Letter Health Consultation

RESIDENTIAL WOOD SMOKE COMPLAINT

JACKSON COUNTY, MICHIGAN

Prepared by the
Michigan Department of Community Health

SEPTEMBER 25, 2009

Prepared under a Cooperative Agreement with the
U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Agency for Toxic Substances and Disease Registry
Division of Health Assessment and Consultation
Atlanta, Georgia 30333

Health Consultation: A Note of Explanation

A health consultation is a verbal or written response from ATSDR or ATSDR's Cooperative Agreement Partners to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material. In order to prevent or mitigate exposures, a consultation may lead to specific actions, such as restricting use of or replacing water supplies; intensifying environmental sampling; restricting site access; or removing the contaminated material.

In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes; conducting biological indicators of exposure studies to assess exposure; and providing health education for health care providers and community members. This concludes the health consultation process for this site, unless additional information is obtained by ATSDR or ATSDR's Cooperative Agreement Partner which, in the Agency's opinion, indicates a need to revise or append the conclusions previously issued.

You May Contact ATSDR Toll Free at
1-800-CDC-INFO
or
Visit our Home Page at: http://www.atsdr.cdc.gov

LETTER HEALTH CONSULTATION

RESIDENTIAL WOOD SMOKE COMPLAINT

JACKSON COUNTY, MICHIGAN

Prepared By:

Michigan Department of Community Health
Under Cooperative Agreement with
U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry

STATE OF MICHIGAN
DEPARTMENT OF COMMUNITY HEALTH
LANSING

JENNIFER M. GRANHOLM
GOVERNOR

JANET OLSZEWSKI
DIRECTOR

August 19, 2009

Ted Westmeier, RS, MPH, Director/Health Officer
Jackson County Health Department
1715 Lansing Ave., Suite 221
Jackson, MI 49202

Director Westmeier,

Preface
Early this year and at your request, I provided a memorandum (see Attachment A) regarding the health implications of wood smoke to sensitive individuals. At the request of Agency for Toxic Substance and Disease Registry (ATSDR) for the purpose of meeting required formatting and documentation, I have re-formatted my original memorandum into a letter health consultation document. This letter health consultation confirms the findings of the January 22, 2009 memorandum and states that this issue has been addressed.

Question
What are the health implications of ongoing wood smoke exposure for medically identified sensitive individuals?

Background
This letter health consultation describes health concerns from wood smoke generation at a residential neighborhood in Jackson County, Michigan in relation to individuals who have medically documented conditions that place their health at risk from continued exposure to wood smoke. MDCH-Division of Environmental Health (MDCH-DEH) conducted an investigation into a smoke and odor complaint at this same location in March of 2008 regarding a different wood burning device (MDCH Memo dated April 1, 2008, Re: Outdoor Wood Boiler Jackson County 2008 – $PM_{2.5}$ Data Collection). The findings of this memorandum are based on a conversation with Michael D. Maillard (Department of Environmental Quality (DEQ), Air Quality Division, District Engineer) who conducted a site visit regarding this current wood smoke source, the neighbors medical reports, visual documentation of the stack and smoke, and government or peer-reviewed science literature.

Discussion

DEQ Findings
Michael D. Maillard, DEQ Engineer, visited the residential area under question and observed the smoke stack placement in relation to the surroundings (topography, trees, buildings, wind direction). The smoke stack is on the west side of the owner's house and

approximately 150 feet west of the medically sensitive neighbor. The prevailing wind is out of the west. The smoke stack exit is some five feet below the peak roof line of the second floor of the owner's home. A rain cap prevents smoke emissions from dispersing properly, with the second floor roof line causing down draft of emissions and resultant ground level impact at the neighbor's residence during prevailing winds. The wood source for the heating unit is not protected from rain or snow (see attached photo, Figure 1); resulting in increased emissions when the fuel used is wet. Mr. Maillard provided suggestions that should reduce some smoke generation and improve dispersion of smoke at a higher elevation. He recommended the owner keep the wood source dry and protected from weather, add 6-7 feet of additional stack height, and remove the rain cap and replace it with a rain sleeve. Mr. Maillard states that these suggestions provide no guarantee that problems would not remain due to the location of the trees, topography of the region and the proximity of the medically sensitive neighbors.

Mr. Maillard spoke with the Henrietta Township Building Inspector that issued a permit for the already installed heating unit. The inspector relayed to Mr. Maillard that the heating unit met the local permitting requirements, as to fire safety and that he thought the unit was not new, however, he believes it was certified because it had a "tag". The inspector was not able to give Mr. Maillard any details on model or year of the unit.

Overview of Neighbors' Medical Conditions

The neighbors with documented medical sensitivity to wood smoke have been under medical evaluation for respiratory and/or cardiovascular conditions. The neighbors are non-smokers and report regular exercise. Medical results from the male complainant's pulmonologist and cardiologist state findings of chronic lung disease and coronary artery disease with severe calcification in two major coronary arteries. The spouse's pulmonologist diagnosed a severe cough likely due to chronic bronchitis and sinusitis brought on by smoke exposure, and possible asthmatic bronchitis secondary to smoke inhalation. During a particularly severe smoke event caused by a smoke source from the property in question, one of the smoke-sensitive neighbors was taken to the hospital due to a severe restriction in breathing. The spirometry report stated a finding of "very severe obstruction". The medical spirometry report stated that the patient's lung function, as measured by FVC, FEV1, FEF25-75, and PEF, were 9-66 percent (%) of normal lung function for a person of that age, height, ethnicity, and gender.

On December 19, 2008, [*name redacted*], M.D., F.C.C.P, cardiopulmonary specialist, advised in writing that these individuals "avoid all smoke exposure", based on their health conditions. The statement further elaborated the severity by stressing that "his" lung condition is "absolutely exacerbated by exposure to smoke" and "ongoing exposure could cause permanent and progressive damage to his lungs, which could cause him significant disability". On December 22, 2008, [*name redacted*],, D.O., F.A.C.O.I., F.A.C.C., F.S.C.A.I., wrote that "Continued exposure from this source could of course cause progression of the disease, serious health complications and eventually contribute to his demise. As a cardiologist, I would strongly advise the patient to be removed from this dangerous source and or other source of smoke caused by burning."

Exposure Pathway
Based on the proximity, prevailing wind direction, observations of the DEQ engineer, and video documentation provided by the neighbor, MDCH concludes that a completed exposure pathway exists from the new smoke stack and the medically sensitive neighbor. Mr. Ted Westmeier (RS, MPH, Director, Health Office) from the Jackson County Health Department described the wood burning unit in question as a heat source for the house and that it presumably would operate 24 hours a day during the winter heating season. The neighbor describes the wood smoke emissions as continuous, which is in agreement with Mr. Westmeier's understanding of the wood burning unit. It is not known if the wood burning unit in question has technology that would allow for more complete combustion of the wood smoke and thus reduce emissions by some amount.

