pollution and ill health in the EU: emissions need to reduce** <http://ow.ly/RYkPk>

## Introduction

During the first half of the twentieth century, air pollution in many European cities was dominated by local emissions from fossil fuel combustion for space heating, energy production and manufacturing. As a consequence pollution episodes were common and often severe, like the smog episodes in London [1] and the Meuse valley [2] in Belgium, both associated with large and sudden increases in mortality. These and other episodes led the governments of different countries to introduce air pollution regulations, e.g. the Clean Air Act introduced in 1956 in the UK. Due to this change in practice and the increased availability of cleaner fuels such as oil and gas during the 1960s, air quality in many European cities improved markedly. Nevertheless, more recent studies have demonstrated continued adverse health effects of air pollution at much lower levels of exposure [3–5]. Recently it has also been shown that further decreases in air pollution had a beneficial effect on the life expectancy of the American population during the past few decades [6]. The World Health Organization (WHO), in its 2005 global update of the Air Quality Guidelines, set a guideline for fine particulate matter (PM) in air of just  $10 \mu\text{g}\cdot\text{m}^{-3}$  as an annual average. This concentration is still exceeded in large parts of Europe, and continued efforts are needed to reduce fine particles from all sources.

Against this background, it is potentially worrying that, as a response to climate change policies and fuel pricing, biomass burning (primarily of wood) for residential space heating as well as energy production is now an increasing source of fine PM emissions in the European Union (EU), thus posing new challenges to human health. As a response to fuel poverty, in some communities, wood/biomass burning is seen as a cheap form of fuel when gathered locally. For example, a recent study in Greece documented a 30% increase in winter PM, a 2.5-fold increase in biomass combustion markers and a 20–30% decrease in fuel oil tracers coinciding with the recent economic crisis [7].

A relatively small number of studies have evaluated the health effects of outdoor and indoor exposure to combustion products from fireplaces or wood stoves in developed countries [8–10]. However, extensive epidemiological literature exists that describes the adverse health effects of high-level exposure to pollution from household biomass combustion for cooking and heating purposes in developing countries [11–13]. This literature indicates causal links between PM and acute lower respiratory infections in young children, chronic obstructive pulmonary disease (COPD) in adults [14], and development of cataracts in women [15]. Lung cancer has also been linked to inhalation of biomass combustion products. Indoor emissions from biomass combustion and cooking in developing countries have been classified as probably carcinogenic (International Agency for Research on Cancer IARC Group 2A) [16].

Although combustion of biomass is known to produce numerous air pollutants, the focus of this report is PM emissions, which have received most of the attention in the scientific literature. Therefore, only epidemiological studies in which the primary measure for evaluation of biomass exposure is PM, or components thereof, are considered. This is consistent with the approach taken in prior reviews of biomass combustion health impacts [17, 18] and the Global Burden of Disease (GBD) 2010 report [19, 20]. This approach also follows the perspective of the WHO Air Quality guidelines [21], the review by NAEHER *et al.* [17] and the recent WHO REVIHAAP project report [22], which concluded that there was a lack of evidence supporting differential toxicity of PM from biomass combustion when compared with urban PM from fossil fuel combustion and secondary atmospheric particles.

This position paper deals with the effects of biomass smoke on respiratory and cardiovascular health in the developed world, where wood burning is primarily, but increasingly, used as an auxiliary source of domestic heating and energy production. When searching the literature, the following terms were used in a PubMed search: “wood smoke” OR “domestic heating” AND “USA OR Canada OR Europe OR Australia” AND “wood stove” AND “health”. In addition to the search we consulted international experts and research centres active in the field.

## Biomass combustion emissions in developed countries

Estimates from several countries have shown that biomass (primarily wood) burning is contributing substantially to the total concentrations of PM in the local environment. Studies from Denmark and Sweden have found air pollution concentrations from wood burning in rural areas with limited traffic to be

---

This article has supplementary material available from [erj.ersjournals.com](http://erj.ersjournals.com)

Received: Oct 08 2014 | Accepted after revision: Sept 01 2015 | First published online: Sept 24 2015

The authors alone are responsible for the views expressed in this publication and they do not necessarily represent the decision or stated policy of the World Health Organization.

Conflict of interest: Disclosures can be found alongside the online version of this article at [erj.ersjournals.com](http://erj.ersjournals.com)

in the same range as those from traffic in major cities [23, 24]. Estimates of the current situation for some European countries (*i.e.* Sweden, Finland, Germany and Austria) indicate 15–25% of particles with a 50% cut-off aerodynamic diameter of  $<2.5\ \mu\text{m}$  (PM<sub>2.5</sub>) originate from residential biomass combustion [25] and data from Alpine valleys indicate contributions of 50% or more. From a European perspective, it has been estimated that small scale domestic wood/biomass combustion will become the dominant source of fine primary particle air pollution by 2020 [26], with a contribution of 38% of total emissions (figs 1 and 2). More recently the International Institute for Applied Systems Analysis estimated that, between 2005 and 2030, the use of biomass for energy production in the EU will double, whereas use of coal, gas and oil are all expected to decrease [27]. Another study estimated that of all external health costs occurring in Denmark as a result of Danish air pollution emissions, woodsmoke generated by domestic heating was responsible for 16% of these costs in 2000, and subsequently reached 30% in 2008 due to an 80% increase in the use of wood for domestic heating in Denmark over that period [28].

Within the EU, biomass is used in small scale residential ( $<50$  kilowatt thermal (kW<sub>th</sub>)) heating appliances and in medium scale ( $<20$  MW<sub>th</sub>) district heating/industrial systems as well as in large centralised heat and power plants. In the small and medium scale sector, a large number of units are in use and the technology covers a vast range, from the simplest fireplaces to fully automated systems. The current number of residential biomass combustion systems within the EU is estimated to be  $\sim 65$  million direct heating appliances (fireplaces, stoves and cookers) and  $\sim 8$  million indirect heating appliances (boilers), firing mainly using wood logs but also wood chips and fuel pellets [24]. The technological level has traditionally been low, as has the combustion efficiency and emission performance. It is generally understood, although poorly documented, that behavioural factors can have a large impact on residential stove emissions, including wood sizing, fuel moisture content, ignition procedure, burn temperatures and stove maintenance [29, 30]. Recent developments have resulted in modern appliances with decreased emissions, but still, these systems are often sensitive to firing procedures and are not optimised in real life-operation. Moreover, emissions during the starting phase remain magnitudes higher than in the stationary phase even in modern appliances [31, 32]. Thus, significant potential for further emission reduction remains [25].

The typical PM<sub>2.5</sub> emission factors given range from 40–300 and 50–2000 mg·MJ<sup>-1</sup> for modern and conventional domestic wood stoves, respectively [33]. When using humid fuels and poorly insulated stoves, the PM<sub>2.5</sub> emissions may be significantly elevated (grammes per MJ). Emissions from such low-temperature combustion are often completely dominated by liquid droplets consisting of a complex array of organic components [34]. Combustion may also be too intense when using very dry fuel in well insulated stoves, thus resulting in so-called “air-starved” conditions. Such combustion may contain the highest emissions of carcinogenic polycyclic aromatic hydrocarbons (PAHs) and is dominated by solid agglomerated soot (black carbon) particles, which shows similarities to diesel soot [32, 35].

