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Technical Memorandum

Evaluation of Brown et al. (2007) article "An Assessment of Risk from Particulate Released from Outdoor Wood Boilers"

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Executive Summary

Exponent conducted a review and critique of the paper by Brown et al. (2007a), "An assessment of risk from particulate released from outdoor wood boilers," published in *Human and Ecological Risk Assessment (HERA)* in 2007. The paper presents an exposure and risk assessment for particulate matter (PM) associated with emissions from outdoor wood-fired hydronic heaters (OWHH). The paper is divided into a cancer and acute non-cancer risk assessment.

Regarding the cancer risk assessment, we identified the following significant technical flaws:

- Brown et al. incorrectly based his cancer risk assessment on short-term rather than long term exposure measurements. For OWHHs, concentrations will significantly vary depending on meteorological conditions, frequency of use, and the activity patterns of the exposed individuals. However, the Brown et al. assessment is based on only 4.3 hours of air concentration data collected during a time period that is expected to result in relatively high levels.

- The scientific tools are available to conduct a much more accurate assessment of long-term exposure, using air dispersion modeling and time-activity pattern data. However, Brown et al. did not consider such methodologies.

- The measurement device from the Johnson (2006) study, relied upon by Brown et al. for air concentration data, is known to significantly overestimate particulate concentrations.

- Brown et al. applied faulty logic to adjust the Johnson (2006) air concentration data to reflect typical OWHH polycyclic aromatic hydrocarbon (PAH) levels (PAHs are carcinogens). The available data clearly indicate that no adjustment is necessary.

- Brown et al. added an arbitrary and unrealistic 10-fold safety factor to the cancer assessment, with limited explanation and dubious reasoning.

Regarding the acute non-cancer risk assessment, we identified the following significant technical flaws:

- Brown et al. reviews the PM health effects literature with the apparent purpose of developing a novel health index for exposures shorter than 24-hours (the lowest averaging time for the EPA standard is 24 hours). However, they failed to consider that EPA had considered and rejected a shorter-term standard. Also, Brown et al. repeatedly cites published articles to bolster scientific statements made in their report, but a review of many of the articles shows that they did not support the statements attributed to them. These numerous false citations to the scientific literature represent extreme carelessness, at the least.

- Brown et al. develop a unique "unhealthy air day" (UAD) concept, based on a similar methodology used by EPA to characterize previous day air quality. The Brown et al. UAD concept is based on 6-hour average exposures. As noted above, there is no basis for the use of exposure durations less than 24 hours for assessing PM health effects. Also, the basis for Brown et al's UAD benchmarks and associated health effects was found to be from unpublished documents co-authored by Brown that included only scant documentation. There is no scientific basis for the UAD concept developed by Brown et al.

- The paper alleges that "a person inside the house will inhale a larger dose than estimated from the average of the 24-hour exposure outside." However, this statement ignores the fundamental laws of mass balance and shows a complete misunderstanding of ventilation.

- Brown et al. uses a "box model" to estimate short-term concentrations from OWHHs. However, the description provided in the paper is insufficient to replicate what Brown et al. actually did. Furthermore, a box model is a crude methodology; the most scientifically appropriate methodology would have been to use dispersion modeling or to rely on the Johnson (2006) data.

One of the most illogical aspects of the Brown et al. assessment is the estimate of substantially higher concentrations for long-term concentrations for cancer risk assessment (186-665 $\mu g/m^3$) compared to short-term concentrations for the acute non-cancer risk assessment (17-42 $\mu g/m^3$). Clearly, long-term average concentrations should be smaller than short-term averages. Curiously, the data collected by Johnson (2006), used by Brown et al. for the cancer risk assessment, were better suited for the non-cancer risk assessment.

In summary, the Brown et al. article represents extremely poor and sloppy science. It was not worthy of publication in a peer-reviewed journal and it should not be relied upon for policy decisions.

An assessment of the potential health risk for OWHHs could clearly be done much better with the existing data:

- More accurate short-term and long-term exposure concentrations could be developed using dispersion modeling coupled with time-activity data (how much time people spend at home) and ventilation modeling (transfer of particles into and out of residences). A variety of plausible scenarios could be considered that included different distances from the source to residences, different meteorological regimes, and varied time-activity assumptions.

- More traditional methods of assessing PM health risks could be employed, while acknowledging the significant uncertainties involved.

- The risks associated with the different chemical constituents of wood smoke could be estimated using commonly accepted methods for cancer risk assessment, combined with realistic exposure assumptions.

This effort could be aided with the collection of air monitoring data. While it is unrealistic to collect long-term air concentration data (and it would only apply to a single scenario anyway), short-term data would be useful for validating the air modeling. Also, it would be helpful to gather more data on the chemical composition of the wood smoke particles. Such data would provide significant refinement to the cancer risk assessment.

Introduction

The Hearth, Patio and Barbecue Association (HPBA) requested that Exponent review and critique the paper by Brown et al. (2007a), "An assessment of risk from particulate released from outdoor wood boilers," published in *Human and Ecological Risk Assessment (HERA)* in 2007. The Brown et al. paper presents a risk assessment of the health risks associated with particulate matter (PM) from the use of outdoor wood-fired hydronic heaters (OWHH). The paper has already engendered some criticism and controversy. HERA published a letter to the editor from Gradient Corporation criticizing the paper (Long and Valberg, 2007) and Brown provided a response (Brown et al., 2007b).

We have independently reviewed the original Brown paper and the subsequent exchange by Gradient and Brown. We largely agree with the Gradient criticisms of the Brown paper, and have numerous other significant comments and criticisms. Our review included the following elements:

- A review of the scientific basis and reasoning used by Brown et al. to estimate exposures associated with OWHH and attribute health effects to PM from OWHHs.
- A literature search to identify other potentially relevant data sources.
- When possible, reproductions of calculations presented by Brown et al. to better understand their methods.
- A comparison of information presented in the air dispersion modeling study conducted by RTP Environmental Associates to the estimates developed by Brown et al.
- Many of the articles cited by Brown et al. as the basis for their conclusions were acquired and evaluated to determine whether the cited articles support the statements attributed to them.