Air concentrations may be expected to be elevated well over background levels, depending on the burning practices (US EPA 1993). As part of a previous wood smoke investigation at this location, ambient $PM_{2.5}$ data was collected from the complainant's property while the neighbor operated a wood smoke source (ATSDR 2009). Findings from that investigation demonstrated that $PM_{2.5}$ concentrations were significantly higher when mild smoke odors were sensed but the air on the complainant's property was clear. Ground level smoke was observed on the neighbor's property when the mild odors were sensed. $PM_{2.5}$ levels would be expected to increase within plumes of ground level wood smoke. Johnson (2006) conducted a controlled experiment with a wood burning source and found that placing the $PM_{2.5}$ meter downwind (150 feet) of the wood smoke source resulted in $PM_{2.5}$ measurement that averaged 130 $\mu g/m^3$ and exceed 810 $\mu g/m^3$ over a 50 minute period. Residential wood burning has been shown to impact local air quality (Hellén et al. 2008).

Toxicology Overview of Wood Smoke and Associated Fine Particles
Description of Wood Smoke - Cordwood (i.e., split short chunks of wood) heaters burn wood in an atmosphere of low oxygen and generate incomplete combustion products such as carbon monoxide and numerous organic chemicals. If these vapors are not immediately oxidized, they cool as they vent to the outside and forms fine particulate matter (PM) that is rich in high molecular weight organic chemicals (US EPA 1993). This fine PM has a size range measured in micrometers (µm). PM less than 10 µm (PM_{10}) and PM less than 2.5 µm ($PM_{2.5}$) are common size ranges of PM found in wood smoke (Park and Lee 2003, Hellén et al. 2008, Johnson 2006, NESCAUM 2006, Gullett et al. 2004). The smaller the PM the further into the lungs these particles can reach. Thus $PM_{2.5}$ contains the size range that can go furthest into the lungs with the smallest of these particles (less than 0.1 µm) having been shown to pass through the lungs into a person's blood stream (Nemmers et al. 2002).

The gas and particles of wood smoke contain numerous types of organic chemicals (polycyclic aromatic hydrocarbons (PAHs), phenols, aldehydes, alkenes, alkanes, and aromatics) (US EPA 1993, Gullett et al. 2004). This includes several chemicals that can increase a person's risk of cancer including benzo(a)pyrene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3 cd)pyrene, benzo(k)fluoranthene,

benzo(a)anthracene, benzo(b)fluoranthene, benzene, and formaldehyde (Brown et al. 2007, Hellén et al. 2008, NESCAUM 2008). Additionally, trace elements can be released during the combustion of wood or other combustible material (US EPA 1993). Amount and types of chemicals in any particular wood smoke will depend on the characteristics of the materials being burned, system used to conduct the combustion, and how the system is operated (Hellén et al. 2008, US EPA 1996, NESCAUM 2008). In residential setting, wood burning can be the dominant source of these chemicals to the atmosphere (e.g. benzene in the air was 70% from wood burning) (Hellén et al. 2008).

Mortality - Daily PM concentrations in ambient air have been associated with death (Ostro et al. 2006, Schwartz 2000, Dockery et al. 1993). Daily deaths increased by 0.67% for a 10 $\mu g/m^3$ increase in PM_{10} (Schwartz 2000). This result was based on a study comparing daily deaths in 10 US cities and the daily PM_{10} air concentrations (Mean PM_{10} concentrations ranged from 27-41 $\mu g/m^3$). Dockery et al. (1993) found increased mortality rates due to cardiopulmonary disease and lung cancer in cities with higher concentrations of PM. Ostro et al. (2006) reports that short-term (1 day) increases in $PM_{2.5}$ results in cardiovascular and respiratory mortality, with respiratory mortality having a stronger association to $PM_{2.5}$. Kan et al. (2008) reported an increase of 10 $\mu g/m^3$ in PM_{10} concentrations, calculated as two-day averages, was associated with increases in all-cause mortality of 0.25% (CI: 0.14-0.37).

Long-term exposure to $PM_{2.5}$ (mean and standard deviation = $17.7 \pm 3.7 \mu g/m^3$) significantly increases the relative risk of dying from cardiopulmonary disease (8 percent per 10 $\mu g/m^3$ increase in average $PM_{2.5}$) and lung cancer (12 percent per 10 $\mu g/m^3$ increase in average $PM_{2.5}$) in a study of 319,000 people from 51 US metropolitan areas between 1979-2000 (Pope et al. 2002). Exposure to $PM_{2.5}$ for people that smoke increases their risk in at least an additive manner and may be greater than additive, making smokers at some of the highest increased risk of cardiovascular disease mortality (Pope et al. 2004). For adults 65 years and older, Mar et al (2000) documented increased risk of cardiovascular mortality relative to several indicators of fine PM concentration including $PM_{2.5}$.

Respiratory Effects - Wood smoke has been found to impair the human respiratory system. Wood smoke is shown to be a risk factor for chronic obstructive pulmonary disease (COPD) (Ocozco-Levi et al. 2006). A study of nine children with mild asthma (not using corticosteroids) exposed to wood smoke reported associations between measures of airway inflammation and decreased lung function with measures of increased wood smoke exposure (Allen et al. 2008). Dennis et al. (1996) reported wood smoke to increase the risk by four times (odd ratio 3.9; range of 1.7-9.1) for women contracting obstructed airway disease (OAD). These women were chronically exposed to wood smoke during childhood and the common risk factors for OAD such as cigarette smoking, were not prevalent in these individuals. Sandoval et al. (1993) suggests that the chronic breathing of wood smoke may result in a more severe level of chronic high blood pressure (pulmonary arterial hypertension) than people suffering from smoking-related COPD. They further found that people with pulmonary arterial hypertension and a history

of wood smoke exposure were less able to move oxygen into their blood than people with smoking-related COPD.

Short-term exposures (one hour to one day) to increases in PM, including $PM_{2.5}$, has been found to result in significant changes in measurable indicators of airway inflammation (Adamkiewicz et al. 2003, Koenig et al. 2003, Koenig et al. 2005, Jansen et al. 2005, Delfino et al. 2006) and lung obstruction (Delfino et al. 2004, Trenga et al. 2006, Delfino et al. 2008), which are commonly used to diagnose asthma. Increased obstruction and airway inflammation can cause more sensitive individuals (elderly or preexisting respiratory condition) to be admitted to the hospital for treatment (Dominici et al. 2006). Host et al. (2007) reported increased risk (6.2% per 10 $\mu g/m^3$ increase in $PM_{2.5-10}$) of respiratory disease for children under 14 years and increased risk of respiratory infections (2.5% per 10 $\mu g/m^3$ increase in $PM_{2.5}$, 4.4% per 10 $\mu g/m^3$ increase in $PM_{2.5-10}$) for all ages.