Modern biomass combustion technologies such as automatic small-scale wood pellet appliances and larger domestic heating plants are commonly more efficient and have much lower particle emissions in the range of 10–50 mg·MJ<sup>-1</sup> (typically  $<30$  mg·MJ<sup>-1</sup>) [33]. Using these technologies, organic and soot emissions may

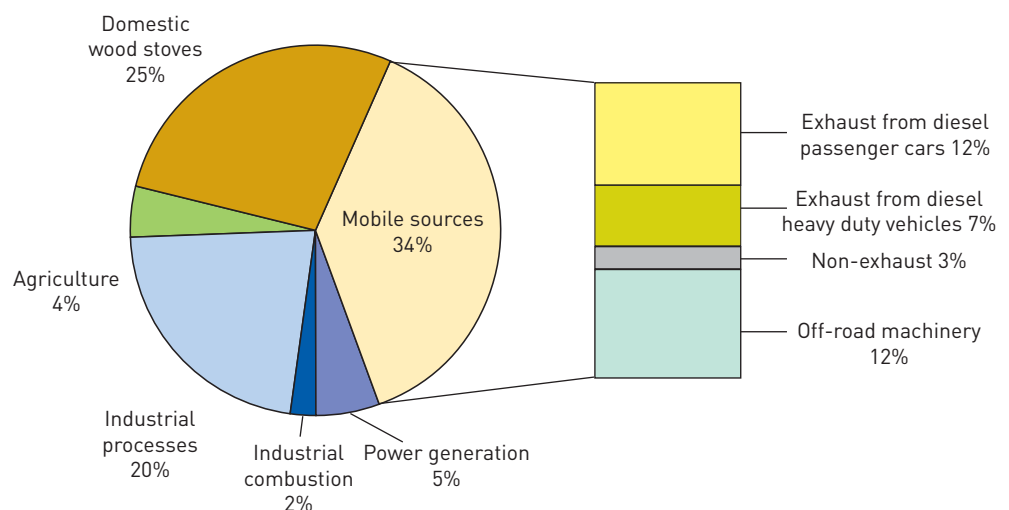


FIGURE 1 Sector contributions to primary particles with a 50% cut-off aerodynamic diameter of  $<2.5\ \mu\text{m}$  emissions in the EU15, 2000. Reproduced from [26].

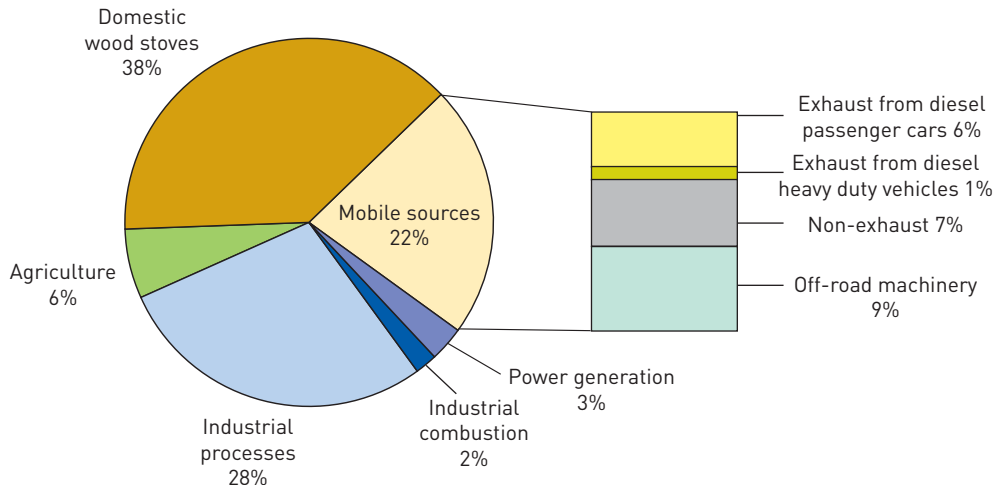


FIGURE 2 Sector contributions to primary particles with a 50% cut-off aerodynamic diameter of  $<2.5 \mu\text{m}$  emissions in the EU15, 2020. Reproduced from [26].

be almost completely eliminated, and the particle emissions are instead dominated by inorganic salts like potassium sulfates and chlorides, with minor amounts of zinc.

Further information on biomass combustion technology is provided in the online supplementary material.

## Exposure

The contribution of woodsmoke to ambient PM mass is highly dependent on season, density of sources and the specific technologies employed as well as meteorology and topography. Studies have estimated that wood/biomass combustion contributes 10–30% or  $\sim 1\text{--}4 \mu\text{g}\cdot\text{m}^{-3}$  to the annual average fine particle concentrations measured in different parts of Europe. Details of the studies and methods applied can be found in the online supplemental material. Tables S1–S3 show the contributions of biomass combustion to outdoor and indoor PM concentrations in the developed world. In some cases wood combustion is the major source of ambient PM, especially during the heating season. Woodsmoke also contributes substantially to some of the particle components that are considered particularly harmful such as particle-bound PAH, volatile organic compounds and metals [23, 36–40].

While air pollution from woodsmoke is often highest in close vicinity to houses where and when wood burning takes place [40], there is also a more global contribution of biomass combustion from indoor heating and cooking, energy production, and from agricultural and wild fires even at distant background stations [41] and over the oceans [42]. Outdoor woodsmoke concentrations may be less predictive for individual exposure compared with other air pollutants because of the potential for wood stoves to emit pollution indoors, and wood combustion for heating occurs in locations and during periods when indoor infiltration may be relatively low due to building construction (insulation) and operation (closed windows) [43, 44]. Therefore, unsurprisingly, studies evaluating fine particle levels in homes with wood-burning appliances have given mixed results, ranging from indoor particles with a 50% cut-off aerodynamic diameter of  $<10 \mu\text{m}$  (PM<sub>10</sub>) levels as high as  $100 \mu\text{g}\cdot\text{m}^{-3}$  [45, 46] to only minor differences between indoor concentrations measured in homes with and without wood burning appliances. In a few studies looking at woodsmoke tracers, significant differences (66–80%) were observed for potassium, calcium and zinc as well as 1,3-butadiene and benzene [47, 48]. In one study, the levels of benzo(a)pyrene and several other PAHs were found to be three- to five-fold higher in homes with wood combustion appliances compared with homes without [49].

## Epidemiology

### *Health effects of anthropogenic biomass combustion products in the developed world*

Whereas there is a vast literature on health effects of indoor air pollution from biomass burning in the developing world [11], comparatively few studies have addressed health effects of biomass burning in the developed world. We will first discuss epidemiological observations from areas where biomass combustion (mostly wood burning) is a relatively important source of ambient air pollution. We will then discuss smaller-scale studies of populations living in homes using wood for heating and/or cooking.

Several studies have looked at the health effects of air pollution in Seattle, USA and Vancouver, Canada where, in winter, residential wood burning is an important contributor to outdoor PM pollution. A time

series study conducted in Seattle documented that in the cold season, ambient PM<sub>2.5</sub> was associated with total and cardiovascular (but not respiratory) mortality [50]. Interestingly, potassium, a marker of woodsmoke, also showed positive associations with total and cardiovascular mortality in the cold season. A study from Vancouver also showed positive associations between PM<sub>10</sub> in winter and total mortality [51].

Other studies from Seattle found associations between PM and respiratory symptoms (chest congestion and wheeze) in young children [52], increased medication use [53], decreased lung function [54], emergency room visits for asthma [55] and hospitalisations [56]. Smaller studies on cardiovascular morbidity end-points generally found little evidence for effects on sudden cardiac arrest [57], myocardial infarction [58], heart rate variability [59], and measures of systemic inflammation or thrombosis [60].