Our review and critique is divided into the two major sections of the Brown et al. paper, the cancer risk and non-cancer risk assessments. We conclude with recommendations for conducting a state-of-the-art assessment to fully address potential health risks associated with OWHH emissions.

Cancer Risk Assessment

Item 1. Use of Short-Term Data to Evaluate Potential Long-Term Health Effects

The methods used by Brown to assess cancer risk are not consistent with EPA guidelines for cancer risk assessment.

The basis for Brown et al.'s long-term human health risk assessment is only two days of late afternoon/early evening air concentration data, representing a total of 4.3 hours. There are substantial uncertainties inherent in this approach, and these data are clearly not representative of long-term average concentrations. In particular, samples were collected under only one set of meteorological conditions, at one time period (late afternoon/early evening), and were not collected in a manner conducive to evaluating changes in source concentrations over time and local changes in wind speed. For estimating exposure concentrations in air, EPA risk assessment guidance (1989) states, "If long term exposures are being evaluated, the exposure concentration should be representative of long-term averages." Johnson himself refers to the study as a "pilot study …not intended to quantify 24-hour or longer-term average exposures." Given the lack of sufficient samples to characterize both temporal and spatial variability of particulate matter (PM) air concentrations, at a minimum, air dispersion modeling should have been used in the cancer risk assessment, along or in conjunction with the collected air monitoring data. Brown et al.'s use of such a small data set without further derivation of a longer-term average concentration results in an overestimate of long-term health risks.

As discussed in subsequent sections, Brown and colleagues chose to consider varying wind speeds and distances as they derived a 6-hour short-term average concentration for a hypothetical resident located 500 and 1000 ft from a source. However, an analysis considering wind speed and distance to receptors was lacking in their derivation of long-term average concentrations. Rather, they simply used the mean and 95th percentile of 15-second short-term measurements collected over a 4.3-hour period at distances of 50 to 150 ft from a source and assumed that a hypothetical person is repeatedly exposed to that 4-hour concentration, 24 hours per day, 210 days per year over 30 years. The use of 4.3 hours of ambient air concentration data to estimate the exposure for a 30-year period is indefensible. Given the paucity of available air monitoring data, the most scientifically appropriate methodology would have been to use air dispersion modeling to account for the changes in concentrations with different meteorological conditions and time-activity data to account for the inevitable movement of individuals into and out of areas where they may be exposed to particles from OWHHs. The scientific tools to

conduct such an analysis are available and widely used in the exposure and risk assessment fields, including frequent use by EPA and other agencies for use in regulatory decision-making.

Furthermore, in Figure 1 of their article, Brown et al. present data that confirm significant hourly and daily variability from differences in local changes in wind speed. They go on to say, "…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." Johnson collected all 4.3 hours of data in one time period (early evening/late afternoon), a period that Brown et al. acknowledge may result in higher PM values than other periods of the day.

One of our biggest concerns with this analysis is the illogical and inconsistent methodologies that were used to evaluate exposure in the cancer versus non-cancer assessment (discussed in more detail later). Table 1 below summarizes the exposure-point concentrations (EPCs) and methods used in the two evaluations. The approach of using a higher long-term average EPC than that used to evaluate an acute, short-term exposure is illogical. How can the peak exposure that someone receives over a 4-hour period be an order of magnitude lower than the exposure averaged over 30 years?

Table 1. Comparison of cancer and acute exposure methods used by Brown et al.

Assessment	EPCs	EPC Methodology	Relevant EPC Distance
Cancer	186 µg/m^3 and 665 µg/m^3	15-second average $PM_{2.5}$ mean and 95th percentile values over 258 minutes	50–150 ft of OWHH
Non-Cancer Acute	17–42 µg/m^3	Modeled concentrations using 100 g/hour emission rate and incorporating 17 µg/m^3 background $PM_{2.5}$	500 and 1000 ft of OWHH

Item 2: Lack of Consideration of Air Dispersion Modeling Estimates

As discussed above, the $PM_{2.5}$ data presented in the Johnson article are not representative of long-term measurements obtained from a properly installed OWHH or at relevant receptor locations. In fact, Brown et al. acknowledges in their paper that "the concentration of PM in the ambient air is highly dependent on the wind speed and the distance from the source"—neither of which they considered in the evaluation of long-term air concentrations. Using data from tests on properly installed OWHHs, various entities have conducted dispersion modeling to

demonstrate that, when operated with appropriate stack heights (i.e., height of at least 2 ft taller than the tallest adjacent structure), OWHH emissions are much lower than those used by Brown et al. in its long-term cancer evaluation. Dispersion modeling for OWHH has been conducted by the New York State Department of Environmental Conservation (NYSDEC) in support of the NESCAUM model rule for outdoor-wood fired hydronic heaters (NYSDEC, 2007), and by RTP Environmental Associates (RTP Associates, 2007)

RTP Environmental Associates reviewed the January 26, 2007 air dispersion modeling report prepared by NYSDEC and found that, although the NYSDEC modeling approach was consistent with industry practice, the stack and building configurations deviated from OWHH manufacturers' recommendations. Additionally, RTP found that NYSDEC overstated the modeled mass emissions expected from a large OWHH. RTP concluded that NYSDEC's modeled $PM_{2.5}$ impacts were overstated. Even without considering RTP's comments and using NYSDEC's maximum Phase I emission rate of 70 g/hour under the most conservative scenario of a 10-ft stack located 20 ft from the unit, the maximum 24-hour PM concentrations ranged from approximately 70 to 100 $\mu g/m^3$, based on different years of meteorological data.

RTP then modeled a large OWHH with a 0.60-lb/MMBtu emission level. The results of the RTP evaluation indicate that the maximum 24-hour $PM_{2.5}$ impact for a 22-ft stack and 20-ft structure scenario with 0.60 lb/MMBtu and 43% weighted average efficient conditions (approximately 40 g/hour) is 17.6 $\mu g/m^3$. The maximum concentrations occurred within approximately 10 ft of the OWHH and were reduced by one-half their original value (approximately 60 ft) from the maximum point. By contrast, the long-term average concentrations estimated by Brown et al. ranged from 186 $\mu g/m^3$ to 665 $\mu g/m^3$.