Cardiovascular Effects - Pope et al. (2006) reported that same day ambient air $PM_{2.5}$ concentrations are associated with acute ischemic heart disease (IHD) for people with pre-existing factor for heart disease. IHD is heart disease that is due to blocked or partially blocked arteries. They found a 3.2-4.8 percent increase in ischemic cardiovascular events with every 10 $\mu g/m^3$ increase in $PM_{2.5}$ on the same day as the cardiac event (primarily myocardial infarction or unstable angina). Pope et al. (2006) conclude that people with diseased coronary arteries were the individuals at greatest acute risk from elevated $PM_{2.5}$ exposures, compared to people with relatively healthy arteries. Host et al. (2007) studied the number of cardiovascular, cardiac, and ischemic heart disease admission in hospitals of five major French cities relative to $PM_{2.5}$ and $PM_{2.5-10}$ ambient air concentrations. Similar to Pope et al., Host et al. reported a 4.5, 2.4, and 1.9 percent increase in excess relative risk of IHD, cardiac diseases, and cardiovascular diseases, respectively, with each 10 $\mu g/m^3$ increase in $PM_{2.5}$ among people 65 years and older. Across all ages, 0.9 percent increase in relative risk of cardiovascular diseases with each 10 $\mu g/m^3$ increase in $PM_{2.5.}$ D'Ippoliti et al. (2003) observed similar findings in a study of 6,531 individuals hospitalized for a first episode of acute myocardial infarction. D'Ippoliti et al. used measures of total suspended solids (TSP), nitrogen dioxide, and carbon monoxide as surrogates for the amount of fine PM exposure. They found that daily hospital admissions for myocardial infarction events were most strongly correlated with TSP measures on the day of hospital admission or on the immediately previous day. Although TSP is a crude measure for fine PM, they found an association of a 2.8 percent increase in hospital admissions with a 10 $\mu g/m^3$ increase in TSP. Peters et al. (2001) used $PM_{2.5}$ measures and found significant increased risk of myocardial infarction with increased exposure (OR: 1.48; 95%CI: 1.09-2.02; for an increase of 25 $\mu g/m^3$ during a 2-hour period prior onset). In a study of 22,000 survivors of a myocardial infarction across five European cities, von Klot et al. (2005) found that cardiac re-admissions to the hospital were significantly higher on days with elevated PM_{10} (rate ratio=1.021; 95% confidence interval=1.004-1.039). Zanobetti and Schwartz (2005) found similar significant correlations between increased same day PM_{10} concentrations and increased hospital admissions for myocardial infarction among US citizens 65 years and older covered by Medicare. Sullivan et al. (2005) reported non-significant higher risk for

people with preexisting heart disease that experience increases in PM (odds ratio: 1.05, 95% confidence interval = 0.95-1.16).

Mechanisms attempting to explain how PM exposures cause cardiovascular damage have been proposed. PM exposures can cause both an inflammation response and an increase in oxidative stress at a cellular level either in the lungs or at specific tissues within the body. This response may be due to either chemicals associated with PM or small particles causing activation of cellular immune responses (Brook 2007). The inflammation response causes a release of proteins throughout the body that can alter the normal function of the inner lining of blood vessels (i.e., endothelial cells) (Rajagopalan et al. 2005). Change in blood vessel function may alter the stability or build-up of plaques in arteries increasing the chance of free-moving plaques (thrombosis) and blockage of arteries (O'Neill et al. 2005, Brook 2007, Rajagopalan et al. 2005, Pope et al. 2004).

PM may alter the function of the nervous system that controls the heart (i.e., autonomic nervous system (ANS)). This altered ANS function may be caused by the inflammation response described above or by the particles causing direct irritation to nerves in the lungs that may results in a nerve reflex altering ANS function (Brook 2007, Pope et al. 2004). Abnormal electrocardiograms (ECG) have been observed in relation to increased concentrations of PM. Gold et al. (2000) studied 21 adults between the ages of 53 and 87 years living in Boston, MA and found that a 24-hr average $PM_{2.5}$ concentration (mean=15.5 $\mu g/m^3$; range 2.3-45.1$\mu g/m^3$) was correlated with a significant decrease in heart rate and the 4-hr average $PM_{2.5}$ concentrations (mean=14.7 $\mu g/m^3$; range 0 – 44.9 $\mu g/m^3$) was correlated with a significant decrease in heart rate variability. Reduced heart rate variability is a predictor of increased risk of cardiovascular mortality and morbidity. Magari et al. (2001) in a study of 40 workers (19-59 years old) found significantly reduced heart rate variability in relation to short-term increased exposure to $PM_{2.5}$. Pekkanen et al. (2002) conducted an observational study of a sensitive population of 45 adults (over 60 years old, current nonsmokers, diagnosed with stable coronary heart disease) living in Helsinki, Finland ($PM_{2.5}$ range 8.1-39.8 $\mu g/m^3$) and found decreased ST-segment depression on ECG during periods of exercise two days after participants experience elevated $PM_{2.5}$ exposures (Odds Ratio (OR): 4.56; 95%CI: 1.73-12.03). ST-segment depression during exercise indicates an increased probability of myocardial ischemia (ACC/AHA guidelines as reported by Pekkanen et al. 2002). Chuang et al. (2008) found similar results in 48 Boston, MA residents (43-75 years old) where the combination of $PM_{2.5}$ and black carbon exposure two days before the ECG correlated with a significant risk of ST-segment depression.

Conclusion

The medically documented health conditions of the neighbor, medical recommendations to avoid wood smoke, the new frequent daily smoke source, and in consideration of the robust science literature on the toxicity of wood smoke and associated small particles, MDCH-Division of Environmental Health concludes that breathing particulate matter from wood smoke on a daily basis over a heating season could harm the health of medically sensitive neighbors at [*address redacted*]. The attached memorandum was issued when this wood smoke source was ongoing. The local health department

facilitated changes in the operation of the wood burning unit that was demonstrated to limit the occurrence of the ground level wood smoke. MDCH concludes that as long as the wood burning unit is operated in a manner that does not result in ground level wood smoke or significantly elevated particulate matter, then the breathing of wood smoke in this neighborhood will be minimal and not expected to harm people's health.

Recommendations

With the technical support of MDCH-DEH, local public health facilitated the necessary steps within the public health code to address the wood smoke exposures associated with the identified medically sensitive individuals.

Public Health Action Plan

MDCH-DEH provided technical support upon request to local public health in their efforts to address the described wood smoke exposure.

References

Adamkiewicz G, Ebelt S, Syring M, Slater J, Speizer FE, Schwartz J, Suh H and Gold DR. 2003. Association between air pollution exposure and exhaled nitric oxide in an elderly population. Thorax 59:204-209.

Allen RW, Mar T, Koenig J, Liu LJ, Gould T, Simpson C, Larson T. 2008. Changes in lung function and airway inflammation among asthmatic children residing in a woodsmoke-impacted urban area. Inhal. Toxicol. 20(4):423-433.

Agency for Toxic Substances and Disease Registry (ATSDR). 2009. Health Consultation. Outdoor Wood Boiler Investigation in Jackson County, Michigan. Prepared by Michigan Department of Community Health. Lansing, MI.

Brook RD. 2007. Is air pollution a cause of cardiovascular disease? Updated review of controversies. Reviews in Environmental Health. 22:115-137.

Chuang KJ, Coull BA, Zanobetti A, Suh H, Schwartz J, Stone PH, et al. 2008. Particulate air pollution as a risk factor for ST-segment depression in patients with coronary artery disease. Circulation. September 23. 1-7.