There are few epidemiological studies of chronic or sub-chronic exposure to biomass PM resulting from residential wood burning as a heat source. In metropolitan Vancouver, extensive mobile monitoring and geospatial modelling were used to develop a spatio-temporal model of ambient winter woodsmoke [61], which was applied to a variety of health outcomes. Positive associations were reported with low birth weight [62], infant bronchiolitis [63], otitis media [64] and COPD hospitalisation. However, no association was found for COPD mortality [65] or for incident childhood asthma [66].

A study conducted in southern California reported significant positive associations between the biomass combustion source fraction and preterm birth [67], although not with term low birth weight [68], which was also not associated with PM mass.

In Europe, some studies have estimated the acute exposure impacts of particle source apportionment in locations where biomass combustion is a more minor contributor to ambient PM. For example, in a study conducted in Copenhagen, associations were reported between the biomass source fraction and respiratory and cardiovascular hospital admissions in the elderly (age >65 years), but not for asthma-related admissions among children [69]. Of these, only the associations with respiratory admissions remained significant after adjustment for the contributions from other sources.

In Christchurch, New Zealand, source apportionment studies indicated that 90% of wintertime PM originated from wood combustion, and ambient PM<sub>10</sub> was associated with increased respiratory and cardiovascular (excluding ischaemic heart disease) hospital admissions [70]. In a study conducted in Temuco, Chile, where an estimated 87% of winter PM<sub>10</sub> was attributable to wood combustion, PM<sub>10</sub> was associated with cardiovascular and respiratory mortality, hospitalisations, and emergency room visits for acute respiratory infections [71]. This study was conducted in an area where the average PM<sub>10</sub> concentration was 46 µg·m<sup>-3</sup> with daily means often well above 150 µg·m<sup>-3</sup>, *i.e.* much higher than concentrations measured in Seattle, USA and Vancouver, Canada.

#### *Health impacts of community and household stove interventions*

Studies of community- or household-level health impacts following wood stove intervention strategies have been limited. Table 1 highlights results on effects of interventions such as wood stove change out programmes on ambient PM levels in the developed world; table S2 shows the effects on indoor PM

TABLE 1 Effects of wood stove interventions on outdoor particulate matter (PM) levels in developed countries

Location	Estimated reduction in PM µg·m <sup>-3</sup>	Notes	References
Launceston, Tasmania, Australia	38% reduction in winter PM <sub>10</sub>	Fuel switching: replacement of wood heating appliances with electric heating appliances. The proportion of households burning wood was reduced from 66% to 30%.	[72]
British Columbia, Canada	22% reduction in winter PM <sub>2.5</sub>	Introduction of improved technology stoves and targeting of open fireplaces. The proportion of homes using open fireplaces was reduced from 15% to 3%, and the proportion of homes with improved technology wood stoves increased from 25% to 41%. The community also had an overall increase in wood stove usage.	[73]
Missoula, MT, USA	45% reduction in PM <sub>10</sub>	Legislative action and enforcement. Over a 10-year period, the proportion of households burning wood was reduced from 44% to 20% and the contribution of residential wood burning to PM <sub>10</sub> was reduced from 47% to 11%.	[74]
Libby, MT, USA	27% reduction in winter PM <sub>2.5</sub>	Introduction of improved technology stoves. Over 1100 older model wood stoves were replaced with improved technology stoves.	[75, 76]

PM<sub>10</sub>: particles with a 50% cut-off aerodynamic diameter of <10 µm; PM<sub>2.5</sub>: particles with a 50% cut-off aerodynamic diameter of <2.5 µm.



concentrations. A 4-year, survey-based study tracked parent-reported respiratory symptoms and conditions among school children during the community wood stove exchange in Libby, Montana, USA. Significant reductions in reported frequency per  $5 \mu\text{g}\cdot\text{m}^{-3}$  decline in ambient  $\text{PM}_{2.5}$  were observed for wheeze (27%), irritant symptoms such as watery eyes (33%), cold (25%), bronchitis (55%), flu (52%) and throat infection (45%) [75]. No significant reductions were observed for reported ear infection, but the population studied was not in the age group most commonly impacted by this condition. In the same community, no consistent effects were observed for school absences.

In Launceston, Tasmania, Australia, substantial mortality changes were observed between the period before (1994–2000) and after (2001–2007) a government-coordinated fuel switching effort. The investigators demonstrated significant reductions in male mortality only: 18% from cardiovascular causes and 28% from respiratory causes [72]. Similar findings of borderline significance were observed for winter mortality for both sexes combined.

Finally, a high-efficiency particulate air filter intervention crossover study examined changes in microvascular endothelial function as measured by reactive hyperaemia index (RHI) as well as changes in serum and urine markers of oxidative stress and inflammation [77]. In this study, filtration units were placed in homes with and without wood stoves to reduce indoor PM concentrations. The use of the filter was associated with significantly higher mean measures of RHI (9%) and significantly lower mean measures of the inflammatory marker C-reactive protein (CRP) (33%). These findings remained robust when the analyses were restricted to participants in wood stove homes, but not among participants in non-wood stove homes. Smaller magnitude and nonsignificant changes in RHI and CRP were found when modelled per  $\mu\text{g}\cdot\text{m}^{-3}$  reduction in indoor  $\text{PM}_{2.5}$  rather than per filter on/off status, possibly reflecting between-home variability in smoke infiltration. A recent study conducted in an urban area at very low levels of ambient and indoor  $\text{PM}_{2.5}$  was, however, not able to reproduce these findings [78].

### *Wild fires*

The respiratory health effects associated with wildfire smoke exposure have recently been reviewed in detail [79, 80], and are summarised here to provide supporting evidence of the health impacts resulting from exposure to biomass combustion products. Similar to the literature described above for domestic biomass combustion, wildfire smoke exposure was associated with respiratory symptoms, increased asthma medication use, outpatient physician visits, emergency room visits, hospital admissions and mortality. Stronger associations were observed for asthma-specific physician visits, hospitalisations and emergency room visits, as well as for respiratory symptoms among individuals with asthma compared with non-asthmatic individuals. Relatively few studies have reported on the relationship between cardiovascular outcomes and wildfire smoke, with varying specificity in exposure estimates. A number have reported null associations for mortality [81], hospital admissions [82] and outpatient physician visits [83], despite evidence of positive associations for measures of respiratory health. However, several recent studies have reported associations between forest fire smoke exposure and cardiac outcomes including emergency visits for heart failure [83], out of hospital cardiac arrest [84–86] and cardiac mortality [87, 88].

### **Toxicity of woodsmoke particles**

The physicochemical properties of biomass combustion PM varies between different combustion conditions. As discussed in the online supplementary material, the heterogeneity of biomass PM characteristics is high. Accordingly, evaluation of the respiratory and cardiovascular toxicity of biomass emission PM is complex. The limited knowledge of physicochemical properties of ambient (real-life) biomass PM exposure further complicates the evaluation of the possible health risks associated with biomass PM exposure.