It is important to note that the NYSDEC and RTP modeling exercises provide maximum 24-hour concentrations. Annual average 24-hour concentrations are always lower than maximum 24-hour concentration because annual average concentrations include all the meteorological conditions over the year, not just the worst set of 24-hour conditions, and an annual average also considers periods when the source is not emitting. Air dispersion models can be used to estimate the annual average concentration and those are the data that are used to conduct health risk assessments in accordance with EPA guidance.

Air dispersion modeling clearly indicates that, even under very conservative scenarios, 24-hour maximum concentrations result in much lower concentrations than those used by Brown et al. in their evaluation of potential long-term health impacts.

Item 3: Measurement Device Overestimates Particle Concentrations

The cancer risk assessment is based on data presented by Johnson (2006). The Johnson data were collected with a measuring device that may overestimate particle concentrations. In addition to the issues associated with light-scattering devices discussed by others (e.g., Trent 2003, 2006; Fisher and Koshland 2006), Exponent performed an independent literature search of studies that evaluated the performance of a DataRAM unit or other light-scattering devices compared to traditional gravimetric methods.

Liu et al. (2002) evaluated the performance of personal DataRAM units (pDR) and nephelometers with stationary and personal gravimetric sampling methods (Harvard Impactors for $PM_{2.5}$). Samples were collocated with DataRAM samplers in indoor, residential settings. For indoor measurements, the mean pDR/gravimetric ratio was 1.56, indicating that pDR overestimates concentrations by 56%.

Chakrabarti et al. (2004) compared the performance of active-flow, stationary DataRam (pDR-1200) collocated with gravimetric methods. The 2-hour geometric mean of pDR measurements to Beta Attenuation Monitor (BAM; method used as a U.S. EPA equivalent method for 24-hour PM_{10}) resulted in a ratio of 1.33 ($p<0.001$), indicating that the pDR reports concentrations 33% higher than the BAM. Wu et al. (2005) evaluated pDRs in various modes compared to gravimetric methods in indoor, outdoor, and personal environments. The final, corrected median ratios of pDR to gravimetric method (Harvard Impactor) for all pDR types were between 1.2 and 1.9, indicating an overestimation of 20% to 90%.

Based on the recognized bias of the DataRAM, there are considerable uncertainties associated with solely using DataRAM data for quantitative health risk assessment.

Item 4: Fraction of PAHs in Particulates

Brown et al. estimated excess cancer risks by assuming particle air concentration data from Johnson (2006) and assuming polycyclic aromatic hydrocarbon (PAH) speciation data from a study of woodstove emissions by Fine et al. (2004). Brown et al. incorporate an organic carbon concentration of 59% in red maple leaf woodstove smoke reported by Fine et al. (2004), and a woodstove-to-OWHH PAH ratio of 6.9, into the calculation of cancer risk. Brown et al. multiplied the PAH data from indoor wood smoke by 6.9, indicating that "OWBs emit 6.9 times the amount of PAHs than an USEPA-Certified non-catalytic wood stove," based on the report by the New York State Environmental Protection Bureau (NYS EPB 2005). The data from the NYS study are reproduced in Table 2. Brown et al. obtained this factor by simply dividing the

average PAH emission rate (0.96 g/hour) for OWHHs by the average PAH emission rate for EPA-certified non-catalytic wood stoves (0.14 grams/hour).

However, Brown et al.'s analysis is illogical. Brown et al. multiplied the exposure concentration of PM, in milligrams per cubic meter (mg/m^3), by the organic carbon fraction, in percent, and then a chemical-specific PAH fraction, in milligrams per gram (mg/g) organic carbon, as provided by Fine et al. (2004). Because they are using the Johnson (2006) air concentration data for OWHHs, but Johnson did not include PAHs, Brown et al. (1) uses a PAH fraction from the Fine et al. indoor wood stove article, and (2) applies ratio of the PAH concentration from OWHHs and an EPA-certified non-catalytic indoor wood stove from a New York State report (NYS EPB, 2005). However, what is relevant to the evaluation is the PAH/PM ratio for OWHHs versus the PAH/PM ratio for the indoor wood stove. The difference in emissions is already accounted for by using air concentration data from a OWHH use. As shown in Table 2, this fraction is quite similar across these units. Although data for direct comparison are limited, data for two OWHH tests are available, as provided in Appendix A of NYS EPB (2005). As shown in Table 2 below, the average of the ratios of PAH/PM for these two tests is 0.027, which is essentially the same as the PAH/PM ratio for EPA-certified noncatalytic wood stoves. These data indicate that there is no large difference in the fraction of PAH/PM between the two units, and no adjustment is necessary. There are no data to support the finding that a "scaling factor," as used by Brown et al., is justified.

Table 2. PAH/PM ratio for OWHH versus EPA-certified noncatalytic wood stoves

Type of Unit	Average PM (g/hr)	Average PAH (g/hr)	*Fraction of PAH in PM (calculated[a])*
OWHH A	73	1.2	*0.016*
OWHH B	26	0.72	*0.027*
Average of ratios			*0.021*
EPA-certified non-catalytic wood stove	6.0	0.14	*0.023*
OWHH-to-wood-stove ratios	--	6.9[b]	

Notes:

[a] Average PAH value divided by average PM value.

[b] Ratio used by Brown et al.

Item 5: Use of Arbitrary and Unrealistic Uncertainty Factor

As explained in Section 1.2, the exposure used to estimate cancer risks are substantially overestimated, because samples were collected under only one type of meteorological condition,

during one time period, and were not collected in a manner to evaluate changes in source concentrations over time and local changes in wind speed. The other exposure parameters used, such as inhalation rate, exposure duration, exposure frequency, and body weight, are typical default conservative regulatory risk assessment assumptions.