Delfino RJ, Quintana PJ, Floro J, Gastañaga VM, Samimi BS, Kleinman MT, Liu LJ, Bufalino C, Wu CF, McLaren CE. 2004. Association of FEV1 in asthmatic children with personal and microenvironmental exposure to airborne particulate matter. Environ Health Perspect. 112(8):932-41.

Delfino RJ, Staimer N, Gillen D, Tjoa T, Sioutas C, Fung K, George SC, Kleinman MT. 2006. Personal and ambient air pollution is associated with increased exhaled nitric oxide in children with asthma. Environ Health Perspect. 114(11): 1736-43.

Delfino RJ, Staimer N, Tjoa T, Gillen D, Kleinman MT, Sioutas C, Cooper D. 2008. Personal and ambient air pollution exposures and lung function decrements in children with asthma. Environ Health Perspect. 116(4):550-8.

Dennis RJ, Maldonado D, Norman S, Baena E, Martinez G. 1996. Woodsmoke exposure and risk for obstructive airways disease among women. Chest. 109(1):115-9.

D'Ippoliti D, Forastiere F, Ancona C, Agabiti N, Fusco D, Michelozzi P, and Perucci CA. 2003. Air pollution and mycocardial infarction in Rome a case-crossover analysis. Epidemiology. 14: 528-535.

Dockery DW, Pope CA 3rd, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE. 1993. An Association between Air Pollution and Mortality in Six U.S. Cities. N Engl J Med. 329:1753-1759

Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, Samet JM. 2006. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 295(10):1127-34.

Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B. 2000. Ambient pollution and heart rate variability. Circulation. 101:1267-1273.

Hellén H, Hakola H, Haaparanta S, Pietarila H, Kauhaniemi M. 2008. Influence of residential wood combustion on local air quality. Science of the Total Environment. 393:283-290.

Host S, Larrieu S, Pascal L, Blanchard M, Declercq C, Fabre P, Jusot J-F et al. 2007. Short-term associations between fine and course particles and cardiorespiratory hospitalizations in six French cities. Occup.Environ. Med. Online at oem.bmj.com. 1-15.

Jansen KL, Larson TV, Koenig JQ, Mar TF, Fields C, Stewart J, Lippmann M. 2005. Associations between health effects and particulate matter and black carbon in subjects with respiratory disease. Environ Health Perspect. 113: 1741-1746.

Kan H, London SJ, Chen G, Zhang Y, Song G, Zhao N. 2008. Season, sex age, and education as modifiers of the effects of outdoor air pollution on daily mortality in Shanghai, China: the public health and air pollution in Asia (PAPA) study. Environ Health Perspect. 116: 1183-1188.

Koenig JQ, Jansen K, Mar TF, Lumley T, Kaufman J, Trenga CA, Sullivan J, Liu LJS, Shapiro GG, Larson TV. 2003. Measurement of offline exhaled nitric oxide in a study of community exposure to air pollution. Environ Health Perspect. 111: 1625-1629.

Koenig JQ, Mar TF, Allen RW, Jansen K, Lumley T, Sullivan JH, Trenga CA, Larson T, Liu LJ. 2005. Pulmonary effects of indoor- and outdoor-generated particles in children with asthma. Environ Health Perspect. 113: 499-503.

Gullett BK, Touati A, Hays MD. 2004. PCDD/F, PCB, HxCBz, PAH, and PM emission factors for fireplace and woodstove combustion in the San Francisco Bay region. Environ Sci Technol. 2003 May 1;37(9):1758-65.

Johnson PRS. 2006. In-field ambient fine particle monitoring of an outdoor wood boiler: public health concerns. Human and Ecological Risk Assessment. 12: 1153-1170.

Magari SR, Hauser R, Schwartz J, Williams PL, Smith TJ, Christiani DC. 2001. Association of heart rate variability with occupation and environmental exposure to particulate air pollution. Circulation. 104: 986-991.

Mar TF, Norris GA, Koenig JQ, Larson TV. 2000. Associations between air pollution and mortality in Phoenix, 1995-1997. Environ Health Perspect. 108: 347-353.

Nemmar A, Hoet PH, Vanquickenborne B, Dinsdale D, Thomeer M, Hoylaerts MF, Vanbilloen H, Mortelmans L, Nemery B. 2002. Passage of inhaled particles into the blood circulation in humans. Circulation. 105: 411-4.

Northeast States for Coordinated Air Use Management (NESCAUM). 2006. Assessment of outdoor wood-fired boilers. Editor: Paul Miller. 101 Merrimac Street, 10th Floor, Boston, MA 02114. March 2006.

O'Neill MS, Veves A, Zanobetti A, Sarnat JA, Gold DR, Economides PA et al. 2005. Diabetes enhances vulnerability to particulate air pollution- associated impairment in vascular reactivity and endothelial function. Circulation. 111:2913-2920.

Orozco-Levi M, Garcia-Aymerich J, Villar J, Ramírez-Sarmiento A, Antó JM, Gea J. 2006. Wood smoke exposure and risk of chronic obstructive pulmonary disease. Eur Respir J. 27: 542-546.

Ostro B, Broadwin R, Green S, Feng WY, Lipsett M. 2006. Fine particulate air pollution and mortality in nine California counties: results from CALFINE. Environ Health Perspect. 114: 29-33.

Park E and Lee K. 2003. Particulate exposure and size distribution from wood burning stoves in Costa Rica. Indoor Air. 13: 253-259.

Pekkanen J, Peters A, Hoek G, Tiittanen P, Brunekreef B, de Hartog J. 2002. Particulate air pollution and risk of St-Segment depression during repeated submaximal exercise tests among subjects with coronary heart disease: the exposure and risk assessment for fine and ultrafine particles in ambient air (ULTRA) study. Circulation. 106: 933-938.

Peters A, Dockery DW, Muller JE. 2001. Increased Particulate Air Pollution and the Triggering of Myocardial Infarction. Circulation. 103:2810-2815.

Pope III CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. 2002. Lung Cancer, cardiopulmonary mortality, long-term exposure to fine particulate air pollution. JAMA. 287(9): 1132-1141.

Pope III CA, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. 2004. Cardiopulmonary mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation 109: 71-77.

Pope III CA, Muhlestein JB, May HT, Renlund DG, Anderson JL, Horne BD. 2006. Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. Circulation. 114: 2443-2448.

Rajagopalan S, Sun Q, Chen LC. 2005. Particulate pollution and endothelial function: déjà vu all over again in the air. Circulation. 111: 2869-2871.

Sandoval J, Salas J, Martinez-Guerra ML, Gómez A, Martinez C, Portales A, Palomar A, Villegas M, Barrios R. 1993. Pulmonary arterial hypertension and cor pulmonale associated with chronic domestic woodsmoke inhalation. Chest. 103: 12-20.

Schwartz J. 2000. Assessing Confounding, Effect Modification, and Thresholds in the Association between Ambient Particles and Daily Deaths. Environ Health Perspect. 108: 563–568.