### *Impact of combustion conditions and particle properties*

Woodsmoke particles constitute a complex and variable mixture of organic-dominated particles, soot agglomerates and inorganic ash alkali particles; three particle types that differ considerably in shape, size, solubility and chemical composition [89]. Health relevant properties of these particle types are described in more detail in the online supplementary material. These differences in physicochemical properties may affect various aspects of PM-induced toxicity, including pulmonary deposition, clearance and cellular effects. For instance, soluble inorganic ash particles are cleared rapidly from the lungs compared with insoluble soot agglomerates. The pulmonary deposition is determined by size, shape and hygroscopicity. For biomass PM, hygroscopic particle growth in the respiratory tract has a large impact on particle deposition probability [90–92]. Biomass PM from complete and incomplete combustion exhibited relatively low deposited fractions in the respiratory tract, while ambient woodsmoke PM (likely originating from mixed combustion conditions) resulted in larger deposited fractions. Although the ambient woodsmoke deposition was lower than for vehicular traffic particles (38% versus 69% for particle number concentrations), the alveolar deposited fractions were estimated to be ~20% for both types of particles [92]. Differences in pulmonary

deposition for different types of biomass PM possibly affect the severity of the respiratory and cardiovascular effects, but this has not been specifically addressed in human inhalation studies. In cell culture studies, the chemical composition and combustion conditions have a large impact on the cellular effects induced by biomass PM. Various organic compounds influence the biological effects of PM resulting from poor combustion, whereas metals are of major importance for the effects induced by inorganic ash particles from complete combustion [89, 93–95]. These findings have also been confirmed *in vivo* in two short-term instillation studies of different types of biomass PM [94, 95]. It is important to keep in mind that these *in vitro* and *in vivo* studies do not account for differences in pulmonary particle deposition and clearance, which are also influenced by the combustion conditions and the resulting PM properties. Physicochemical ageing in the atmosphere, by reaction of the emissions with hydroxyl radicals and ozone, chemically transforms the primary biomass PM. For example PAHs may be transformed to quinones and nitro-PAHs [96]. This also leads to formation of low volatility vapours that add new secondary organic particle mass (on the timescale of minutes to days). Currently, it is not known if atmospheric ageing leads to increased or decreased toxicity. An initial study showed effects of ageing by ozone on biomass PM characteristics and toxicity [97].

#### ***Toxicological effects related to human health***

Human controlled exposure studies report that short-term inhalation of woodsmoke induces mild inflammatory effects including distal airway inflammation, increased oxidative stress and immune cell recruitment, but no effects on measures of lung function [98–103]. Studies also report systemic effects such as increased levels of coagulation markers, and decreased heart rate variability and systemic inflammation [100, 104–107]. In mice and rats, long-term inhalation of woodsmoke in concentrations relevant for ambient exposure induces mild inflammatory effects in the airways, systemic inflammation and decreased lung function [17, 108–112]. Woodsmoke PM has also been reported to exacerbate allergic inflammation and allergic sensitisation, and to decrease pulmonary macrophage function in terms of impaired infection resistance [17, 109, 112–114], this was recently demonstrated to apply equally to particles from Malawian and Norwegian woodsmoke [115]. Overall, *in vivo* and *in vitro* experiments demonstrate that woodsmoke PM can induce inflammatory responses, cytotoxicity, genotoxicity, oxidative stress and immunosuppressive effects [17, 93–95, 104, 111, 116–122]. Atopy alone did not appear to increase susceptibility to the toxic effects of woodsmoke PM [123], whereas conditions affecting deposition, such as pre-existing lung disease, have yet to be studied in biomass combustion product experiments.

While some human inhalation studies report significant respiratory and systemic effects, other studies have not found these. This ambiguity could partly be due to the great heterogeneity in experimental set-ups, fuel type, biomass exposure generation and physicochemical properties of PM. Interestingly, the two studies reporting significant effects for either respiratory or cardiovascular end-points applied biomass PM originating from flaming combustion of wood logs in conventional stoves, rendering emissions of soot and organics [98, 99, 104, 105]. Other studies applied PM from more complete or more incomplete (smouldering) combustion conditions. This may point towards an influence of combustion conditions and physicochemical properties on the effects reported in the human exposure studies.

The high variability in respiratory and cardiovascular effects reported in the human inhalation studies could be due, in part, to differences in the physicochemical properties of the applied biomass PM. This reflects the inherent complexities in the evaluation of health risks from a very heterogeneous class of PM. The mild inflammatory effects following short-term, controlled inhalation of woodsmoke in healthy individuals are in accordance with the mild inflammatory effects reported in animal models. However, these data cannot be extrapolated to long-term exposure scenarios or effects in susceptible individuals. Also, based on the current, limited experimental findings, we cannot conclude that exposure to residential biomass emissions in developed countries is less harmful than exposure to combustion particles from fossil fuel combustion.

#### **Policy implications**

Air pollution arising from wood burning has been recognised as a problem for some time in Scandinavian and Alpine countries, especially during the winter period. Across Europe, the Renewable Energy Directive has set a goal to produce 20% of energy from renewable sources by 2020, increasing wood/biomass combustion for power generation. As mentioned in the Introduction, biomass combustion is expected to become the major source of primary PM emissions over the next 5–15 years. This will compromise efforts to reduce ambient PM concentrations to below the current WHO Air Quality Guidelines. This, in turn, will probably result in large numbers of avoidable, premature deaths across Europe over that time period.

Between 2010 and 2020, biomass burning is forecast to increase by 57–110% across the EU [124]. The UK alone is stimulating through financial incentives 700 000 homes to convert to biomass heating systems, and biomass boilers are increasingly installed to meet renewable energy requirements.

So far, the emission limits that exist within the EU and the rest of the developed world for new small-scale biomass combustion devices are rather tolerant and easily fulfilled by today's systems. The measuring methods applied and emission limits still vary, thus the need for harmonisation is obvious. To date, insufficient efforts have been made towards the development of particulate emission control devices for these units. The situation is, however, currently under revision, and considerably tougher and harmonised emission standards are under discussion within the EU (e.g. related to the EcoDesign directive). In parallel with new technological solutions, it is also important to educate wood stove owners in the best wood burning practices based on recent research. Still, a considerable pollution contribution from existing residential installations will most probably continue.

Furthermore, it is expected that smaller, decentralised biomass installations will become more important in future energy supply scenarios. To enable an increased bioenergy sector, unexploited raw materials other than those based on stem wood (e.g. from forestry, agricultural sector and industrial residues) will have to be utilised. Compared with traditional wood fuels, these fuels have a higher content of fine particle forming ash matter and trace metals [125].

As outlined earlier in this review, there is increasing evidence of adverse health effects of wood/biomass combustion emissions as currently dominated by those from small inefficient stoves, which places this renewable energy source in direct conflict with its perception as a healthy source of energy and domestic heating. The potential for adverse health effects associated with biomass emissions also conflicts with their role in reducing other exposures forcing climate change. A conservative estimate of the current contribution of biomass smoke to premature mortality in Europe would amount to at least 40 000 deaths per year. This is based on a contribution of biomass smoke to population exposure of 10%, and recent estimates of the total mortality burden due to PM exposure in Europe (EU28) of over 400 000 premature deaths each year [126]. A recent WHO report estimated that ambient PM from residential heating with wood and coal is responsible for 61 000 premature deaths per year in the EU28 [127]. This latter estimate is dominated by woodsmoke.

Modern and more efficient biomass technologies are available, and further development and implementation of such systems are clearly motivated from an air pollution mitigation perspective. In addition, an important regulatory step would be the unconditional adoption of the PM<sub>10</sub> and PM<sub>2.5</sub> WHO annual mean Air Quality Guideline values to protect public health [128]. Compliance with these science-based guideline values (annual mean PM<sub>10</sub> and PM<sub>2.5</sub> concentrations of 20 and 10 µg·m<sup>-3</sup>, respectively) would inevitably require the development and implementation of clean air strategies and "best available technology" for biomass combustion, comparable to the EU control policies for vehicle emissions.

## Conclusions

Biomass combustion is widespread, is increasing, and makes an important contribution to ambient PM<sub>2.5</sub>, especially in winter, in the developed world.

Epidemiological studies strongly suggest that there are adverse health effects related to short-term as well as long-term exposure to biomass smoke in the developed world. Intervention studies performed, to date, suggest beneficial health effects of reducing exposure to biomass smoke. We recommend that emissions from biomass combustion should be kept to a minimum to protect public health.

