An Assessment of Risk from Particulate Released from Outdoor Wood Boilers

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ABSTRACT

Use of outdoor wood boilers (OWB) has increased due to cost of fossil fuels. OWB short stacks release particles close to the breathing level, producing high levels of particulate matter ≤ 2.5 μm in diameter ($PM_{2.5}$). This assessment determines OWB contribution to local cancer risk and estimates thresholds for acute non-cancer risks. Carcinogenic PAHs in wood smoke ($PM_{2.5}$) cancer risks range from 2.7×10^{-3} for the upper bound scenario (95% UCL value of $PM_{2.5}$ (665 μg/m^3)) to 7.6×10^{-5} for the lower bound (mean (186 μg/m^3)). These risks represent a 7-fold increase of acceptable cancer risk for the lower bound value and 2 orders of magnitude above acceptable levels for the upper bound values. Non-cancer effects such as asthma and cardiopathies include respiratory attacks, hospital emergency room visits, and hospitalizations. Inhaled dose acute risk thresholds of 96, 120, and 250 μg PM 2.5/6 hours are proposed. Operation of an OWB that emits 100 grams $PM_{2.5}$/h was modeled and found to increase the exposures that exceed the 120-μg-risk level at and in residences within 500 to 1000 feet. The increases are projected to occur during periods of poor air mixing due to decreased wind speeds or inversions. Our analysis proposes a 6-h $PM_{2.5}$ inhaled dose threshold to predict peak periods of unhealthy air quality instead of 24-h and annual averages standards, which mask peak emissions.

Key Words: wood boilers, particulate, respiratory disease, risk assessment, air quality index.

INTRODUCTION

Outdoor wood boilers (OWB) are gaining popularity in their bid to serve as an alternative heating source. Typically, an OWB is a wood-burning firebox surrounded by a water jacket vented by a chimney stack. Wood is burned and heats the water, which is then pumped into the home or other building through insulated underground pipes. The associated combustion of the wood in an OWB produces dense emissions of wood smoke at ground level. The human exposures that occur are

substantially different from the exposures from typical wood stoves. The assessment of the human health risk from wood smoke is itself a challenge because of the presence of mixtures of carcinogens and air toxics in the presence of fine particles equal to or less than 2.5 μm (PM$_{2.5}$). It is necessary to both determine cancer risks from chronic exposures and evaluate the respiratory and cardiovascular risks from acute and sub-chronic exposures. It is also necessary to assess the synergism in the exposure induced when the fine particulate alter the distribution of the water soluble gases from the upper respiratory tract to the deep lung. Therefore this assessment of the risk requires the simultaneous application of the classic carcinogenic risk assessment methodology and the determination of thresholds for acute risks.

The analysis is separated into two parts: Part 1 assesses the cancer risk from the mixture of carcinogenic polycyclic aromatic hydrocarbons (PAHs) present, whereas Part 2 assesses the threshold of acute respiratory and cardiovascular risks from the entire mixture. Finally, OWBs produce emissions under two different conditions (oxygen-rich and oxygen-starved) so that it is necessary to consider differences in the consistency of the mixture during different time periods.

Wood Smoke from OWB

Wood smoke is made up of both gaseous and particulate components such as volatile organic compounds (VOCs), PAHs, metals, dioxins, and furans. Of major interest is the fine particle portion of wood smoke (PM$_{2.5}$) because it is linked to asthma, chronic obstructive pulmonary disease (COPD), cardiac effects, and lung cancer (reviewed by Naeher *et al.* 2005).

Indoor wood stoves and OWBs are both sources of significant PM$_{2.5}$ contamination (Johnson 2006). OWB pollution is exacerbated because the low stack design does not disperse the smoke as well as conventional chimneys. Johnson (2006) shows that release of emissions from a residential OWB can produce episodes of very high ambient levels of PM$_{2.5}$. Human exposure levels are dependent on: operating conditions of the boiler, type of fuel; time elapsed from the addition of fuel to the boiler, local weather, and activity patterns and location of those living or working nearby. These parameters and topography will determine the amounts of emissions inhaled.

The types of chemicals and particulates present in wood smoke and the risk parameters have been identified (USEPA 1988, 1993, 1998; Johnson 2006). Long-term exposures over time raise the lifetime cancer potential and appear to produce chronic changes in the lung or cardiovascular systems. Simultaneously, short-term exposures increase reports of acute respiratory disease and cardiovascular accidents. The mixture of gases with particulates may cause interactions that increase the exposure intensity and alter the distribution of the gases in the body.

No Standards Exist for Acute or Carcinogenic Risk from OWB Wood Smoke

There is little regulatory guidance or standards applicable to OWB emissions with respect to public health impact. Some states have attempted control through local regulations or ambient air pollution statutes (*e.g.*, NYS EPB 2005). Neither approach

is public-health protective because OWB emissions are episodic and variable,[1] producing health effects in time frames shorter than those addressed by the air standards. For example, the current National Ambient Air Quality Standard for $PM_{2.5}$ is based on a 24-h exposure ($65 \, \mu g/m^3$ and annual mean ($15 \, \mu g/m^3$)) (USEPA 1997). USEPA (2006) has recently published a proposed final rule for lowering the 24-h $PM_{2.5}$ standard to $35 \, \mu g/m^3$, while maintaining the annual standard of $15 \, \mu g/m^3$. Although this new proposed standard is more in line with the Clean Air Scientific Advisory Committee recommended 24-h standard (of no higher than $35 \, \mu g/m^3$ and an annual standard no higher than $14 \, \mu g/m^3$ (USEPA 2005)), there are studies that support that cardiopulmonary health effects are induced by a few hours of exposure (Zanobetti *et al.* 2000; Dockery *et al.* 1993).