Sullivan J, Sheppard L, Schreuder A, Ishikawa N, Siscovick D, Kaufman J. 2005. Relation between short-term fine-particulate matter exposure and onset of myocardial infarction. Epidemiology 16: 41-48.

Trenga CA, Sullivan JH, Schildcrout JS, Shepherd KP, Shapiro GG, Liu LJS, Kaufman JD and Koenig JQ. 2006. Effect of particulate air pollution on lung function in adult and pediatric subjects in a Seattle panel study. Chest 129:1614-1622

U.S. EPA. 1993. A summary of emissions characterization and noncancer respiratory effects of wood smoke. Office of Air Quality Planning and Standards, Research Triangle Park, NC 27711 U.S. EPA- 453/R-93-036. Prepared by Timothy V. Larson and Jane Q. Koenig. University of Washington, Seattle, WA.

von Klot S, Peters A, Aalto P, Bellander T, Berglind N, D'Ippoliti, Elosua R, Hormann A, Kulmala M, Lanki T, Hannelore L, Pekkanen J, Picciotto S, Sunyer J, Forastiere F et al. 2005. Ambient air pollution is associated with increased risk of hospital cardiac readmissions of myocardial infarction survivors in five European cities. Circulation. 112: 3073-3079.

Zanobetti A, Schwartz J. 2005. The effect of particulate air pollution on emergency admissions for myocardial infarction: a multicity case-crossover analysis. Environ Health Perspect. 113:978–982.

Certification

This Health Consultation was prepared by the Michigan Department of Community Health under a cooperative agreement with the Agency for Toxic Substances and Disease Registry (ATSDR). It is in accordance with approved methodology and procedures. Editorial review was completed by the cooperative agreement partner.

Technical Project Officer, Cooperative Agreement Program Evaluation Branch (CAPEB), Division of Health Assessment and Consultation (DHAC), ATSDR

for Alan Yarbrough

The Division of Health Assessment and Consultation, ATSDR, has reviewed this public health consultation and concurs with the findings.

Team Leader, CAPEB, DHAC, ATSDR

Figure 1. Recent (1/19/2009) photo of wood pile used to fire the wood burning device in question.

Attachment A. Original Memorandum Issued January 22, 2009

MEMORANDUM

DEPARTMENT OF COMMUNITY HEALTH

LANSING, MICHIGAN 48913

To: David Wade, PhD, Director, Division of Environmental Public Health
Linda Dykema, PhD, Manager, Toxicology and Response Section

From: Kory Groetsch, Toxicologist, Toxicology and Response Section

Date: January 22, 2009

Re: Health Concerns Regarding Wood Smoke and Sensitive Individuals.

Background

This memorandum describes health concerns from wood smoke generation at a residential neighborhood in Jackson County, Michigan in relation to individuals who have medically documented conditions that place their health at risk from continued exposure to wood smoke. MDCH-Division of Environmental Health conducted an investigation into a smoke and odor complaint at this same location in March of 2008 regarding a different wood burning device (MDCH Memo dated April 1, 2008, Re: Outdoor Wood Boiler Jackson County 2008 – $PM_{2.5}$ Data Collection). The findings of this memorandum are based on a conversation with Michael D. Maillard (Department of Environmental Quality (DEQ), Air Quality Division, District Engineer) who conducted a site visit regarding this current wood smoke source, the neighbors medical reports, visual documentation of the stack and smoke, and government or peer-reviewed science literature.

DEQ Findings

Michael D. Maillard, DEQ Engineer, visited the residential area under question and observed the smoke stack placement in relation to the surroundings (topography, trees, buildings, wind direction). The smoke stack is on the west side of the owner's house and approximately 150 feet west of the medically sensitive neighbor. The prevailing wind is out of the west. The smoke stack exit is some five feet below the peak roof line of the second floor of the owner's home. A rain cap prevents smoke emissions from dispersing properly, with the second floor roof line causing down draft of emissions and resultant ground level impact at the neighbor's residence during prevailing winds. The wood source for the heating unit is not protected from rain or snow (see attached photo, Figure 1); resulting in increased emissions when the fuel used is wet. Mr. Maillard provided suggestions that should reduce some smoke generation and improve dispersion of smoke at a higher elevation. He recommended the owner keep the wood source dry and protected from weather, add 6-7 feet of additional stack height, and remove the rain cap and replace it with a rain sleeve. Mr. Maillard states that these suggestions provide no

guarantee that problems would not remain due to the location of the trees, topography of the region and the proximity of the medically sensitive neighbors.

Mr. Maillard spoke with the Henrietta Township Building Inspector that issued a permit for the already installed heating unit. The inspector relayed to Mr. Maillard that the heating unit met the local permitting requirements, as to fire safety and that he thought the unit was not new, however, he believes it was certified because it had a “tag”. The inspector was not able to give Mr. Maillard any details on model or year of the unit.

Overview of Neighbors’ Medical Conditions

The neighbors with documented medical sensitivity to wood smoke have been under medical evaluation for respiratory and/or cardiovascular conditions. The neighbors are non-smokers and report regular exercise. Medical results from the male complainant's pulmonologist and cardiologist state findings of chronic lung disease and coronary artery disease with severe calcification in two major coronary arteries. The spouse's pulmonologist diagnosed a severe cough likely due to chronic bronchitis and sinusitis brought on by smoke exposure, and possible asthmatic bronchitis secondary to smoke inhalation. During a particularly severe smoke event caused by a smoke source from the property in question, one of the smoke-sensitive neighbors was taken to the hospital due to a severe restriction in breathing. The spirometry report stated a finding of "very severe obstruction". The medical spirometry report stated that the patient’s lung function, as measured by FVC, FEV1, FEF25-75, and PEF, were 9-66 percent (%) of normal lung function for a person of that age, height, ethnicity, and gender.

On December 19, 2008, Robert D. Ablertson, M.D., F.C.C.P, cardiopulmonary specialist, advised in writing that these individuals "avoid all smoke exposure", based on their health conditions. The statement further elaborated the severity by stressing that "his" lung condition is “absolutely exacerbated by exposure to smoke” and “ongoing exposure could cause permanent and progressive damage to his lungs, which could cause him significant disability”. On December 22, 2008, Mark A Rasak, D.O., F.A.C.O.I., F.A.C.C., F.S.C.A.I., wrote that "Continued exposure from this source could of course cause progression of the disease, serious health complications and eventually contribute to his demise. As a cardiologist, I would strongly advise the patient to be removed from this dangerous source and or other source of smoke caused by burning."

Exposure Pathway

Based on the proximity, prevailing wind direction, observations of the DEQ engineer, and video documentation provided by the neighbor, MDCH concludes that a completed exposure pathway exists from the new smoke stack and the medically sensitive neighbor. Mr. Ted Westmeier (RS, MPH, Director, Health Office) from the Jackson County Health Department described the wood burning unit in question as a heat source for the house and that it presumably would operate 24 hours a day during the winter heating season. The neighbor describes the wood smoke emissions as continuous, which is in agreement with Mr. Westmeier’s understanding of the wood burning unit. It is not known if the

wood burning unit in question has technology that would allow for more complete combustion of the wood smoke and thus reduce emissions by some amount.