As the evidence from studies in the developed world is still limited, further studies are necessary to more precisely quantify the adverse health effects of biomass combustion. This should include comparative studies to document similarities and differences between effects of combustion products from biomass and fossil fuels.

## Acknowledgements

This position paper is the result of a workshop convened and supported by the European Respiratory Society in Brussels, Belgium, March 6–7, 2014. We sincerely thank the constructive assessment and many suggestions received from the *European Respiratory Journal* peer reviewers, which significantly increased the quality and scope of our original submission.

## References

- 1 Logan WP. Mortality in the London fog incident, 1952. *Lancet* 1953; 1: 336–338.
- 2 Nemery B, Hoet PH, Nemmar A. The Meuse Valley fog of 1930: an air pollution disaster. *Lancet* 2001; 357: 704–708.
- 3 Pope CA 3rd, Burnett RT, Thun MJ, *et al.* Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* 2002; 287: 1132–1141.
- 4 Beelen R, Raaschou-Nielsen O, Stafoggia M, *et al.* Effects of long-term exposure to air pollution on natural-cause mortality: an analysis of 22 European cohorts within the multicentre ESCAPE project. *Lancet* 2014; 383: 785–795.
- 5 Crouse DL, Peters PA, van Donkelaar A, *et al.* Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. *Environ Health Perspect* 2012; 120: 708–714.

- 6 Correia AW, Pope CA 3rd, Dockery DW, *et al.* Effect of air pollution control on life expectancy in the United States: an analysis of 545 U.S. counties for the period from 2000 to 2007. *Epidemiology* 2013; 24: 23–31.
- 7 Saffari A, Daher N, Samara C, *et al.* Increased biomass burning due to the economic crisis in Greece and its adverse impact on wintertime air quality in Thessaloniki. *Environ Sci Technol* 2013; 47: 13313–13320.
- 8 Triche EW, Belanger K, Bracken MB, *et al.* Indoor heating sources and respiratory symptoms in nonsmoking women. *Epidemiology* 2005; 16: 377–384.
- 9 Van Miert E, Sardella A, Nickmilder M, *et al.* Respiratory effects associated with wood fuel use: a cross-sectional biomarker study among adolescents. *Pediatr Pulmonol* 2012; 47: 358–366.
- 10 Robin LF, Less PS, Winget M, *et al.* Wood-burning stoves and lower respiratory illnesses in Navajo children. *Pediatr Infect Dis J* 1996; 15: 859–865.
- 11 Mortimer K, Gordon SB, Jindal SK, *et al.* Household air pollution is a major avoidable risk factor for cardiorespiratory disease. *Chest* 2012; 142: 1308–1315.
- 12 Po JY, FitzGerald JM, Carlsten C. Respiratory disease associated with solid biomass fuel exposure in rural women and children: systematic review and meta-analysis. *Thorax* 2011; 66: 232–239.
- 13 Smith KR, Bruce N, Balakrishnan K, *et al.* Millions dead: how do we know and what does it mean? Methods used in the comparative risk assessment of household air pollution. *Annu Rev Public Health* 2014; 35: 185–206.
- 14 Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in non-smokers. *Lancet* 2009; 374: 733–743.
- 15 Pokhrel AK, Smith KR, Khalakdina A, *et al.* Case-control study of indoor cooking smoke exposure and cataract in Nepal and India. *Int J Epidemiol* 2005; 34: 702–708.
- 16 International Agency for Research on Cancer. Household Use of Solid Fuels and High-Temperature Frying (IARC Monographs on the Evaluation of Carcinogenic Risks to Humans). Lyon, International Agency for Research on Cancer, 2010.
- 17 Naeher LP, Brauer M, Lipsett M, *et al.* Woodsmoke health effects: a review. *Inhal Toxicol* 2007; 19: 67–106.
- 18 Boman BC, Forsberg AB, Jarvholm BG. Adverse health effects from ambient air pollution in relation to residential wood combustion in modern society. *Scand J Work Environ Health* 2003; 29: 251–260.
- 19 Lim SS, Vos T, Flaxman AD, *et al.* A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet* 2012; 380: 2224–2260.
- 20 Brauer M, Amann M, Burnett RT, *et al.* Exposure assessment for estimation of the global burden of disease attributable to outdoor air pollution. *Environ Sci Technol* 2012; 46: 652–660.
- 21 WHO. WHO Air Quality Guidelines. Global Update 2005, Copenhagen, WHO Regional Office for Europe, 2006.
- 22 WHO. Review of evidence on health aspects of air pollution – REVIHAAP project technical report. Copenhagen, WHO Regional Office for Europe, 2013.
- 23 Glasius M, Ketzel M, Wahlin P, *et al.* Impact of wood combustion on particle levels in a residential area in Denmark. *Atmos Environ* 2006; 40: 7115–7124.
- 24 Krecl P, Larsson EH, Strom J, *et al.* Contribution of residential wood combustion and other sources to hourly winter aerosol in Northern Sweden determined by positive matrix factorization. *Atmos Chem Phys* 2008; 8: 3639–3653.
- 25 Jokiniemi J, Hytönen K, Tissari J. Biomass combustion in residential heating: Particulate measurements, sampling, and physicochemical and toxicological characterisation. Final report of the project BiomassPM within the ERA-NET Bioenergy Programme. Kuopio, University of Kuopio, 2008.
- 26 Amann M, Bertok I, Cofala J, *et al.* Baseline Scenarios for the Clean Air for Europe (CAFE) Programme. Laxenburg, International Institute for Applied Systems Analysis, 2005.
- 27 Amann M. The Final Policy Scenarios of the EU Clean Air Policy Package. Laxenburg, International Institute for Applied Systems Analysis, 2014.
- 28 Brandt J, Silver JD, Christensen JH, *et al.* Contribution from the ten major emission sectors in Europe and Denmark to the health-cost externalities of air pollution using the EVA model system - an integrated modelling approach. *Atmos Chem Phys* 2013; 13: 7725–7746.
- 29 Petersen LK. Autonomy and proximity in household heating practices: the case of wood-burning stoves. *J Environ Policy Plann* 2008; 10: 423–438.
- 30 Johansson LS, Leckner B, Gustavsson L, *et al.* Emission characteristics of modern and old-type residential boilers fired with wood logs and wood pellets. *Atmos Environ* 2004; 38: 4183–4195.
- 31 Heringa MF, DeCarlo PF, Chirico R, *et al.* Time-resolved characterization of primary emissions from residential wood combustion appliances. *Environ Sci Technol* 2012; 46: 11418–11425.
- 32 Eriksson AC, Nordin EZ, Nystrom R, *et al.* Particulate PAH emissions from residential biomass combustion: time-resolved analysis with aerosol mass spectrometry. *Environ Sci Technol* 2014; 48: 7143–7150.
- 33 Bolling AK, Pagels J, Yttri KE, *et al.* Health effects of residential wood smoke particles: the importance of combustion conditions and physicochemical particle properties. *Part Fibre Toxicol* 2009; 6: 29.
- 34 Orasche J, Schnelle-Kreis J, Schon C, *et al.* Comparison of emissions from wood combustion. Part 2: impact of combustion conditions on emission factors and characteristics of particle-bound organic species and polycyclic aromatic hydrocarbon (PAH)-related toxicological potential. *Energy Fuels* 2013; 27: 1482–1491.
- 35 Pettersson E, Boman C, Westerholm R, *et al.* Stove performance and emission characteristics in residential wood log and pellet combustion, part 2: wood stove. *Energy Fuels* 2011; 25: 315–323.
- 36 Molnár P, Sallsten G. Contribution to PM<sub>2.5</sub> from domestic wood burning in a small community in Sweden. *Environ Sci Process Impacts* 2013; 15: 833–838.
- 37 Glasius M, Ketzel M, Wählin P, *et al.* Characterization of particles from residential wood combustion and modelling of spatial variation in a low-strength emission area. *Atmos Environ* 2008; 42: 8686–8697.
- 38 Mandalakis M, Gustafsson O, Alsberg T, *et al.* Contribution of biomass burning to atmospheric polycyclic aromatic hydrocarbons at three European background sites. *Environ Sci Technol* 2005; 39: 2976–2982.
- 39 Gaeggeler K, Prevot ASH, Dommen J, *et al.* Residential wood burning in an Alpine valley as a source for oxygenated volatile organic compounds, hydrocarbons and organic acids. *Atmos Environ* 2008; 42: 8278–8287.
- 40 Hellen H, Hakola H, Haaparanta S, *et al.* Influence of residential wood combustion on local air quality. *Sci Total Environ* 2008; 393: 283–290.
- 41 Puxbaum H, Caseiro A, Sánchez-Ochoa A, *et al.* Levoglucosan levels at background sites in Europe for assessing the impact of biomass combustion on the European aerosol background. *J Geophys Res* 2007; 112: 16.