However, Brown et al. add an uncertainty factor of 10 for the upper-bound risk, citing the following reasons: more semivolatiles would adhere to particles in the lower temperatures of the Northeast than in the Los Angeles area, where Fine conducted his study; organic content of trees in the Northeast may be higher than expected; heating water over 12 months rather than 7 is more likely; and other carcinogens such as benzene, formaldehyde, and dioxins would add to risk.

It is common to add uncertainty factors to risk assessment to account for scientific issues that are incompletely understood and to assure that the assessment is protective of public health. For example, when using animal toxicology data in non-cancer assessments, it is typical to add a 10-fold uncertainty factor to account for the uncertainty in extrapolating from animals to humans. However, it is much less common to add uncertainty factors to cancer risk assessments as the potency factors already incorporate conservative assumptions in its derivation and exposure estimates are typically derived using assumptions that are unlikely to underestimate the actual exposure of individuals (as in this case).

The uncertainty factor applied by Brown et al. is arbitrary and unnecessary. Brown et al. did not cite any studies that show that more semivolatiles would adhere to particles at lower temperatures or that there is higher organic content in Northeast trees; if these assertions are true, then they should be quantified rather than applying an arbitrary factor of 10. The use of a 10-fold safety factor for heating water over 12 months rather than 7 is absurd. If it is typical to heat water for 12 months, then 12 months should be used in the risk calculations. Applying a factor of ten for this issue greatly overestimates this parameter (e.g., 210 days of exposure/year × 10 = 2100 days/year). Brown et al. does not cite any sources to support these assumptions, and it is possible that each of these factors (with the exception of the presence of other carcinogens) could also underestimate exposures, making a 10-fold uncertainty factor unnecessarily conservative in a risk assessment that is already highly implausible and conservative. Finally, Brown et al. state that benzene and other chemicals would add to risk. If these chemicals are at issue, then they should be evaluated in the risk assessment, rather than being added arbitrarily. There is no recommendation by EPA to apply an uncertainty factor to the parameters cited. Adding such factors with no discretion adds even greater uncertainty to risk assessment and reduces its value.

Non-Cancer Acute Assessment

For the non-cancer risk assessment, Brown et al. inexplicably uses an entirely different approach to estimate concentrations, constructing a "box model" to estimate fine-particle concentrations 500–1000 ft from a source, based on an assumed emission rate. Oddly, the database they use for cancer risk was far more suited for non-cancer risk assessment. Brown et al. takes a rarely used measure called the "Air Quality Index" (partly relying on an unpublished paper that he wrote) and adjusts it from a 24-hour exposure (EPA's intention) to a 6-hour exposure. They then leap to the conclusion that OWHH emissions cause "acute respiratory and cardiovascular disease."

Item 1: Discussion of PM Health Effects

A vast literature has been published over the last decade or so on the potential health effects of PM. Most of the evidence for health effects comes from epidemiologic studies. These include long-term exposure studies that have found associations between health effects, including mortality, in metropolitan areas and PM concentrations in the areas (e.g., Dockery et al., 1993; Pope et al., 2002). Additionally, there are short-term studies that have found associations between PM concentrations on the day or days preceding mortality of other morbidity endpoints (e.g., HEI, 2003). These studies include both PM_{10} (PM with diameters <10 microns) and $PM_{2.5}$ (<2.5 microns). $PM_{2.5}$ is generally acknowledged to penetrate more deeply in the lung and is of greater concern for health effects. While uncertainties remain, the EPA has been persuaded by this evidence and has established a National Ambient Air Quality Standard (NAAQS) for $PM_{2.5}$ based on an annual average of 15 $\mu g/m^3$ and a peak 24-hour daily exposure of 35 $\mu g/m^3$. A separate PM_{10} 24-hour standard is set at 150 $\mu g/m^3$.

Despite EPA's standard, there remains skepticism that the reported epidemiologic associations may be due to residual confounding or are artifacts of flawed statistical methods (e.g., Moolgavkar, 2005; Reiss et al., 2007). Furthermore, EPA's standard assumes that all forms of particles have equal toxicity, whereas it is generally acknowledged that this assumption is a fallacy (e.g., Cooke et al., 2007; Reiss et al., 2007), though there is not broad agreement on apportioning the toxicity to individual components of PM.

Brown et al. devote considerable discussion to the notion that smaller averaging periods than 24 hours are necessary to evaluate the health effects of wood smoke exposures. The smallest averaging period used by EPA for the PM NAAQS is 24 hours. Despite that, Brown et al. developed a 6-hour averaging period, which they allege is necessary due to the uniquely high short-term peak concentrations of PM caused by wood smoke emissions.

EPA produced a Staff Paper in the development of the PM NAAQS that synthesized all of the relevant scientific information (EPA, 2005). In it, EPA considered but rejected a shorter-term averaging period for PM based on a lack of evidence stating, "information remains too limited to serve as a basis for establishing a shorter-than-24-hour fine particle primary standard at this time." EPA also noted that the bulk of the health effects are attributed to longer-term exposures (longer than 24-hours):

> "EPA's updated risk assessment supports the previous conclusion that peak 24-hour $PM_{2.5}$ concentrations contribute a relatively small amount to the total health risk associated with short-term exposures on an annual basis, such that much if not most of the aggregated annual risk results from the large number of days during which the 24-hour average concentrations are in the low- to mid-range range."

In other words, the highest risks are from longer-term exposures, which contradicts Brown et al.'s hypothesis that shorter-term averaging periods are needed.