Toxicology Overview of Wood Smoke and Associated Fine Particles

Description of Wood Smoke

Cordwood (i.e., split short chunks of wood) heaters burn wood in an atmosphere of low oxygen and generate incomplete combustion products such as carbon monoxide and numerous organic chemicals. If these vapors are not immediately oxidized, they cool as they vent to the outside and forms fine particulate matter (PM) that is rich in high molecular weight organic chemicals (US EPA 1993). This fine PM has a size range measured in micrometers (µm). PM less than 10 µm (PM_{10}) and PM less than 2.5 µm ($PM_{2.5}$) are common size ranges of PM found in wood smoke (Park and Lee 2003, Hellén et al. 2008, Johnson 2006, NESCAUM 2006, Gullett et al. 2004). The smaller the PM the further into the lungs these particles can reach. Thus $PM_{2.5}$ contains the size range that can go furthest into the lungs with the smallest of these particles (less than 0.1 µm) having been shown to pass through the lungs into a person's blood stream (Nemmers et al. 2002).

The gas and particles of wood smoke contain numerous types of organic chemicals (polycyclic aromatic hydrocarbons (PAHs), phenols, aldehydes, alkenes, alkanes, and aromatics) (US EPA 1993, Gullett et al. 2004). This includes several chemicals that can increase a person's risk of cancer including benzo(a)pyrene, chrysene, dibenzo(a,h)anthracene, indeno(1,2,3 cd)pyrene, benzo(k)fluoranthene, benzo(a)anthracene, benzo(b)fluoranthene, benzene, and formaldehyde (Brown et al. 2007, Hellén et al. 2008, NESCAUM 2008). Additionally, trace elements can be released during the combustion of wood or other combustible material (US EPA 1993). Amount and types of chemicals in any particular wood smoke will depend on the characteristics of the materials being burned, system used to conduct the combustion, and how the system is operated (Hellén et al. 2008, US EPA 1996, NESCAUM 2008). In residential setting, wood burning can be the dominant source of these chemicals to the atmosphere (e.g. benzene in the air was 70% from wood burning) (Hellén et al. 2008).

Mortality

Daily PM concentrations in ambient air have been associated with death (Ostro et al. 2006, Schwartz 2000, Dockery et al. 1993). Daily deaths increased by 0.67% for a 10 $\mu g/m^3$ increase in PM_{10} (Schwartz 2000). This result was based on a study comparing daily deaths in 10 US cities and the daily PM_{10} air concentrations (Mean PM_{10} concentrations ranged from 27-41 $\mu g/m^3$). Dockery et al. (1993) found increased mortality rates due to cardiopulmonary disease and lung cancer in cities with higher concentrations of PM. Ostro et al. (2006) reports that short-term (1 day) increases in $PM_{2.5}$ results in cardiovascular and respiratory mortality, with respiratory mortality having a stronger association to $PM_{2.5}$. Kan et al. (2008) reported an increase of 10 $\mu g/m^3$ in PM_{10} concentrations, calculated as two-day averages, was associated with increases in all-cause mortality of 0.25% (CI: 0.14-0.37).

Long-term exposure to $PM_{2.5}$ (mean and standard deviation = 17.7 ± 3.7μg/m^3) significantly increases the relative risk of dying from cardiopulmonary disease (8 percent per 10 μg/m^3 increase in average $PM_{2.5}$) and lung cancer (12 percent per 10 μg/m^3 increase in average $PM_{2.5}$) in a study of 319,000 people from 51 US metropolitan areas between 1979-2000 (Pope et al. 2002). Exposure to $PM_{2.5}$ for people that smoke increases their risk in at least an additive manner and may be greater than additive, making smokers at some of the highest increased risk of cardiovascular disease mortality (Pope et al. 2004). For adults 65 years and older, Mar et al (2000) documented increased risk of cardiovascular mortality relative to several indicators of fine PM concentration including $PM_{2.5}$.

Respiratory Effects

Wood smoke has been found to impair the human respiratory system. Wood smoke is shown to be a risk factor for chronic obstructive pulmonary disease (COPD) (Ocozco-Levi et al. 2006). A study of nine children with mild asthma (not using corticosteroids) exposed to wood smoke reported associations between measures of airway inflammation and decreased lung function with measures of increased wood smoke exposure (Allen et al. 2008). Dennis et al. (1996) reported wood smoke to increase the risk by four times (odd ratio 3.9; range of 1.7-9.1) for women contracting obstructed airway disease (OAD). These women were chronically exposed to wood smoke during childhood and the common risk factors for OAD such as cigarette smoking, were not prevalent in these individuals. Sandoval et al. (1993) suggests that the chronic breathing of wood smoke may result in a more severe level of chronic high blood pressure (pulmonary arterial hypertension) than people suffering from smoking-related COPD. They further found that people with pulmonary arterial hypertension and a history of wood smoke exposure were less able to move oxygen into their blood than people with smoking-related COPD.

Short-term exposures (one hour to one day) to increases in PM, including $PM_{2.5}$, has been found to result in significant changes in measurable indicators of airway inflammation (Adamkiewicz et al. 2003, Koenig et al. 2003, Koenig et al. 2005, Jansen et al. 2005, Delfino et al. 2006) and lung obstruction (Delfino et al. 2004, Trenga et al. 2006, Delfino et al. 2008), which are commonly used to diagnose asthma. Increased obstruction and airway inflammation can cause more sensitive individuals (elderly or preexisting respiratory condition) to be admitted to the hospital for treatment (Dominici et al. 2006). Host et al. (2007) reported increased risk (6.2% per 10 μg/m^3 increase in $PM_{2.5-10}$) of respiratory disease for children under 14 years and increased risk of respiratory infections (2.5% per 10 μg/m^3 increase in $PM_{2.5}$, 4.4% per 10 μg/m^3 increase in $PM_{2.5-10}$) for all ages.

Cardiovascular Effects

Pope et al. (2006) reported that same day ambient air $PM_{2.5}$ concentrations are associated with acute ischemic heart disease (IHD) for people with pre-existing factor for heart disease. IHD is heart disease that is due to blocked or partially blocked arteries. They