- 42 Hu QH, Xie ZQ, Wang XM, *et al.* Levoglucosan indicates high levels of biomass burning aerosols over oceans from the Arctic to Antarctic. *Sci Rep* 2013; 3: 3119.
- 43 Barn P, Larson T, Noullett M, *et al.* Infiltration of forest fire and residential wood smoke: an evaluation of air cleaner effectiveness. *J Expo Sci Environ Epidemiol* 2008; 18: 503–511.
- 44 Noullett M, Jackson PL, Brauer M. Estimation and characterization of children's ambient generated exposure to PM<sub>2.5</sub> using sulphate and elemental carbon as tracers. *Atmos Environ* 2010; 44: 4629–4637.
- 45 Hoek G, Kos G, Harrison R, *et al.* Indoor–outdoor relationships of particle number and mass in four European cities. *Atmos Environ* 2008; 42: 156–169.
- 46 Nasir ZA, Colbeck I. Particulate pollution in different housing types in a UK suburban location. *Sci Total Environ* 2013; 445: 165–176.
- 47 Molnar P, Gustafson P, Johannesson S, *et al.* Domestic wood burning and PM<sub>2.5</sub> trace elements: personal exposures, indoor and outdoor levels. *Atmos Environ* 2005; 39: 2643–2653.
- 48 Gustafson P, Barregard L, Strandberg B, *et al.* The impact of domestic wood burning on personal, indoor and outdoor levels of 1,3-butadiene, benzene, formaldehyde and acetaldehyde. *J Environ Monit* 2007; 9: 23–32.
- 49 Gustafson P, Ostman C, Sallsten G. Indoor levels of polycyclic aromatic hydrocarbons in homes with or without wood burning for heating. *Environ Sci Technol* 2008; 42: 5074–5080.
- 50 Zhou J, Ito K, Lall R, *et al.* Time-series analysis of mortality effects of fine particulate matter components in Detroit and Seattle. *Environ Health Perspect* 2011; 119: 461–466.
- 51 Vedal S, Brauer M, White R, *et al.* Air pollution and daily mortality in a city with low levels of pollution. *Environ Health Perspect* 2003; 111: 45–51.
- 52 Browning KG, Koenig JQ, Checkoway H, *et al.* A questionnaire study of respiratory health in areas of high and low ambient wood smoke pollution. *Pediatr Asthma Allergy Immunol* 1990; 4: 183–191.
- 53 Slaughter JC, Lumley T, Sheppard L, *et al.* Effects of ambient air pollution on symptom severity and medication use in children with asthma. *Ann Allergy Asthma Immunol* 2003; 91: 346–353.
- 54 Trenga CA, Sullivan JH, Schildcrout JS, *et al.* Effect of particulate air, pollution on lung function in adult and pediatric subjects in a Seattle panel study. *Chest* 2006; 129: 1614–1622.
- 55 Norris G, YoungPong SN, Koenig JQ, *et al.* An association between fine particles and asthma emergency department visits for children in Seattle. *Environ Health Perspect* 1999; 107: 489–493.
- 56 Sheppard L, Levy D, Norris G, *et al.* Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987–1994. *Epidemiology* 1999; 10: 23–30.
- 57 Levy D, Sheppard L, Checkoway H, *et al.* A case-crossover analysis of particulate matter air pollution and out-of-hospital primary cardiac arrest. *Epidemiology* 2001; 12: 193–199.
- 58 Sullivan J, Sheppard L, Schreuder A, *et al.* Relation between short-term fine-particulate matter exposure and onset of myocardial infarction. *Epidemiology* 2005; 16: 41–48.
- 59 Sullivan JH, Schreuder AB, Trenga CA, *et al.* Association between short term exposure to fine particulate matter and heart rate variability in older subjects with and without heart disease. *Thorax* 2005; 60: 462–466.
- 60 Sullivan JH, Hubbard R, Liu SLJ, *et al.* A community study of the effect of particulate matter on blood measures of inflammation and thrombosis in an elderly population. *Environ Health* 2007; 6: 3.
- 61 Larson T, Su J, Baribeau AM, *et al.* A spatial model of urban winter woodsmoke concentrations. *Environ Sci Technol* 2007; 41: 2429–2436.
- 62 Gehring U, Tamburic L, Sbihi H, *et al.* Impact of noise and air pollution on pregnancy outcomes. *Epidemiology* 2014; 25: 351–358.
- 63 Karr CJ, Demers PA, Koehoorn MW, *et al.* Influence of ambient air pollutant sources on clinical encounters for infant bronchiolitis. *Am J Respir Crit Care Med* 2009; 180: 995–1001.
- 64 MacIntyre EA, Karr CJ, Koehoorn M, *et al.* Residential air pollution and otitis media during the first two years of life. *Epidemiology* 2011; 22: 81–89.
- 65 Gan WQ, FitzGerald JM, Carlsen C, *et al.* Associations of ambient air pollution with chronic obstructive pulmonary disease hospitalization and mortality. *Am J Respir Crit Care Med* 2013; 187: 721–727.
- 66 Clark NA, Demers PA, Karr CJ, *et al.* Effect of early life exposure to air pollution on development of childhood asthma. *Environ Health Perspect* 2010; 118: 284–290.
- 67 Wilhelm M, Ghosh JK, Su J, *et al.* Traffic-related air toxics and preterm birth: a population-based case-control study in Los Angeles County, California. *Environ Health* 2011; 10: 89.
- 68 Wilhelm M, Ghosh JK, Su J, *et al.* Traffic-related air toxics and term low birth weight in Los Angeles County, California. *Environ Health Perspect* 2012; 120: 132–138.
- 69 Andersen ZJ, Wahlin P, Raaschou-Nielsen O, *et al.* Ambient particle source apportionment and daily hospital admissions among children and elderly in Copenhagen. *J Expo Sci Environ Epidemiol* 2007; 17: 625–636.
- 70 McGowan JA, Hider PN, Chacko E, *et al.* Particulate air pollution and hospital admissions in Christchurch, New Zealand. *Aust N Z J Public Health* 2002; 26: 23–29.
- 71 Sanhueza PA, Torreblanca MA, Diaz-Robles LA, *et al.* Particulate air pollution and health effects for cardiovascular and respiratory causes in Temuco, Chile: a wood-smoke-polluted urban area. *J Air Waste Manag Assoc* 2009; 59: 1481–1488.
- 72 Johnston FH, Hanigan IC, Henderson SB, *et al.* Evaluation of interventions to reduce air pollution from biomass smoke on mortality in Launceston, Australia: retrospective analysis of daily mortality, 1994–2007. *BMJ* 2013; 346: e8446.
- 73 Jeong CH, Evans GJ, Dann T, *et al.* Influence of biomass burning on wintertime fine particulate matter: source contribution at a valley site in rural British Columbia. *Atmos Environ* 2008; 42: 3684–3699.
- 74 Missoula County. History of Missoula's Air Quality Program. <http://www.co.missoula.mt.us/airquality/MissoulasAir/aqhistory.htm> Date last updated: 1999.
- 75 Noonan CW, Ward TJ, Navidi W, *et al.* A rural community intervention targeting biomass combustion sources: effects on air quality and reporting of children's respiratory outcomes. *Occup Environ Med* 2012; 69: 354–360.
- 76 Ward TJ, Palmer CP, Noonan CW. Fine particulate matter source apportionment following a large woodstove changeout program in Libby, Montana. *J Air Waste Manag Assoc* 2010; 60: 688–693.
- 77 Allen RW, Carlsen C, Karlen B, *et al.* An air filter intervention study of endothelial function among healthy adults in a woodsmoke-impacted community. *Am J Respir Crit Care Med* 2011; 183: 1222–1230.