Several of the references cited by Brown et al. alleging health effects after short averaging periods (about 2-4 hours) actually make no reference to short averaging periods. Some of the other studies cited by Brown do consider shorter averaging periods, but Brown et al. often misinterprets these studies. For example, on page 193, Brown et al. state, "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 $PM_{2.5}$ exposures (Peters et al. 2001, Gent et al. 2003)" (italics in original). However, the Gent et al. (2003) study evaluated respiratory symptoms (e.g., wheeze, cough, shortness of breath, bronchodilator use), but not hospitalizations. Also, Gent et al. found an effect for ozone but <u>not</u> $PM_{2.5}$. Gent et al. concluded that the "ozone level but not $PM_{2.5}$ was significantly associated with respiratory symptoms and rescue medication use among children using maintenance medication." Peters et al. (2001) evaluated associations with PM and myocardial infarction in a time-series study. The authors found significant associations with 2-hour and 24-hour PM concentrations. However, the effect sizes were similar for both averaging periods. Therefore, this study provides no evidence that peak 2-hour exposures result in an additive risk above the 24-hour average concentrations.

Additional misleading uses of literature are found in the following examples:

- On page 193, Brown et al. state, "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)." However, neither of these two studies includes any exposure index of only a few hours duration. The

smallest averaging period in the Zanobetti study is 24 hours, and the smallest averaging periods for PM in the Dockery study are multi-year averages. These studies do not even mention smaller averaging periods.

- On page 197, Brown et al. state, "Animal and human studies show an association between wood smoke exposure and increased visits to the doctor, emergency rooms, and hospitalizations." Of course, there are no animal studies that associate PM levels with "visits to the doctor, emergency rooms, and hospitalizations."

- Referencing Koenig et al. (1993), Brown et al. state "health effects from PM occur after exposure of 2 to 4 hours or less in duration of wood smoke at the 12 to 20 $\mu g/m^3$ range." However, Koenig et al. clearly state in their conclusions: "In summary, the 12-hr average of fine particulate matter measured by light scattering in a wood-burning community is strongly associated with acute changes in pulmonary function in asthmatic children exposed during a winter heating season…" There is no mention of 2-4 hour exposures. Also, the PM measurements discussed by Koenig were not specific to wood smoke and included contributions from other sources such as vehicles and industrial emissions. Additionally, Koenig et al. relied on a light-scattering instrument that provided data in units of light-scattering coefficients for a 12-hour and weekly average period, rather than units of $\mu g/m^3$ over a 2- to 4-hour period as claimed by Brown et al.

- On page 198, Brown et al. state, "Epidemiology studies report increased cardiovascular events, exacerbation of asthma, and chronic obstructive pulmonary disease as well as links to cancer (Pope et al., 2002)." The Pope et al. study included only mortality and lung cancer endpoints, not exacerbation of asthma and chronic obstructive pulmonary disease (COPD).

- On page 198, Brown et al. state, "Some clinical studies demonstrate a protective effect for anti-inflammatory medications." Presumably, Brown et al. is trying to say that PM health effects are modified by anti-inflammatory medications. While there may be epidemiologic studies that found this, we are not aware of any "clinical studies" that have been conducted along these lines.

- On page 199, Brown et al. state, "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 $\mu g/m^3$) for 2 to 4 h

> can induce cardiopulmonary effects in humans." It is not clear whether Brown et al. is referencing Zanobetti and Schwartz for their statement that effects can occur at 2 to 4 hr averaging time, but the second sentence certainly doesn't logically follow the first sentence, or the discussion prior to the quote above.

On page 197, Brown et al. has a discussion of the basis for PM toxicity. They state:

> "The primary concern is the formation of highly respirable particulate[1], less than $PM_{2.5}$, enhanced by active absorption[2] 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."

The presumption here is that organic gases adsorbed to the particles increase the toxicity of the particles. This would be highly relevant for wood smoke given the high amount of organic gases. However, there is no scientific evidence to support this contention. Based on a mathematical model of gas-particle-mucus heat and mass transport in human airways, Wexler and Sarangapani (1998) concluded that "only the most soluble atmospheric compounds are transported to the pulmonary region via particles and at most only about 1% of the total mass of these compound[s] is in the particle phase because the particle liquid-water content is so small." Also, Rothenberg et al. (1989) studied the surface area, adsorption and desorption of formaldehyde adsorbed onto dust particles. Their results showed that only small quantities of formaldehyde were associated with the PM and the dose from the gas-phase formaldehyde delivered to the respiratory tract was substantially greater than the dose delivered from the PM-associated formaldehyde. Friedlander and Yeh (1998) have speculated that fine particles containing sulfate contribute to enhanced lung deposition of certain hazardous compounds by creating a hygroscopic or absorbing medium for volatile gases. However, wood smoke does not contain much sulfate. Also, the Wexler and Sarangapani modeling contradict the theoretical speculation of Friedlander and Yeh.

Brown et al. also cite several studies specifically associated with wood smoke exposure (although, as noted above, they incorrectly attribute the Koenig et al. study as being purely

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[1] Throughout the paper, Brown uses the word "particulate" as a noun, but it is an adjective. As a noun, the proper word is "particle."

[2] Presumably, Brown means "adsorption" not "absorption" here.

wood smoke exposure, even adding "in a wood burning community" to the title[3]). For example, Brown et al. cite the Naeher et al. (2005) report and Zelikoff et al. (2002) articles, which are literature reviews of health effects due to woodsmoke. These two papers report on animal toxicology studies that mostly find inflammatory-type effects associated with acute woodsmoke exposures at high concentrations (well above ambient levels). These effects are not consistent with the mortality impacts postulated by Brown et al. at lower ambient concentrations. Also, both Naeher et al. and Zelikoff et al. lament the availability of subchronic exposure toxicology data. After the publication of these papers, Reed et al. (2006) published a subchronic exposure study for woodsmoke and concluded "the results reported here show few and only modest health hazards from short-term and subchronic exposures to realistic concentrations of hardwood smoke."

Item 2: Misuse of the "Unhealthy Air Day" Concept

To assess health effects associated with particulate matter (PM), Brown et al. developed a concept the authors call the "Unhealthy Air Day (UAD)." Brown et al. assigns PM concentrations for three risk categories (at risk, moderate risk, and high risk) as shown in Table 1. The categories are defined by different 6-hour average concentrations and Brown et al. lists a progression of health effects alleged to occur at each of the UAD levels.