The U.S. Environmental Protection Agency's (USEPA's) Air Quality Index (AQI) for particulate PM_{10} (particulates of size 10 μm or less) and $PM_{2.5}$ is an index of probable health risk from particulate matter (PM) based on 12-h averaging that could be applied to short-term health concerns. Five levels of risk are designated based on the review of extensive epidemiologic, case study, and animal research (USEPA 2003). However, the AQI is a regional guidance tool that has not been applied in local settings and does not consider specific cardiopulmonary risks. While it is helpful, the AQI is not designed for OWB-type of problem.

Rationale of this Risk Assessment

The objective of this risk assessment is to determine the contribution of OWB devices to local cancer risk and to estimate the threshold levels of acute non-cancer adverse health effects, most notably cardiac and respiratory outcomes. The release by OWB of $PM_{2.5}$ as measured by Johnson (2006) will be the basis for this risk assessment. Seven of the PAHs in the mixture, listed by the USEPA as carcinogenic (USEPA 2006), are evaluated in Part 1. This risk from the carcinogenic PAH component of $PM_{2.5}$ is assumed to be cumulative with other chemical components of wood smoke. Cancer risks are derived using standard procedures and assumptions based on the organic matter attached to particulates in the smoke.

The acute risk assessment uses a dose-based analysis approach from the perspective of thresholds for acute actions and based on potential 6-hour exposures to particulate $PM_{2.5}$. It has been found that current *ambient* exposure episodes to $PM_{2.5}$ in the Northeast U.S. increase hospitalization rates and emergency room visits for both cardiovascular and respiratory disease after only a few hours of $PM_{2.5}$ exposure (Peters *et al.* 2001; Gent *et al.* 2003). The threshold dose for inducing an acute cardiopulmonary event for each day is used to assess the risk. The approach "unhealthy

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[1]Although wood stove emissions are also episodic and variable, one key difference is that OWB have no emission reduction requirements in their design technology as opposed to USEPA-certified wood stoves, which do. OWB also emit much more PM than indoor stoves.

air day"[2] (UAD) is designed to measure the potential increase in adverse health effects by assessing the risks "unhealthy air days."

METHODOLOGY

The cancer risk assessment utilizes the paradigm outlined by the National Research Council, which consists of: Hazard Identification; Dose-Response Assessment; Exposure Assessment; and Risk Characterization (NRC 1983; USEPA 1989). The risk assessment focuses on particulate matter ($PM_{2.5}$) and gives priority to susceptible subpopulations with lung and cardiovascular disease that respond to the formation of highly respirable particulates. The toxicity of the particles is enhanced by the active absorption of organic matter. Cancer risk is characterized as increased risk due to extractable organic matter (EOM) bound to particulate. Toxicity of elemental components is not considered in this risk assessment. Non-cancer risks are characterized by comparing the estimated [6-h] inhaled doses of OWB-generated PM to ambient air levels that produced respiratory and cardiovascular responses in human studies.

PART 1: CANCER RISK

Hazard Identification

The component of the fine particulate chosen to investigate *cancer* was the seven carcinogenic PAHs identified by the USEPA that pose the greatest risk for cancer: benzo(a)pyrene (BaP), chrysene, dibenzo(a,h)anthracene, indeno(1,2,3 cd)pyrene, benzo(k)fluoranthene, benzo(a)anthracene, and benzo(b)fluoranthene. We did not have quantitative data for dioxins, formaldehyde and benzene—all components of wood smoke—and, therefore, may have underestimated cancer risk.

Comparison of data for concentration calculations

We utilized chemical data from an indoor woodstove (Fine *et al.* 2004) and applied it to an OWB (Johnson 2006). These data are only an *estimate* of potential risk. The wood fuels compared were dry hardwoods. Data from Fine *et al.* (2004) are from a "no emission control unit" on wood stoves. OWBs emit 6.9 times the amount of PAHs than an USEPA-Certified non-catalytic wood stove as estimated in NYS EPB (2005) so the chemical species data from Fine *et al.* (2004) were adjusted for the larger volume OWB. Cooler dilution air *increases* the semi-volatile species in the particle rather than the gas phases. The Johnson (2006) data are from two winter days in the Northeast U.S., therefore the data we are using in this assessment may imply an increased amount of semi-volatiles adsorbed onto the particles. Different stoves and

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[2]An unhealthy air day is defined as a day in which one or more 6-h exposures, 1/4 of a day, occurs in which the inhalation dose of $PM_{2.5}$ exceeds the levels shown to induce respiratory or cardiovascular actions that require hospitalizations or medical attention including use of salvage treatments. There are three dose levels of exposure: "At Risk" (90 ug); "Moderate Risk" (120 ug); and "High Risk" (250 ug) of inhaled $PM_{2.5}$.

Table 1. Percentage of carcinogenic PAHs by weight and cancer slope factors.

Carcinogenic PAHs	Concentrations of PAHs in Red Maple mg/g/OC*	CSF_{inh}** $(mg/kg.d)^{-1}$
Benzo(a)pyrene	0.253	3.9
Chrysene	0.340	0.039
Dibenzo(a,h)anthracene	0.016	4.1
Indeno(1,2,3 cd)pyrene	0.191	0.39
Benzo(k)fluoranthene	0.285	0.39
Benzo(a)anthracene	0.277	0.39
Benzo(b)fluoranthene	0.183	0.39

Data from Fine *et al.* (2004).* OC = organic carbon. From OEHHA 2006 online database.**

different conditions modify the particulate released and weather conditions affect the dispersion and composition of particulate (NESCAUM 2006). Organic carbon (OC) was estimated at 59.4% (Fine *et al.* 2004) (specific to the Red Maple). PAH concentrations are reported in mg/g OC in Table 1.