found a 3.2-4.8 percent increase in ischemic cardiovascular events with every 10 $\mu g/m^3$ increase in $PM_{2.5}$ on the same day as the cardiac event (primarily myocardial infarction or unstable angina). Pope et al. (2006) conclude that people with diseased coronary arteries were the individuals at greatest acute risk from elevated $PM_{2.5}$ exposures, compared to people with relatively healthy arteries. Host et al. (2007) studied the number of cardiovascular, cardiac, and ischemic heart disease admission in hospitals of five major French cities relative to $PM_{2.5}$ and $PM_{2.5-10}$ ambient air concentrations. Similar to Pope et al., Host et al. reported a 4.5, 2.4, and 1.9 percent increase in excess relative risk of IHD, cardiac diseases, and cardiovascular diseases, respectively, with each 10 $\mu g/m^3$ increase in $PM_{2.5}$ among people 65 years and older. Across all ages, 0.9 percent increase in relative risk of cardiovascular diseases with each 10 $\mu g/m^3$ increase in $PM_{2.5.}$ D'Ippoliti et al. (2003) observed similar findings in a study of 6,531 individuals hospitalized for a first episode of acute myocardial infarction. D'Ippoliti et al. used measures of total suspended solids (TSP), nitrogen dioxide, and carbon monoxide as surrogates for the amount of fine PM exposure. They found that daily hospital admissions for myocardial infarction events were most strongly correlated with TSP measures on the day of hospital admission or on the immediately previous day. Although TSP is a crude measure for fine PM, they found an association of a 2.8 percent increase in hospital admissions with a 10 $\mu g/m^3$ increase in TSP. Peters et al. (2001) used $PM_{2.5}$ measures and found significant increased risk of myocardial infarction with increased exposure (OR: 1.48; 95%CI: 1.09-2.02; for an increase of 25 $\mu g/m^3$ during a 2-hour period prior onset). In a study of 22,000 survivors of a myocardial infarction across five European cities, von Klot et al. (2005) found that cardiac re-admissions to the hospital were significantly higher on days with elevated PM_{10} (rate ratio=1.021; 95% confidence interval=1.004-1.039). Zanobetti and Schwartz (2005) found similar significant correlations between increased same day PM_{10} concentrations and increased hospital admissions for myocardial infarction among US citizens 65 years and older covered by Medicare. Sullivan et al. (2005) reported non-significant higher risk for people with preexisting heart disease that experience increases in PM (odds ratio: 1.05, 95% confidence interval = 0.95-1.16).

Mechanisms attempting to explain how PM exposures cause cardiovascular damage have been proposed. PM exposures can cause both an inflammation response and an increase in oxidative stress at a cellular level either in the lungs or at specific tissues within the body. This response may be due to either chemicals associated with PM or small particles causing activation of cellular immune responses (Brook 2007). The inflammation response causes a release of proteins throughout the body that can alter the normal function of the inner lining of blood vessels (i.e., endothelial cells) (Rajagopalan et al. 2005). Change in blood vessel function may alter the stability or build-up of plaques in arteries increasing the chance of free-moving plaques (thrombosis) and blockage of arteries (O'Neill et al. 2005, Brook 2007, Rajagopalan et al. 2005, Pope et al. 2004).

PM may alter the function of the nervous system that controls the heart (i.e., autonomic nervous system (ANS)). This altered ANS function may be caused by the inflammation response described above or by the particles causing direct irritation to nerves in the lungs that may results in a nerve reflex altering ANS function (Brook 2007, Pope et al. 2004).

Abnormal electrocardiograms (ECG) have been observed in relation to increased concentrations of PM. Gold et al. (2000) studied 21 adults between the ages of 53 and 87 years living in Boston, MA and found that a 24-hr average $PM_{2.5}$ concentration (mean=15.5 $\mu g/m^3$; range 2.3-45.1$\mu g/m^3$) was correlated with a significant decrease in heart rate and the 4-hr average $PM_{2.5}$ concentrations (mean=14.7 $\mu g/m^3$; range 0 – 44.9 $\mu g/m^3$) was correlated with a significant decrease in heart rate variability. Reduced heart rate variability is a predictor of increased risk of cardiovascular mortality and morbidity. Magari et al. (2001) in a study of 40 workers (19-59 years old) found significantly reduced heart rate variability in relation to short-term increased exposure to $PM_{2.5}$. Pekkanen et al. (2002) conducted an observational study of a sensitive population of 45 adults (over 60 years old, current nonsmokers, diagnosed with stable coronary heart disease) living in Helsinki, Finland ($PM_{2.5}$ range 8.1-39.8 $\mu g/m^3$) and found decreased ST-segment depression on ECG during periods of exercise two days after participants experience elevated $PM_{2.5}$ exposures (Odds Ratio (OR): 4.56; 95%CI: 1.73-12.03). ST-segment depression during exercise indicates an increased probability of myocardial ischemia (ACC/AHA guidelines as reported by Pekkanen et al. 2002). Chuang et al. (2008) found similar results in 48 Boston, MA residents (43-75 years old) where the combination of $PM_{2.5}$ and black carbon exposure two days before the ECG correlated with a significant risk of ST-segment depression.

Conclusion

Given the medically documented health conditions of the neighbor, medical recommendations to avoid wood smoke, the new frequent daily smoke source, and in consideration of the robust science literature on the toxicity of wood smoke and associated small particles, MDCH-Division of Environmental Health concludes that a significant health risks exist for the medically sensitive neighbors at 10328 Hankerd Road, Pleasant Lake, Michigan.

References

Adamkiewicz G, Ebelt S, Syring M, Slater J, Speizer FE, Schwartz J, Suh H and Gold DR. 2003. Association between air pollution exposure and exhaled nitric oxide in an elderly population. Thorax 59:204-209.

Allen RW, Mar T, Koenig J, Liu LJ, Gould T, Simpson C, Larson T. 2008. Changes in lung function and airway inflammation among asthmatic children residing in a woodsmoke-impacted urban area. Inhal. Toxicol. 20(4):423-433.

Brook RD. 2007. Is air pollution a cause of cardiovascular disease? Updated review of controversies. Reviews in Environmental Health. 22:115-137.

Chuang KJ, Coull BA, Zanobetti A, Suh H, Schwartz J, Stone PH, et al. 2008. Particulate air pollution as a risk factor for ST-segment depression in patients with coronary artery disease. Circulation. September 23. 1-7.

Delfino RJ, Quintana PJ, Floro J, Gastañaga VM, Samimi BS, Kleinman MT, Liu LJ, Bufalino C, Wu CF, McLaren CE. 2004. Association of FEV1 in asthmatic children with personal and microenvironmental exposure to airborne particulate matter. Environ Health Perspect. 112(8):932-41.

Delfino RJ, Staimer N, Gillen D, Tjoa T, Sioutas C, Fung K, George SC, Kleinman MT. 2006. Personal and ambient air pollution is associated with increased exhaled nitric oxide in children with asthma. Environ Health Perspect. 114(11): 1736-43.

Delfino RJ, Staimer N, Tjoa T, Gillen D, Kleinman MT, Sioutas C, Cooper D. 2008. Personal and ambient air pollution exposures and lung function decrements in children with asthma. Environ Health Perspect. 116(4):550-8.

Dennis RJ, Maldonado D, Norman S, Baena E, Martinez G. 1996. Woodsmoke exposure and risk for obstructive airways disease among women. Chest. 109(1):115-9.

D'Ippoliti D, Forastiere F, Ancona C, Agabiti N, Fusco D, Michelozzi P, and Perucci CA. 2003. Air pollution and mycocardial infarction in Rome a case-crossover analysis. Epidemiology. 14: 528-535.

Dockery DW, Pope CA 3rd, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE. 1993. An Association between Air Pollution and Mortality in Six U.S. Cities. N Engl J Med. 329:1753-1759

Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, Samet JM. 2006. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 295(10):1127-34.

Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B. 2000. Ambient pollution and heart rate variability. Circulation. 101:1267-1273.

Hellén H, Hakola H, Haaparanta S, Pietarila H, Kauhaniemi M. 2008. Influence of residential wood combustion on local air quality. Science of the Total Environment. 393:283-290.

Host S, Larrieu S, Pascal L, Blanchard M, Declercq C, Fabre P, Jusot J-F et al. 2007. Short-term associations between fine and course particles and cardiorespiratory hospitalizations in six French cities. Occup.Environ. Med. Online at oem.bmj.com. 1-15.

Jansen KL, Larson TV, Koenig JQ, Mar TF, Fields C, Stewart J, Lippmann M. 2005. Associations between health effects and particulate matter and black carbon in subjects with respiratory disease. Environ Health Perspect. 113: 1741-1746.

Kan H, London SJ, Chen G, Zhang Y, Song G, Zhao N. 2008. Season, sex age, and education as modifiers of the effects of outdoor air pollution on daily mortality in Shanghai, China: the public health and air pollution in Asia (PAPA) study. Environ Health Perspect. 116: 1183-1188.

Koenig JQ, Jansen K, Mar TF, Lumley T, Kaufman J, Trenga CA, Sullivan J, Liu LJS, Shapiro GG, Larson TV. 2003. Measurement of offline exhaled nitric oxide in a study of community exposure to air pollution. Environ Health Perspect. 111: 1625-1629.

Koenig JQ, Mar TF, Allen RW, Jansen K, Lumley T, Sullivan JH, Trenga CA, Larson T, Liu LJ. 2005. Pulmonary effects of indoor- and outdoor-generated particles in children with asthma. Environ Health Perspect. 113: 499-503.

Gullett BK, Touati A, Hays MD. 2004. PCDD/F, PCB, HxCBz, PAH, and PM emission factors for fireplace and woodstove combustion in the San Francisco Bay region. Environ Sci Technol. 2003 May 1;37(9):1758-65.

Johnson PRS. 2006. In-field ambient fine particle monitoring of an outdoor wood boiler: public health concerns. Human and Ecological Risk Assessment. 12: 1153-1170.

Magari SR, Hauser R, Schwartz J, Williams PL, Smith TJ, Christiani DC. 2001. Association of heart rate variability with occupation and environmental exposure to particulate air pollution. Circulation. 104: 986-991.

Mar TF, Norris GA, Koenig JQ, Larson TV. 2000. Associations between air pollution and mortality in Phoenix, 1995-1997. Environ Health Perspect. 108: 347-353.

Nemmar A, Hoet PH, Vanquickenborne B, Dinsdale D, Thomeer M, Hoylaerts MF, Vanbilloen H, Mortelmans L, Nemery B. 2002. Passage of inhaled particles into the blood circulation in humans. Circulation. 105: 411-4.

Northeast States for Coordinated Air Use Management (NESCAUM). 2006. Assessment of outdoor wood-fired boilers. Editor: Paul Miller. 101 Merrimac Street, 10th Floor, Boston, MA 02114. March 2006.

O'Neill MS, Veves A, Zanobetti A, Sarnat JA, Gold DR, Economides PA et al. 2005. Diabetes enhances vulnerability to particulate air pollution- associated impairment in vascular reactivity and endothelial function. Circulation. 111:2913-2920.

Orozco-Levi M, Garcia-Aymerich J, Villar J, Ramírez-Sarmiento A, Antó JM, Gea J. 2006. Wood smoke exposure and risk of chronic obstructive pulmonary disease. Eur Respir J. 27: 542-546.

Ostro B, Broadwin R, Green S, Feng WY, Lipsett M. 2006. Fine particulate air pollution and mortality in nine California counties: results from CALFINE. Environ Health Perspect. 114: 29-33.

Park E and Lee K. 2003. Particulate exposure and size distribution from wood burning stoves in Costa Rica. Indoor Air. 13: 253-259.

Pekkanen J, Peters A, Hoek G, Tiittanen P, Brunekreef B, de Hartog J. 2002. Particulate air pollution and risk of St-Segment depression during repeated submaximal exercise tests among subjects with coronary heart disease: the exposure and risk assessment for fine and ultrafine particles in ambient air (ULTRA) study. Circulation. 106: 933-938.

Peters A, Dockery DW, Muller JE. 2001. Increased Particulate Air Pollution and the Triggering of Myocardial Infarction. Circulation. 103:2810-2815.

Pope III CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. 2002. Lung Cancer, cardiopulmonary mortality, long-term exposure to fine particulate air pollution. JAMA. 287(9): 1132-1141.

Pope III CA, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. 2004. Cardiopulmonary mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. Circulation 109: 71-77.

Pope III CA, Muhlestein JB, May HT, Renlund DG, Anderson JL, Horne BD. 2006. Ischemic heart disease events triggered by short-term exposure to fine particulate air pollution. Circulation. 114: 2443-2448.

Rajagopalan S, Sun Q, Chen LC. 2005. Particulate pollution and endothelial function: déjà vu all over again in the air. Circulation. 111: 2869-2871.

Sandoval J, Salas J, Martinez-Guerra ML, Gómez A, Martinez C, Portales A, Palomar A, Villegas M, Barrios R. 1993. Pulmonary arterial hypertension and cor pulmonale associated with chronic domestic woodsmoke inhalation. Chest. 103: 12-20.

Schwartz J. 2000. Assessing Confounding, Effect Modification, and Thresholds in the Association between Ambient Particles and Daily Deaths. Environ Health Perspect. 108: 563–568.

Sullivan J, Sheppard L, Schreuder A, Ishikawa N, Siscovick D, Kaufman J. 2005. Relation between short-term fine-particulate matter exposure and onset of myocardial infarction. Epidemiology 16: 41-48.

Trenga CA, Sullivan JH, Schildcrout JS, Shepherd KP, Shapiro GG, Liu LJS, Kaufman JD and Koenig JQ. 2006. Effect of particulate air pollution on lung function in adult and pediatric subjects in a Seattle panel study. Chest 129:1614-1622

U.S. EPA. 1993. A summary of emissions characterization and noncancer respiratory effects of wood smoke. Office of Air Quality Planning and Standards, Research Triangle Park, NC 27711 U.S. EPA- 453/R-93-036. Prepared by Timothy V. Larson and Jane Q. Koenig. University of Washington, Seattle, WA.

von Klot S, Peters A, Aalto P, Bellander T, Berglind N, D'Ippoliti, Elosua R, Hormann A, Kulmala M, Lanki T, Hannelore L, Pekkanen J, Picciotto S, Sunyer J, Forastiere F et al. 2005. Ambient air pollution is associated with increased risk of hospital cardiac readmissions of myocardial infarction survivors in five European cities. Circulation. 112: 3073-3079.

Zanobetti A, Schwartz J. 2005. The effect of particulate air pollution on emergency admissions for myocardial infarction: a multicity case-crossover analysis. Environ Health Perspect. 113:978–982.

Figure 1. Recent (1/19/2009) photo of wood pile used to fire the wood burning device in question.