- 78 Kajbafzadeh M, Brauer M, Karlen B, *et al.* The impacts of traffic-related and woodsmoke particulate matter on measures of cardiovascular health: a HEPA filter intervention study. *Occup Environ Med* 2015; 72: 394–400.
- 79 Henderson SB, Johnston FH. Measures of forest fire smoke exposure and their associations with respiratory health outcomes. *Curr Opin Allergy Clin Immunol* 2012; 12: 221–227.
- 80 Youssouf H, Liousse C, Roblou L, *et al.* Non-accidental health impacts of wildfire smoke. *Int J Environ Res Public Health* 2014; 11: 11772–11804.
- 81 Morgan G, Sheppeard V, Khalaj B, *et al.* Effects of bushfire smoke on daily mortality and hospital admissions in Sydney, Australia. *Epidemiology* 2010; 21: 47–55.
- 82 Henderson SB, Brauer M, Macnab YC, *et al.* Three measures of forest fire smoke exposure and their associations with respiratory and cardiovascular health outcomes in a population-based cohort. *Environ Health Perspect* 2011; 119: 1266–1271.
- 83 Rappold AG, Stone SL, Cascio WE, *et al.* Peat bog wildfire smoke exposure in rural North Carolina is associated with cardiopulmonary emergency department visits assessed through syndromic surveillance. *Environ Health Perspect* 2011; 119: 1415–1420.
- 84 Straney L, Finn J, Dennekamp M, *et al.* Evaluating the impact of air pollution on the incidence of out-of-hospital cardiac arrest in the Perth Metropolitan Region: 2000–2010. *J Epidemiol Community Health* 2014; 68: 6–12.
- 85 Dennekamp M, Straney LD, Erbas B, *et al.* Forest fire smoke exposures and out-of-hospital cardiac arrests in Melbourne, Australia: a case-crossover study. *Environ Health Perspect* 2015 [In press DOI: 10.1289/ehp.1408436].
- 86 Haikerwal A, Akram M, Del Monaco A, *et al.* Impact of fine particulate matter (PM<sub>2.5</sub>) exposure during wildfires on cardiovascular health outcomes. *J Am Heart Assoc* 2015; 4: e001653.
- 87 Analitis A, Georgiadis I, Katsouyanni K. Forest fires are associated with elevated mortality in a dense urban setting. *Occup Environ Med* 2012; 69: 158–162.
- 88 Sastry N. Forest fires, air pollution, and mortality in southeast Asia. *Demography* 2002; 39: 1–23.
- 89 Bølling AK, Totlandsdal AI, Sallsten G, *et al.* Wood smoke particles from different combustion phases induce similar pro-inflammatory effects in a co-culture of monocyte and pneumocyte cell lines. *Part Fibre Toxicol* 2012; 9: 45.
- 90 Londahl J, Massling A, Swietlicki E, *et al.* Experimentally determined human respiratory tract deposition of airborne particles at a busy street. *Environ Sci Technol* 2009; 43: 4659–4664.
- 91 Londahl J, Pagels J, Boman C, *et al.* Deposition of biomass combustion aerosol particles in the human respiratory tract. *Inhal Toxicol* 2008; 20: 923–933.
- 92 Kristensson A, Rissler J, Londahl J, *et al.* Size-resolved respiratory tract deposition of sub-micrometer aerosol particles in a residential area with wintertime wood combustion. *Aerosol Air Qual Res* 2013; 13: 24–35.
- 93 Jalava PI, Aakko-Saksa P, Murtonen T, *et al.* Toxicological properties of emission particles from heavy duty engines powered by conventional and bio-based diesel fuels and compressed natural gas. *Part Fibre Toxicol* 2012; 9: 37.
- 94 Happonen MS, Uski O, Jalava PI, *et al.* Pulmonary inflammation and tissue damage in the mouse lung after exposure to PM samples from biomass heating appliances of old and modern technologies. *Sci Total Environ* 2013; 443: 256–266.
- 95 Uski OJ, Happonen MS, Jalava PI, *et al.* Acute systemic and lung inflammation in C57Bl/6J mice after intratracheal aspiration of particulate matter from small-scale biomass combustion appliances based on old and modern technologies. *Inhal Toxicol* 2012; 24: 952–965.
- 96 Bruns EA, Krapf M, Orasche J, *et al.* Characterization of primary and secondary wood combustion products generated under different burner loads. *Atmos Chem Phys* 2015; 15: 2825–2841.
- 97 Nordin EZ, Uski O, Nystrom R, *et al.* Influence of ozone initiated processing on the toxicity of aerosol particles from small scale wood combustion. *Atmos Environ* 2015; 102: 282–289.
- 98 Stockfelt L, Sallsten G, Olin AC, *et al.* Effects on airways of short-term exposure to two kinds of wood smoke in a chamber study of healthy humans. *Inhal Toxicol* 2012; 24: 47–59.
- 99 Barregard L, Sallsten G, Andersson L, *et al.* Experimental exposure to wood smoke: effects on airway inflammation and oxidative stress. *Occup Environ Med* 2008; 65: 319–324.
- 100 Ghio AJ, Soukup JM, Case M, *et al.* Exposure to wood smoke particles produces inflammation in healthy volunteers. *Occup Environ Med* 2012; 69: 170–175.
- 101 Sehlstedt M, Dove R, Boman C, *et al.* Antioxidant airway responses following experimental exposure to wood smoke in man. *Part Fibre Toxicol* 2010; 7: 21.
- 102 Forchhammer L, Loft S, Roursgaard M, *et al.* Expression of adhesion molecules, monocyte interactions and oxidative stress in human endothelial cells exposed to wood smoke and diesel exhaust particulate matter. *Toxicol Lett* 2012; 209: 121–128.
- 103 Riddervold IS, Bonlokke JH, Molhave L, *et al.* Wood smoke in a controlled exposure experiment with human volunteers. *Inhal Toxicol* 2011; 23: 277–288.
- 104 Barregard L, Sallsten G, Gustafson P, *et al.* Experimental exposure to wood-smoke particles in healthy humans: effects on markers of inflammation, coagulation, and lipid peroxidation. *Inhal Toxicol* 2006; 18: 845–853.
- 105 Unosson J, Blomberg A, Sandstrom T, *et al.* Exposure to wood smoke increases arterial stiffness and decreases heart rate variability in humans. *Part Fibre Toxicol* 2013; 10: 20.
- 106 Tan WC, Qiu D, Liam BL, *et al.* The human bone marrow response to acute air pollution caused by forest fires. *Am J Respir Crit Care Med* 2000; 161: 1213–1217.
- 107 Swiston JR, Davidson W, Attridge S, *et al.* Wood smoke exposure induces a pulmonary and systemic inflammatory response in firefighters. *Eur Respir J* 2008; 32: 129–138.
- 108 Seagrave J, McDonald JD, Reed MD, *et al.* Responses to subchronic inhalation of low concentrations of diesel exhaust and hardwood smoke measured in rat bronchoalveolar lavage fluid. *Inhal Toxicol* 2005; 17: 657–670.
- 109 Barrett EG, Henson RD, Seilkop SK, *et al.* Effects of hardwood smoke exposure on allergic airway inflammation in mice. *Inhal Toxicol* 2006; 18: 33–43.
- 110 Burchiel SW, Lauer FT, Dunaway SL, *et al.* Hardwood smoke alters murine splenic T cell responses to mitogens following a 6-month whole body inhalation exposure. *Toxicol Appl Pharmacol* 2005; 202: 229–236.
- 111 Reed MD, Campen MJ, Gigliotti AP, *et al.* Health effects of subchronic exposure to environmental levels of hardwood smoke. *Inhal Toxicol* 2006; 18: 523–539.
- 112 Tesfaigzi Y, McDonald JD, Reed MD, *et al.* Low-level subchronic exposure to wood smoke exacerbates inflammatory responses in allergic rats. *Toxicol Sci* 2005; 88: 505–513.