Dose-Response

The Dose-Response values were equivalents of benzo(a)pyrene (BaP) provided by the California Office of Environmental Health Hazard Assessment (OEHHA 2006). The cancer slope factor for inhalation for BaP is 3.9 $(mg/kg.d)^{-1}$ and fractions of this value for other PAHs are based on their relative toxicity with one exception as shown in Table 1. Concentrations of PAHs in Red Maple by weight are also presented in terms of milligrams (PAH) per gram (of Red Maple) per organic carbon (Fine *et al.* 2004).

Exposure Assessment

Assumptions used for this risk assessment are presented in Table 2. In addition to the typical exposure factors used such as 20 m^3/day inhalation rate and lifetime exposure period of 30 years, a conversion factor is provided that allows scaling differences in burn box capacity (*i.e.*, wood stove *vs.* OWB). An assumption was made that an OWB would operate only 7 months of the year, yielding an exposure frequency of 210 days of use per year. However, this timeframe may be low because OWB are often used year long for supplying hot water beyond the cold weather season in the Northeast U.S. Johnson (2006) reported a mean value air concentration (damper open and closed) of 186 $\mu g/m^3$ and a 95% UCL of 665 $\mu g/m^3$; the maximum concentration (15-s average) was 8880 $\mu g/m^3$. Both the 665 $\mu g/m^3$ UCL and the mean served as the exposure point concentrations (EPC) used to derive cancer risk. The UCL value calculated by Johnson (2006) is a combination of hour 1 after fuel is added "when the values were highest during air intake (p. 1153)" and combined with 22–24 h subsequently.[3]

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[3]The 95th percentile value is from the entire 4.3 h dataset—76% of this time period was sampled 22–24 h after most recent fuel loading; 24% of the time period was sampled 0–1 h after most recent fuel loading. During the time period the damper was open 60% and closed 40% (Johnson 2006).

Table 2. Exposure parameters and risk equation.

Parameter		Value	Source
EPC $_{air}$	Exposure Conc.	0.665 (.186) mg/m^3 (a)	Johnson (2006)
OC	% Organic Carbon	59%	Fine *et al.* 2004(b)
PAH	mg/g OC	chemical specific	Fine *et al.* 2004(b)
C1	Wood Stove to OWB	6.9	NYS EPB (2005)
C2	Conversion factor	0.001 g/mg	Constant
IR	Inhalation rate	20 m^3/day	USEPA 1999
EF	Exposure frequency	210 d/yr	7 month exposure
EP	Exposure period	30 years	USEPA 1999, residence time
BW	Body weight	70 kg	USEPA 1999
AP$_c$	Averaging period	25550 days	USEPA 1999, lifetime, 70 yr
CSF$_{inh}$	Cancer Slope Factor	chemical specific	OEHHA 2006
UF (c)	Uncertainty for upper bound risk only	10	More particulate w/ higher T, dispersion changes, different woods, OC increase (d)

(a) 95% upper confidence limit and (mean).
(b) Red Maple data.
(c) upper bound only.
(d) as described in Discussion section.

Cancer Risk Characterization

Cancer risk calculations combine the exposure point concentrations with the other exposure assumptions detailed in Table 2 to derive the excess lifetime cancer risk as expressed in the following equation:

$$CancerRisk = EPC_{air} * OC * PAH * C1 * C2 * IR * EF * EP * UF/(BW * AP) * CSF_{inh}$$

The resulting range of cancer risks is: 2.7×10^{-3} for the upper bound scenario using 95% UCL value of PM$_{2.5}$ of 665 μg/m^3 and 7.6×10^{-5} for the lower bound using the mean value of PM$_{2.5}$ (186 μg/m^3). This translates into a 7-fold increase of acceptable cancer risk (*lower bound value)* with an upper bound risk of 2 orders of magnitude above acceptable levels within the zone of influence of OWB emissions.

PART 2: NON-CANCER ACUTE AND CHRONIC RISK

The non-cancer risk assessment is designed to measure the threshold at which daily health effects would occur. The acute actions of toxics bound to the particulate are considered. The cardiopulmonary health responses occur after short-term episodes (a few hours of) exposure to particulate in the respirable range (Brook *et al.* 2004). These responses are not accurately assessed by the average 24-h air concentration because daily averaging does not capture time periods of peak exposures. Therefore, an alternative method is needed to evaluate peak exposures. Inhaled dose, rather than concentration in air, partly resolves this problem and is used in the evaluation of acute risk. Further, the Unhealthy Air Day approach is suggested as a unit to measure trends in the acute risks.