Table 3. Definition of Brown et al.'s unhealthy air day (UAD) concept

Risk Category, UAD	Mass Exposure (μg)	6-Hour Average $PM_{2.5}$ Concentration ($\mu g/m^3$)	Health Effect
At risk	96	19	Asthma attacks increase
Moderate risk	120	25	Medical intervention, chronic obstructive pulmonary disease (COPD), asthma
High risk	250	53	Hospital or emergency room visits, asthma or cardiovascular events

[3] The actual title of the Koenig et al. article is "Pulmonary function changes in children associated with fine particulate matter." Brown references the title as "Pulmonary function changes in children associated with fine particulate matter air pollution in a wood burning community."

There are several departures in Brown et al.'s UAD concept compared to EPA's National Ambient Air Quality Standard (NAAQS) for PM. The $PM_{2.5}$ NAAQS is based on an annual average of 15 $\mu g/m^3$ and a peak 24-hour daily exposure of 35 $\mu g/m^3$. Furthermore, EPA also has an Air Quality Index (AQI) that represents the air quality conditions for the previous day. For $PM_{2.5}$, it defines "good" air quality as having a daily concentration below 20 $\mu g/m^3$ and "moderate" air quality having a daily concentration below 40 $\mu g/m^3$. Brown et al. defines these levels as "at risk" or "moderate risk" based on a redefinition of the average period. The reasoning used by Brown et al. to modify EPA's AQI is convoluted, poorly documented, and discordant with EPA's assessment of the current state-of-the-science.

To support the values listed in Table 1, Brown et al. references two unpublished documents that he authored for an advocacy group, The Connecticut Fund for the Environment (Brown et al., 2005; Brown et al., 2006). The 2006 document lists the micrograms of exposure over a 24-hour period for the three UAD levels. If one divides these values by four to adjust to a 6-hour period, the mass exposure values in Table 3 are reproduced. The equivalent air concentrations are estimated by assuming a 0.8 m^3/hour breathing rate. Brown et al. (2006) justifies these values with a single statement:

> "Based upon a broad set of epidemiological and medical studies examining asthma, bronchitis, chronic obstructive pulmonary disease (COPD), and cardiovascular disease, we defined health risks in this way."

In a footnote, the 2006 document references the discussion on page 5 of the 2005 document as the scientific support for the statement. The short discussion on page 5 of the 2005 document, a document authored by Brown and two attorneys, references three studies, which hardly qualifies as a "broad set."

There is no attempt at any quantitative justification of the levels in Table 1, just the short one sentence that references to a "broad set" of studies.

The justification for the PM health benchmarks used by Brown et al. can be summarized as coming from an unpublished document produced for an advocacy group with scant documentation provided to justify them. Brown et al. managed to get them published in a peer-reviewed journal by referencing to these unpublished documents. It's highly doubtful that the paper's reviewers downloaded and reviewed the advocacy documents.

Item 3: Incorrect Inferences About Indoor versus Outdoor Concentrations

Brown et al. states that, under episodic conditions, "a person inside the house will inhale a larger dose than estimated from the average of the 24-hour exposure outside." This statement is false—it violates the fundamental law of mass balance and shows a complete misunderstanding of ventilation.

Indoor exposure to pollutants of outdoor origin depends significantly on the behavior of the pollutants indoors. For example, some fraction of the particles might not make it through the building envelope. Particles can also deposit on indoor surfaces and be removed by air filters. Particles can also grow or shrink as they interact with other pollutants in the indoor air, which further changes their fate indoors.

Unless the air-exchange rate changes significantly from the period of infiltration (outdoor concentrations *higher* than indoors) to the period of exfiltration (outdoor concentrations *lower* than indoors), the time-integrated concentration indoors would be the same regardless of the air-exchange rate.

The time-integrated exposure indoors always equals that of the outdoors, regardless of whether the outdoor concentration is episodic or not. It can be shown mathematically that a person inside a house will inhale exactly the same dose as the average 24-hour exposure outdoors.

For example, the 24-hour time-integrated exposure outdoors, would be $C_{out} \times T = 1\ \mu g/m^3$-hr. Let k be the air-exchange rate (hour^{-1}), and the time-integrated indoor concentration would be:

$$\int_0^T C_{out}\left[1-\exp(-kt)\right]dt + \int_0^\infty C_{out}\left[1-\exp(-kT)\right]\times\exp(-kt)dt$$
$$= C_{out}\left[T + \frac{\exp(-kT)}{k} - \frac{1}{k}\right] + C_{out}\left[1-\exp(-kT)\right]\frac{1}{k}$$
$$= C_{out} \times T$$

This shows that the time-integrated exposure indoors equals that of the outdoors. As a result, the statement by Brown et al. that "*a person inside the house will inhale a larger dose than estimated from the average of the 24-h exposure outside"* is false.

In fact, Brown et al. also verified this concept when they calculated the 6-hour indoor dose to be 130 $\mu g/m^3$ (page 200) when the outdoor concentration is 47 $\mu g/m^3$. At the inhalation rate of 0.8 m^3/h used by Brown et al., the 6-hour outdoor dose would be 47 $\mu g/m^3 \times 0.8\ m^3/h \times 6$ hours

= 130 μg/m^3. The fact that the indoor dose and the outdoor dose are the same means that the air exchange rate does not affect time-integrated exposure.

Brown et al. also make inaccurate statements regarding the fundamental laws of mass balance and ventilation, such as: *"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"* (Page 200 footnote #6, unreferenced). However, as wind speed increases, the air-exchange rate also tends to increase, because wind is one of the driving forces for air infiltration. The result of an increasing air exchange rate is that the indoor concentrations would decrease more rapidly once the outdoor concentration is lower than the indoor value. This means that, as higher wind promotes the dilution of PM in ambient air, it also tends to lower the indoor concentrations.