- 113 Samuelsen M, Nygaard UC, Lovik M. Allergy adjuvant effect of particles from wood smoke and road traffic. *Toxicology* 2008; 246: 124–131.
- 114 Migliaccio CT, Kobos E, King QO, *et al.* Adverse effects of wood smoke PM<sub>2.5</sub> exposure on macrophage functions. *Inhal Toxicol* 2013; 25: 67–76.
- 115 Rylance J, Fullerton DG, Scriven J, *et al.* Household air pollution causes dose-dependent inflammation and altered phagocytosis in human macrophages. *Am J Respir Cell Mol Biol* 2015; 52: 584–593.
- 116 Tapanainen M, Jalava PI, Maki-Paakkanen J, *et al.* Efficiency of log wood combustion affects the toxicological and chemical properties of emission particles. *Inhal Toxicol* 2012; 24: 343–355.
- 117 Torvela T, Uski O, Karhunen T, *et al.* Reference particles for toxicological studies of wood combustion: formation, characteristics, and toxicity compared to those of real wood combustion particulate mass. *Chem Res Toxicol* 2014; 27: 1516–1527.
- 118 Danielsen PH, Loft S, Kocbach A, *et al.* Oxidative damage to DNA and repair induced by Norwegian wood smoke particles in human A549 and THP-1 cell lines. *Mutat Res* 2009; 674: 116–122.
- 119 Kocbach A, Herseth JI, Lag M, *et al.* Particles from wood smoke and traffic induce differential pro-inflammatory response patterns in co-cultures. *Toxicol Appl Pharmacol* 2008; 232: 317–326.
- 120 Kocbach A, Namork E, Schwarze PE. Pro-inflammatory potential of wood smoke and traffic-derived particles in a monocytic cell line. *Toxicology* 2008; 247: 123–132.
- 121 Moller P, Loft S. Oxidative damage to DNA and lipids as biomarkers of exposure to air pollution. *Environ Health Perspect* 2010; 118: 1126–1136.
- 122 Sevastyanova O, Binkova B, Topinka J, *et al.* *In vitro* genotoxicity of PAH mixtures and organic extract from urban air particles part II: human cell lines. *Mutat Res* 2007; 620: 123–134.
- 123 Riddervold IS, Bonlokke JH, Olin AC, *et al.* Effects of wood smoke particles from wood-burning stoves on the respiratory health of atopic humans. *Part Fibre Toxicol* 2012; 9: 12.
- 124 Wagner F, Amann M, Bertok I, *et al.* NEC Scenario Analysis Report Nr. 7. Baseline emission projections and further cost-effective reductions of air pollution impacts in Europe – a 2010 perspective. Laxenburg, International Institute for Applied Systems Analysis, 2010.
- 125 European Commission. EC (2013) Commission staff working document: Impact assessment SWD(2013)531. Brussels, European Commission, 2013.
- 126 European Environment Agency. Air quality in Europe — 2014 report. Luxembourg, European Environment Agency, 2014.
- 127 Chafe Z, Brauer M, Heroux ME, *et al.* Residential heating with wood and coal: health impacts and policy options in Europe and North America. Copenhagen, WHO Regional Office for Europe, 2015.
- 128 Brunekreef B, Künzli N, Pekkanen J, *et al.* Clean air in Europe: beyond the horizon? *Eur Respir J* 2015; 45: 7–10.

### EPA: HSC BIOMASS PLANT'S PERMIT NOT RENEWED FOR 20 YEARS

1. The EPA found that **the air district had not renewed the biomass plant's permit in over 20 years**, violating its own rules and federal regulations. Both sets of rules have the same requirements: permit renewal every 5 years in a process that includes public notification and comment, EPA review and approval, and final action by the district within 18 months. Instead of following the law, the district repeatedly and unilaterally extended the 1998 permit's expiration date, avoiding public and EPA oversight.
2. The EPA found that **the air district was wrong to say HSC qualified to operate legally after its permit expired in July 2023**. To qualify, the renewal application had to be submitted no less than 6 months prior to expiration. HSC's application was 4 months late.
3. While the plant has been operating for 20 years without an approved permit, **the EPA found a loophole to make it legal**. Because the owners submitted a permit renewal application on time back in 2003, the plant qualified to keep operating pending air district action. Because the air district never acted, the EPA said that waiver is still in effect.

#### Why it matters

Being legal on a technicality doesn't mean that nobody got hurt. One of the main reasons the Clean Air Act requires permit renewal every 5 years is to keep major sources of pollution up to date with the latest emissions and operating standards. EPA regulations for biomass plants were first adopted in 2011 but these and subsequent revisions were never incorporated into HSC's permit. **Important federal standards were not complied with or enforced for over a decade**, leading to increased exposure to air pollutants that cause heart and lung disease, cancer, and premature death.

RCEA has used air district assurances to counter critics' claims that HSC's biomass, which makes up 40% of RCEA's renewable energy portfolio, is dirty energy from a plant that repeatedly violates the Clean Air Act. The EPA's findings make it clear that the air district has a long history of failure to enforce the Clean Air Act and of making decisions which put the welfare of the biomass plant's wealthy owners before interests of the public. RCEA has a duty to exercise its own due diligence when there are questions about one of its suppliers.

#### Air district just approved more biomass pollution

The air district's lack of concern for public health was exhibited again recently by its approval of changes at the plant that will lead to as much as a 5 fold increase in particulate emissions. The Humboldt Coalition for Clean Energy asked the air district to require installation of better pollution controls and monitoring equipment, which they have the authority to do, but these requests were denied. The Fisher Family, which owns a controlling interest in Humboldt Sawmill Company, has a net worth of over ten billion dollars.

The EPA's report and other relevant source documents can be accessed at:

[https://drive.google.com/drive/folders/149naU1Rr7T9DHv7oxInoAMh5hAi\\_VYke?usp=sharing](https://drive.google.com/drive/folders/149naU1Rr7T9DHv7oxInoAMh5hAi_VYke?usp=sharing)