Hazard Identification

Particulate matter was identified as the major hazard because of actions on susceptible sub-populations, including those with cardiac and respiratory conditions (Zanobetti *et al.* 2000; Liu *et al.* 2003; Delfino 2006). Johnson and Graham (2005) reviewed how key regulatory and research organizations determined which subgroups were considered to be at elevated risk to PM. The primary concern is the formation of highly respirable particulate, less than $PM_{2.5}$, enhanced by the active absorption of water-soluble organic matter to the particles. Thus, the chemicals of concern are those adsorbed to the particles and the particulate in the $PM_{2.5}$ range or less. Other moieties such as gases that are not as water soluble contribute to the effects seen from epidemiologic and some animal studies. For instance, there are two gases not included in the assessment—carbon monoxide and nitrogen dioxide—linked to biomass combustion, both of which are health hazards. No quantitative measures were found in OWB emissions of either carbon monoxide or nitrogen dioxide. It is recommended that evaluation of risks use the Occupational Safety and Health Administration (OSHA) Time Weighted Average (TWA) of 40 mg/m^3 for carbon monoxide and 9 mg/m^3 for nitrogen dioxide. Some risk assessors may adjust for 24-h exposure.

Other agents such as bioactive-aldehydes and acrolein can be adsorbed to the particulate and transported to the deep lung. The carcinogenic PAHs are also transported to the deep lung and rapidly distributed throughout the system. In the absence of the particulate, the water soluble gases do not reach the deep lung, but are absorbed in the upper respiratory tract and removed from the body, greatly reducing target organ exposure and the accompanying toxicity.

Dose-Response

Animal and human studies show an association between wood smoke exposure and increased visits to the doctor, emergency rooms and hospitalizations. PM_{10} and $PM_{2.5}$ are measured in both the epidemiology and animal studies of wood smoke and its components. Because PM is the major component of wood smoke, it is a surrogate of exposure. The studies show associations with acute and chronic health effects in the ambient air. Wood smoke particulate and ambient air particulate toxicity have been reviewed elsewhere (*e.g.*, Boman *et al.* 2003; Naeher *et al.* 2005; Butterfield *et al.* 1989; Cupitt *et al.* 1994; Dominici *et al* 2006; Koenig *et al.* 1993; ALA 2001; Zelikoff *et al.* 2002).

Wood smoke

As a brief overview, the following health effects have been linked (*ibid.*) to *wood smoke*:

1. Airway changes including hyper responsiveness, lower air way respiratory infections, and inflammation.
2. Bronchiolitis, hyperplasia, and hypertrophy including increase in lung cancer risk.
3. Shortness of breath and moderate to severe cough in asthmatics and waking up with a cough.

4. Inflammation of the middle ear marked by pain, fever and dizziness.
5. Significant decreases in lung function including Forced Ventilatory Capacity (FVC) and 1 minute ventilatory rate (FEV1).
6. A pattern of increased symptoms and chronic illness in children based in part on a compromised immune system.

Particulate matter

Current ambient exposure episodes to $PM_{2.5}$ in the Northeast U.S. increase hospitalization rates for cardiovascular and respiratory disease. More specifically, some of the well established health effects of *ambient particulate matter* include the following (USEPA 2006; Burnett *et al.* 2000; Delfino *et al.* 2002; Dockery 2001; Steib *et al.* 2003):

1. Epidemiology studies report increased cardiovascular events, exacerbation of asthma, and chronic obstructive pulmonary disease as well as links to cancer (Pope *et al.* 2002).
2. Case report studies show increased admission to emergency rooms for both respiratory and cardiovascular events.
3. Some clinical studies demonstrate a protective effect for anti-inflammatory medications.
4. Statistical analyses of some national mortality data show an increase in mortality in areas with higher particulate materials in the ambient air.

Particulate matter exposures also produce biochemical actions at the cellular level (Naeher *et al.* 2005). These biochemical studies suggest plausible modes of action, the release of bioactive materials. This effect that occurs at low doses strongly indicates that direct irritation of the respiratory tract is not the sole basis for a portion of the many health actions. Furthermore, health effects from PM occur after exposures of 2 to 4 h or less in duration of wood smoke at the 12 to 29 $\mu g/m^3$ range (Koenig *et al.* 1993).

Quantitative $PM_{2.5}$ ambient air studies as a quantitative measure for wood smoke

The strongest dose-response information for action between particulate and health effects is found in the reports based on ambient measures of $PM_{2.5}$. Peters *et al.* (2001) and Gent *et al.* (2003) demonstrated that health actions occur after PM exposures of 2 h or less. Peters found an increase in myocardial infarctions 2 h after an increase of 25 $\mu g/m^3$ over background (odds ratio 1.48) and another 24 h later after a 20 $\mu g/m^3$ increase in $PM_{2.5}$ over background (Odds ratio 1.69). Similarly, Gent *et al.* (2003) showed that, within 1 h, groups of children with severe asthma showed 35% increase in wheezing and 47% increase in chest tightness after 50 ppb ozone and 12 to 18 $\mu g/m^3$ $PM_{2.5}$ (odds ratio for chest tightness is 1.24). Dockery and coworkers (1993) found that exposures in the 11 to 29 $\mu g/m^3$ PM (measured as annual PM levels) range revealed a dose-related difference between six cities for the following conditions:

- All cause of death (odds ratio 1.26 (CI[4] 1.08–1.47))
- Lung Cancer (odds ratio 1.37 (0.81–2.31))
- Cardiopulmonary (odds ratio 1.37 (1.11–1.68))

The actions appear to be specific for the cardiopulmonary system and cancer because the odds ratio for "all other causes" studied was 1.01 (CI 0.79–1.30); moreover, there were 2-h and 24-h lags between the increase in PM and the health outcome.

The dose levels showing the above actions are in the 15 to 30 μg/m^3 range. This compares with the 12 to 29 μg/m^3 median values found in wood stove data. Zanobetti and Schwartz's (2003) analysis (and reanalysis) of morbidity found similarly high relative risks. These findings show that increased PM$_{2.5}$ (at levels of 12 to 30 μg/m^3) for 2 to 4 h can induce cardiopulmonary effects in humans.