Item 4: Use of "Box Model"

To estimate an indoor air concentration, Brown et al. used an emission level of 100 g/hr and a "simple box exposure model." We are uncertain what model Brown et al. used to estimate outdoor concentrations. It also appears that Brown et al. added a background concentration (17 μg/m^3) to their calculated residence concentration and that the modeled air concentrations, prior to and after adding background were as follows:

Wind speed (mph)	Distance (feet)	Concentration without background (µg/m3)	Concentration with background (µg/m3)
2 (0.89 meters/sec)	500	25	42
2	1000	10	27
5 (2.2 meters/sec)	500	1	18
5	1000	0.5	17

If Brown et al. indeed used a box model to predict outdoor concentrations, they might have used a different mixing height for wind speed at 2 mph and for wind speed at 5 mph. For example, if Brown et al. used the following equation to estimate outdoor concentrations C_{out}:

$$C_{out} = \frac{E}{U \times L \times H}$$

where *E* (g/hr) is the emission rate, *U* (m/s) is the wind speed, *L* (m) is the distance between the source and the receptor, and *H* (m) is the mixing height, we calculated that a mixing height of about 9.2 m was used for the 2 mph case, and 81.6 m was used for the 5 mph case.

An inversion layer at 9.2 m can only occur under very calm conditions on clear nights. Also, the average wind speed of <1 mph is a very low estimate. Even for nighttime conditions, if a more reasonable value of wind speed (1.5 meters/second) and mixing height (15 m) is used, the estimated outdoor concentration would only be one-third of Brown et al.'s original estimate for the 2 mph and 500 feet distance scenario (i.e. 8 $\mu g/m^3$ instead of 25 $\mu g/m^3$).

Additionally, a box model cannot otherwise take into account the effect of atmospheric stability. If Brown et al. instead used a Gaussian model to predict outdoor concentrations, we are uncertain of the stability class that they used in their calculations, nor are we sure that the input parameters they used to run the Gaussian model are valid.

It is also important to point out that Brown et al. calculate $PM_{2.5}$ concentrations of 18 and 17 $\mu g/m^3$ at wind speeds of 5 mph for residences 500 and 1000 ft from the source, respectively. Assuming that the above outdoor concentration estimates included the contribution of background, then OWHHs add only 1 and <1 $\mu g/m^3$ of particles to indoor environments, which is insignificant. This is important because 5-mph winds occur much more often than 2-mph wind in most of the U.S. (NOAA, 1998).

The most scientifically appropriate methodology for this evaluation would have been to use a dispersion model to estimate air concentrations during a variety of meteorological conditions. These results could be coupled with a ventilation model to estimate indoor concentrations. Brown et al.'s use of "box model" is not scientifically defensible. Furthermore, the results of the "box model" cannot be reproduced.

Conclusions and Recommendations

The Brown et al. risk assessment contains numerous flaws in methodology that significantly limit its usefulness to provide credible conclusions regarding short and particularly long-term and health impacts from OWHHs. The cancer risk assessment reaches illogical conclusions and does not follow EPA guidance. With regard to the acute evaluation, Brown et al. misquote and misunderstand the PM health effects literature, and the basis for the conclusion that OWHH emissions cause "acute respiratory and cardiovascular disease" is unfounded.

Exponent conducted a literature search to determine whether additional sources of exposure data related to OWHHs are available that can be used to provide a supplemental evaluation. No additional data were found.

Exponent provides the following recommendations for consideration:

- Conduct air dispersion modeling under a variety of scenarios to understand highest area of impacts for maximum 1-hour, 24-hour and annual average concentrations.
- Conduct a refined state-of the-art risk assessment using the air dispersion modeling results coupled with (1) a time-activity pattern model to account for the actual amount of time individuals could be exposed to particles from OWHHs and (2) an accurate ventilation model to estimate indoor concentrations. Publish findings in a scientific journal. The article should also contain a balanced discussion on the acute health effects of PM, as well as the basis for the EPA 24-hour $PM_{2.5}$ standard.
- Consider conducting an air sampling study to capture 24-hour exposure data under realistic and typical OWHH operations associated with a variety of use scenarios. These data could be used to validate the air dispersion model. Also, PAH speciation data should also be collected to refine the cancer risk assessment.

References

Brown, D.R., Bruhl, R.J., Reyonds, R. and Rothenberger, C.J. 2005. Hot spots and health risks – diesel construction in Connecticut. Connecticut Fund for the Environment. Available at www.cfenv.org.

Brown, D.R., Rothenberger, C.J. and Reynolds, R. 2005. Diesel emissions and unhealthy air in Connecticut. Connecticut Fund for the Environment. Available at www.cfenv.org.

Brown, D.R., Callahan, B.G. and Boissevain, A.L. 2007. An assessment of risk from particulate released from outdoor wood boilers. *Human and Ecological Risk Assessment*, 13: 191-208.

Brown, D.R., Callahan, B.G. and Boissevain, A.L. 2007. Response to Christopher M. Long and Peter Valberg (2007) "Comments on Brown et al. (2007): an assessment of risk from particulate released from outdoor wood boilers" Hum. Ecol. Risk Assess. 13(1). *Human and Ecological Risk Assessment*, 13: 686-690.

Chakrabarti, B, Fine, M.P., Delfino, R.,Sioutas C. 2004. Performance evaluation of the active-flow personal DataRam $PM_{2.5}$ mass monitor designed for continuous personal exposure measurements. *Atmospheric Environment* 38:3329-3340.

Cooke, R.M., Wilson, A.M., Tuomisto, J.T., Morales, O., Tainio, M. and Evans, J.S. 2007. A probabilistic characterization of the relationship between fine particulate matter and mortality: elicitation of European experts. *Environmental Science and Technology*, 41:6598-6605.

Dockery, D.W., Pope, C.A., Xu, X. Spengler, J.D., Ware, J.H., Fay, M.E., Ferris, B.G. and Speizer, F.E. 1993. An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine.* 329:1753-1759.

EPA. 2005. Review of the National Ambient Air Quality Standards for particulate matter, policy assessment of scientific and technical information, OAQPS staff paper.

Fine, PM, Cass GR, and Simoneit BRT. 2004. Chemical characterization of fine particle emissions form the wood stove combustion of prevalent United States tree species. *Environmental Engineering Science* 21(6): 705-721.