Exposure Assessment

OWB emissions

Several reports that analyzed exposures to wood smoke in regions with extensive wood burning show that human exposure is related to four variables:

1. The amount of emissions released to the ambient air;
2. The dilution in the ambient air prior to a human inhalation;
3. The amount in indoor air due to penetration from the surrounding ambient air and the time that lapses before indoor air levels off-gas to the outdoors; and
4. The behavioral activity of the persons exposed.

NESCAUM (2006) and the New York Attorney General's reports (NYS EPB 2005) characterized the hourly emissions of PM$_{2.5}$ in the Northeast U.S. and New York State, respectively. The New York Attorney General's office made public 10 measures for OWB that showed hourly releases from 18 g/h to a high of 269 g/h. Based on this collection of findings and our box model described below, it is assumed that the reasonable range for the releases of particulate matter from OWB is 50 to 150 g/h. Valenti and Clayton's report (1998) found emissions of 143 grams per hour (g/h) under high-heat demand conditions and 55.4 g/h under low-heat removal. A study in Vermont concludes that there are releases of 93.76 g/h on average released from wood stoves (Sexton *et al.* 1984).

Assessing potential indoor exposures using a box model[5]

Exposures to indoor air, infiltrated by PM-contaminated "fresh air" will add to a person's total exposure; therefore the importance of indoor exposures should not be overlooked (Abt *et al.* 2000; Meng *et al.* 2005; Molnár *et al.* 2005). Once the interior

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[4]Confidence Intervals.

[5]Assumptions used for the box model: (1) 24-h use for heating of house and water; (2) Damper open 50% of the time; (3) Damper closed 50% of the time; (4) Residence distances of 100, 500, and 1000 feet; (5) Inhalation rate of 0.8 m^3 of air/h; (6) Risk is based on exposures of 6 h or less; (7) Use of the unhealthy air day concept; (8) Health effects have been observed after 6-h exposures to 20, 30, or 40 ug/m^3; (9) Background PM is 12 to 17 ug/m^3; and (10) Low air speed is <2 mph and high air speed is >5 mph.

of the house reaches steady state conditions, it will take several hours (4 to 7) for dilution with cleaner outside air to eliminate the wood smoke $PM_{2.5}$ trapped indoors. Under conditions of episodic emissions (*i.e.*, 5 to 10 min of very high levels of PM as reported by Johnson (2006)) aggregated data [indoor and outdoor] exposures will yield higher than predicted exposures from ambient measures alone. Therefore, a person inside the house will inhale a larger dose than estimated from the average of the 24-h exposure outside.

In order to quantify this, a bounding estimate applicable to the 50 to 150 g/h ranges using an emission level of 100 g/h was evaluated using the simple box exposure model. The concentration of PM in the ambient air is highly dependent on the wind speed and the distance from the source. The model assessed the air stability variable as measured by wind speed and distance from the source. Ground level emissions were used. The emission rate of 100 g/h and background $PM_{2.5}$ of 17 μg/m^3 were used to approximate exposure levels in houses located at different distances from the source. At low wind speeds, 2 mph, the ambient concentrations of $PM_{2.5}$ would be 42 and 27 μg/m^3 at residences 500 and 1000 feet from the source, respectively. At wind speeds of 5 mph the $PM_{2.5}$ ambient concentrations would be 18 and 17 μg/m^3 at residences 500 and 1000 feet from the source, respectively. The number of air changes in a house determines the indoor levels, but the wind speed determines the concentration at the house. Wind speed is thus a strong determinate of the level of exposures found indoors. It is nearly as important as distance from the source, amount of daily emissions, or air exchange rates in the house.[6]

Deriving indoor human inhaled dose at 6 h

As stated earlier, inhaled dose is an appropriate metric to assess the potential for adverse health effects. The inhaled dose was calculated for a resident of a house near an OWB by assuming 1 air exchange per hour for the house. The 6-h periods of highest outside ambient levels of $PM_{2.5}$ were used in order to determine the period of greatest risk. Based on these criteria and the adult inhalation rate (0.8 m^3/h), the inhaled dose of $PM_{2.5}$ was determined to be 130 μg/6 hours for persons 1000 feet from the source and 200 μg/6 h for persons 500 feet from the source when wind speeds are 2 mph. If the wind speed increased to 5 mph, these exposures would fall to 81 and 86 μg/6-h periods, respectively.

When the inhaled dose is considered, the activity of the persons determines the actual dose inhaled. The average inhalation rate, 0.8 m^3/h, used in the example above may be too low. During sleep that rate would be less; and during moderate activity, it could be 50 to 100% higher, yielding a greater inhaled dose.