Fisher S.L. and Koshland C.P. 2006. Field performance of a nephelometer in rural kitchens: Effects of high humidity excursions and correlations to gravimetric analyses. *Journal of Exposure Science and Environmental Epidemiology*, 17**:** 141-150.

Friedlander, S.K. and Yeh, E.K. 1998. The submicron atmospheric aerosol as a carrier of reactive chemical species: case of peroxides, *Applied Occupational and Environmental Hygiene*. 13:416-420.

Gent, J.F., Triche, E.W., Holford, T.R., Belanger, K., Bracken, M.B., Beckett, W.S., Leaderer, B.P. 2008. Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *Journal of the American Medical Association*. 290:1859-1867.

Health Effects Institute (HEI). 2003. Revised analyses of time-series studies of air pollution and health.

Koenig, J.Q., Larson, T.V., Hanley, Q.S., Rebolledo, V., Dumler, K., Checkoway, H., Wang, S-Z., Lin, D. and Pierson, W.E. 1993. Pulmonary function changes in children associated with fine particulate matter. 63:26-38.

Liu et al. 2002. Comparison of Light Scattering Devices and Impactors for Particulate Measurements in Indoor, Outdoor and Personal Environments. *Environmental Science and Technology*. 36:2977-2986.

Long, C.M. and Valberg, P. 2007. Comment on "An assessment of risk from particulate released from outdoor wood boilers" by Brown et al. (2007). *Human and Ecological Risk Assessment*, 13: 681-685.

Moolgavkar, S.H. 2005. A review and critique of the EPA's rationale for a fine particle standard, *Regulatory Toxicology and Pharmacology.* 42:123-144.

Naeher, L.P., Smith, K.R., Brauer, M., Chowdhury, Z., Simpson, C., Koenig, J.Q., Lipsett, M., Zelikoff, J.T. 2005. Critical review of the health effects of woodsmoke. Prepared for Health Canada.

NOAA. 1998. National Oceanic and Atmospheric Administration. US Department of Commerce. National Climatic Data Center.
http://www5.ncdc.noaa.gov/documentlibrary/pdf/wind1996.pdf

NYSESB 2005. New York State Environmental Protection Bureau. Smoke Gets in Your Lungs: Outdoor Wood Boilers in New York State. New York State Office of the Attorney General, Environmental Protection Bureau, Albany, NY.

NYSDEC. 2007. New York State Department of Environmental Conservation. Dispersion modeling assessment of impacts of outdoor wood boiler emissions in support of NESCAUM's model rule. Impact Assessment and Meteorology Section Division of Air Resources. January 26.

Peters, A., Dockery, D.W., Muller, J.E., and Mittleman, M.A. 2008. Increased particulate air pollution and the triggering of myocardial infarction, *Circulation*, 103:2810-2815.

Pope, C.A., Burnett, R.T., Thun, M., Calle, E.E., Krewski, D., Ito, K. and Thurston, G.D. 2002. Lung cancer, cardiopulmonary mortality and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association*. 287:1132-1141.

Reed, M.D., Campen, M.J., Giglotti, A.P., Harrod, K.S., McDonald, J.D., Seagrave, J.C., Mauderly, J.L., and Seilkop, S.K. 2006. Health effects of subchronic exposure to environmental levels of hardwood smoke. *Inhalation Toxicology*, 18:523-539.

Reiss, R., Anderson, E.L., Cross, C.E., Hidy, G., Hoel, D., McClellan, R. and Moolgavkar, S. 2007. Evidence of health impacts of sulfate- and nitrate-containing particles in ambient air. *Inhalation Toxicology*. 19:419-449.

Rothenberg, S., Nagy, P.A., Pickrell, J.A. and Hobbs, C.H. 1989. Surface area, adsorption and desorption studies in indoor dust samples. *American Industrial Hygiene Association* 50:15-23.

RTP Associates. 2007. Review of NYSDEC modeling study for NESCAUM model rule and NAAQS compliance evaluation for EPA voluntary phase 1 compliant outdoor hydronic heater. Report submitted to US Environmental Protection Agency.

Sioutas, C, et al. 2000. Field evaluation of a modified DataRAM MIE scattering monitor for real time $PM_{2.5}$ mass concentration measurements. *Atmospheric Environment* 34:4829-4838.

Thorpe, A. and Walsh, P.T.. 2002. Performance Testing of Three Portable, Direct-reading Dust Monitors. Annals of Occupational Hygiene 46: 197-207.

Trent A. 2003. Laboratory evaluation of new real-time smoke particulate monitors. Tech. Rep. 0325-2834- MTDC. US Department of Agriculture Forest Service, Missoula Technology and Development Center, Missoula MT.

Trent A. 2006. Smoke particulate monitors: 2006 update. Tech Rep 0625-2842 MTDC. US Department of Agriculture Forest Service, Missoula Technology and Development Center, Missoula MT.

Wu, C-F, et al. 2005. Evaluation and quality control of personal nephelometers in indoor, outdoor and personal environments. *Journal of Exposure Analysis and Environmental Epidemiology* 15, 99-110.

Wexler, A.S. and Sarangapani, R. 1998. Particles do not increase vapor deposition in human airways. *Journal of Aerosol Science* 29:197-204.

U.S. EPA Risk Assessment Guidance for Superfund Volume 1. Human health Evaluation Manual (Part A). Office of Emergency and Remedial Response. 1989.

Zanobetti A.Z., Schwartz, J. and Dockery D.W. 2000. Airborne particles are a risk factor for hospital admissions for heart and lung disease. *Environmental Health Perspectives* 108:1071-7.

Zanobetti, A.Z. and Schwartz, J. 2003. Airborne particles and hospital admissions for heart and lung disease. In HEI (2003).

Zelikoff, J.T., Chen, L.C., Cohen, M.D., Schlesinger, R.B. 2002. The toxicology of inhaled woodsmoke, *Journal of Toxicology and Environmental Health*, 5:269-282.