Non-Cancer Risk Characterization

Acute inhalation risk is based on the increase in number of unhealthy air days based on 6-h exposures. It is assumed that, once the inhaled dose threshold for a

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[6]Very low wind speeds of less than 2 mph tend to occur in the morning and evening period during which time the OWB output would be highest due to heat demand. Therefore, emission clouds stay near the ground with little dilution. During this period the actual amount that penetrates the house is high and remains high in the house for several hours even when the wind speed increases, diluting the ambient PM due to increased mixing.
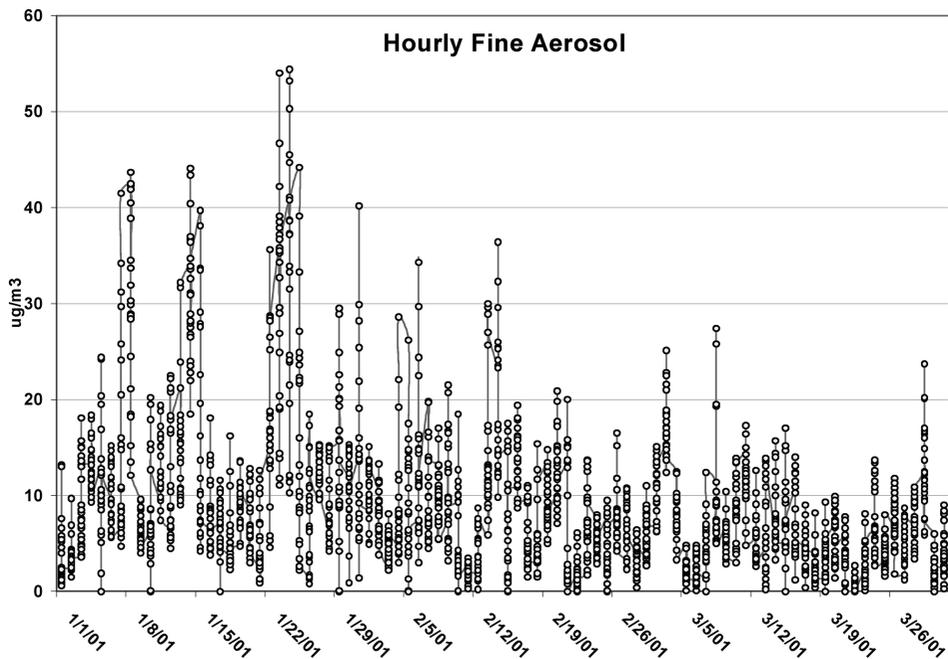
Figure 1. Example of actual measures from a PM$_{2.5}$ monitor in Connecticut. The data collected for compliance purposes would be reported as 9.2 μg/m^3, whereas daily and hourly exposures range over 40 μg/m^3 (22% of the days exceed 30 μg/m^3 for 6 h) (Brown *et al.* 2005, 2006).

health effect occurs on a day, the health event is established for that day. Accurately characterizing a 6-h exposure is important. Using a metric such as an average daily concentration would underestimate the maximum 2- to 4-h exposures. Shorter averaging times of 6 h capture the episodic exposures that are associated with respiratory or cardiovascular responses with minimal sampling variability.

The 6-h total inhaled dose in micrograms of PM is a reproducible metric for dose-response, especially under conditions of high variability in air, such as exist for PM$_{2.5}$ even in the absence of OWB. Figure 1 illustrates how a 3-month long series of hourly observations would be collapsed into a single value of 9.2 μg/m^3, highlighting the flaw in using aggregate measures to characterize acute exposure to PM. Alternatively, a 6-h averaging period involves one-fourth of the daily inhalation and provides a representative measure of the personal exposure doses during different daily activity cycles. Each bar in Figure 1 represents a single day; each dot, an hourly measurement on that day. Because there are no sources close to the monitors, the daily variability reflects differences in dilution volumes (mixing depth) due to local weather changes in wind speed and not variable effects from source differences. Johnson (2006) notes similar variability with time and movement of the plume over the monitor during a period of low air mixing. In Connecticut, the higher PM values generally occur in the morning or early evenings due to changes in insolation from the sun and variable weather or wind patterns.

Table 3. Air quality classification and corresponding ambient and inhaled dose of $PM_{2.5}$.

Air quality	PM concentration	6-hour inhaled dose
GOOD	0–20 μg/m^3	Less than 96 μg
MODERATE	21–40 μg/m^3	96–192 μg
UNHEALTHY FOR SENSITIVE GROUPS	41–60 μg/m^3	193–288 μg
UNHEALTHY FOR ALL	61–80 μg/m^3	288–384 ug
VERY UNHEALTHY	81–120 μg/m^3	385–586 μg

The Air Quality Index (AQI) was designed by the USEPA to provide warnings of the sub-daily (less than 24 hours) elevations in exposure to $PM_{2.5}$ or PM_{10}. The inhaled dose exposures were derived from the AQI data and the epidemiologic cardiopulmonary dose-response findings. The range of doses at which health effects are expected is outlined in Table 3.

The AQI scale is not linked to specific health outcomes and is not a specific scale of respiratory of cardiovascular risk. The dose-response evaluation shows adverse health effects from wood smoke in the 12–39 μg/m^3 range and particulate responses in the 15–30 μg/m^3 range. Acute respiratory attacks and cardiovascular incidents occur after an exposure of a few hours. In order to adjust for this factor we developed the Unhealthy Air day as a measure of risk.

Unhealthy Air Day (UAD) concept for quantitative acute risk assessment

The unhealthy air day model is described in the Connecticut Fund for the Environment report *Diesel Emissions and Unhealthy Air* and *Hot Spots and Health Risks Diesel Construction in Connecticut* (Brown *et al.* 2005, 2006). The Unhealthy Air Day (UAD) metric is a quantitative measure that adjusts for the hour-to-hour variability in maximal exposure that normally occurs in ambient PM, as illustrated in Figure 1. The UAD-inhaled dose response for the health risk is defined as a day when there is one or more contiguous 6-h period(s) where the inhaled dose of $PM_{2.5}$ exceeds the threshold for a cardiopulmonary health effect (Brown *et al.* 2005). This model is based on the 6-h dose of $PM_{2.5}$ and three levels of risk. A day would be considered an unhealthy air day according to the three criteria presented in Table 4.

This scale translates into 6-h average exposures of 19, 25, and 53 μg/m^3 due to the OWB particulate emissions. All of the exposure could occur in a few minutes as found in the Johnson report (2006).[7]

DISCUSSION

Our analysis concluded that those within the zone of influence of OWB emissions have a 7-fold increase of acceptable cancer risk (*lower bound value)* with an upper bound risk of 2 orders of magnitude above acceptable levels.

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[7]These exposures are indoor or personal exposure approximations. The "At Risk," "Moderate Risk," and "High Risk" scale is based on the expectation of a specific set of health outcomes in susceptible persons. An alternative is to use the USEPA AQI scale (converted to 6-h exposure doses). However, that scale is based on population data rather than individual case findings.

Table 4. Risk categories, 6-hour inhaled dose and corresponding health effects.

Risk category, UAD	6-hour inhalation dose	Health effect
At risk	96 μg or more	Asthma attacks increase
Moderate risk	120 μg or more	Medical intervention, COPD, asthma
High risk	250 μg or more	Hospital or ER visit, asthma or cardiovascular events

The analysis also shows an increase in the unhealthy air days at levels that induce acute respiratory and cardiovascular disease. The precise identification of the number of persons at increased risk is dependent on factors specific to locations and the size of OWB. If the amount of particulate released is as much as 100 g/h, inhalation doses of 130 to 200 μg/6-h periods would occur in persons who live between 500 and 1000 feet; the inhaled doses exceed the UAD threshold of 120 μg/6-h period, increasing risk for cardiovascular and pulmonary attacks. If the emissions exceed the 250 μg/h estimates, the risk would be in the range of increased emergency room visits and hospitalizations. If there are more than one OWB in a topographically restricted area, the chances are increased of exposures that exceed the UAD.

The exposure assessment example described how wind speed substantively controls the exposure outside and inside the houses. A reduction of wind speed is sufficient to produce a 6-h exposure inside a house that will create an unhealthy air day. Distance in itself is not sufficient to assure acceptable exposure levels. At low wind speeds under stable conditions the plume travels many meters with little dilution, placing distant houses at risk. Data from Johnson (2006) show the futility of short-term monitoring to evaluate such risks because the plume changes direction under typical low-wind speed conditions.

Two obvious recommendations to reduce risk are to increase the height of the stack and increase the distance to the nearest house or other building. However, enough is known about the parameters that influence human exposures to raise concern that such actions will fail to address the public health problem. For instance, topography plays a clear role in influencing exposures, and one that cannot be reduced in and of itself. It is very likely that a handful of OWBs or few dozen wood stoves could fill up a valley, home to thousands of people during an inversion (Luhar 2006; Noullett 2006; Brown *et al.* 2005, 2006). From a regulatory standpoint, an alternative approach for reducing exposures to particles is suggested by Johnson and Graham (2005).

There are regions in New England that exceed UAD-inhaled risk levels based only on current ambient levels of $PM_{2.5}$. For example, using Connecticut data depicted in Figure 1 (Brown 2005), the average exposure in suburban areas in Connecticut would be 9, 15, and 17 μg/m^3. This is based on the assumptions that the 50th percentile day has moderate air mixing, the 75th percentile day has lower air mixing and the 90th percentile day has poor or stagnant air mixing, Thus 50% of the days a year exceed 9 μg/m^3 or a 6-h dose of 54 ug, 25% of the days exceed 15 μg/day or 90 μg/6-h and 10% of the days exceed 17 μg/m^3 or 102 μg/6-h

Hum. Ecol. Risk Assess. Vol. 13, No. 1, 2007

203

period.[8] Persons in the zone of influence of an OWB will experience higher exposures on those days, possibly moving them to a higher risk category. The number of Unhealthy Air Days would also be increased.

During a period of low wind speed (less than 2 mph), based on the example in this report, persons 500 feet and 1000 feet would be exposed to a 6-h dose of 200 and 300 μg, respectively, placing them in the moderate to high risk category for that day. These exposures are consistent with increased hospitalizations for Chronic Obstructive Pulmonary Disease (COPD), cardiovascular actions, and asthma medication use as well as increased bronchitis seen in ambient air PM studies. In order to be fully protective, the inhalation exposures for 6 h would have to be less than 90 μg.[9]

Precise identification of the number of persons at increased risk is dependent on factors specific to locations and the size of OWB. The current USEPA cancer risk guidance of 1×10^{-5} should be implemented based on plausible modeling of the location and surrounding areas. Based on this risk assessment it requires that the average annual $PM_{2.5}$ exposures to wood smoke emissions be no greater than 6 μg/m^3, based on the upper bound scenario using conservative parameters. The upper bound risk calculation is a factor of 10 higher based on an assumption that more semi-volatiles would adhere to particles in the lower temperature of the Northeast U.S. than in the Los Angeles area where Fine and his coworkers (2004) completed their analysis. Another factor that would add to the risk include the possibility that organic content may be higher in the Northeast trees than expected, that heating water over 12 months rather than 7 was likely, and the other carcinogens such as benzene, formaldehyde, and dioxins (wood smoke combustion by-products) would add to risk.

The UAD approach allows a regulator to use inhalation dose as a threshold as the point of discrimination. The UAD inhalation dose approach offers advantages over the AQI concentration range approach. First, it aggregates the exposures based on multiple wind speeds, dilution volumes, indoor and outdoor locations and background $PM_{2.5}$ measures. The regulator can decide the level of protection desired. Risk levels can be categorized similarly to those in Table 4. "At Risk" is where asthma responses are expected; "Moderate Risk" is where victims begin to seek medical intervention; "High Risk" is where emergency room visits and hospitalizations begin. No safety or uncertainty factors are incorporated in these thresholds.

Our study has several limitations. One was that we considered emissions from just one OWB. Exposures stemming from a community where several OWB were operating would pose yet another factor that would substantially increase exposures.

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[8]The periods of reduced air mixing and dilution are dependent on meteorological conditions present over large regions of the state at the same time. Those are surface wind speed, Boundary layer depth, Pasquill stability, class, and ambient temperature. (The dew point incorporates some of these factors.) Sexton *et al.* (1984). characterized these factors for Rutland, Vermont, that are similar to locations in the Northeast U.S. A profile of exposures for a "hot spot" location would be developed using the occurrence of these factors.

[9]The NESCAUM (2006) report presents profiles of ambient impact (per pound/h emitted) closer to the OWB, set back distances of 25 to 100 feet. If 50 to 80% of the PM is exchanged into a house an unhealthy 6-h dose is consistent with the simplified example shown and an unhealthy air day would occur.

We also only consider a portion of the components of wood smoke. Further, the synergistic responses are not specifically evaluated although may have some consideration in synergistic effects implied in epidemiological studies.

CONCLUSION

Based on our evaluation of the data from OWBs it appears that USEPA's 1997 accepted fine particle standard ($PM_{2.5}$) of 65 $\mu g/m^3$ (mean per year) is not health protective. Although USEPA (2006) has recently published a proposed final rule for a lower value (35 $\mu g/m^3$), implementation of the new 24-h $PM_{2.5}$ standard may still not be adequately protective from both a cancer and non-cancer health effects perspective. From a cancer risk of 2.7×10^{-3} (upper bound risk), we find a 2-order of magnitude reduction of $PM_{2.5}$ would be needed to produce an acceptable 10^{-5} risk. The acceptable concentration would be approximately 6 $\mu g/m^3$ fine particulate concentration. The PM value correlating with the lower bound scenario (using a mean of 186 $\mu g/m^3$) would be 24 $\mu g/m^3$. These values are roughly consistent with data from the Harvard Six Cities Study (Dockery *et al.* 1993) that demonstrate a correlation between fine particle exposure (range 11–29.6 $\mu g/m^3$) with lung cancer (odds ratio 1.37).

From an acute perspective, a 1-h increase in exposures from OWB may result in experiencing an unhealthy air day, as defined earlier. Children may be in a susceptible group since their lungs are not fully developed and a child breathes 50% more air per kilogram of body weight than an adult. Other susceptible sub-populations also exist such as older persons and those with infirmities. In addition, submicron particles penetrate residences easily during the normal air exchange each day, thus facilitating additional exposure inside the home.

The presence of an outdoor wood boiler near residences and other buildings and other populations constitutes both a cancer and non-cancer health risk that is substantially in excess of risk from the use of indoor wood stoves certified under current USEPA guidelines. OWB particulate emissions are not characterized with respect to EOM content during high oxygen and low oxygen conditions. Based on the wood stove studies the magnitude of the exposures will induce serious health effects. In order to accurately assure that 6-h exposures do not occur that are life threatening, air mixing conditions that include the parameters of hourly wind speed, mixing depths and temperatures are needed at each site.

In summary, cancer appears to be the sensitive endpoint with a 7-months-a-year, lifetime exposure of 6 $\mu g/m^3$: it yields over 1 in 100,000 risk of cancer; presented earlier in Table 3, an exposure level of 18 $\mu g/m^3$ (over 6 h) puts people at risk for health problems like asthma. Other risk levels highlighted in Table 5 include: exposures to concentrations of 24 $\mu g/m^3$ is a moderate risk for hospitalization due to asthma or COPD, whereas exposure levels of 30 $\mu g/m^3$ places people at high risk for serious health problems and hospitalization from asthma, COPD and cardiovascular disease for those most susceptible.

Based on the findings in this report, it is clear that the effects of wood smoke are not insignificant. We compared the wood smoke concentrations determined by Johnson (2006) from the OWB to epidemiologic data presented in Table 4. Effect concentrations are far below the mean of 186 $\mu g/m^3$ and a 95% UCL of 665 $\mu g/m^3$.

Table 5. Health effects *vs.* exposure concentrations fine particles.

Fine particle conc.	Odds ratio or risk	Effect	Source
11–29.6 μg/m^3	1.37	Lung cancer	Dockery (1993)
11–29.6 μg/m^3	1.37	Cardio-pulmonary	Dockery (1993)
25 μg/m^3 (>2 h)	1.48	Myocardial infarction	Peters *et al.* (2001)
20 μg/m^3 (>1 day)	1.69	Myocardial infarction	Peters *et al.* (2001)
6.65 μg/m^3 (7 mo./year)	10^{-5} Lifetime Cancer risk	Cancer	Calculations from PAHs alone, this paper
18 μg/m^3	At risk	Respiratory effects (asthma)	Calculations from Brown *et al.* (2005)
24 μg/m^3	Moderate risk	Exacerbation asthma, COPD	Calculations from Brown *et al.* (2005)
30 μg/m^3	High risk	Respiratory & cardiovascular	Calculations from Brown *et al.* (2005)

The aggregated 24-h and annual average exposure measures would not be a sensitive measure of the relationships between dose and response for acute wood smoke effects of OWB.

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