Gina McCarthy, Administrator
US Environmental Protection Agency
EPA Docket Center
Mail Code 2822T
1200 Pennsylvania Ave NW
Washington DC 20460

Attention: Docket ID No. EPA-HQ-OAR-2009-0734

Re: Standards of Performance for New Residential Wood Heaters, New Residential Hydronic Heaters and Forced-Air Furnaces, and New Residential Masonry Heaters (comment deadline May 5, 2014)

Dear Administrator McCarthy:

The Utah Physicians for a Healthy Environment (UPHE) thank you for the opportunity to comment on the proposed rule, entitled Standards of Performance for New Residential Wood Heaters, New Residential Hydronic Heaters and Forced-Air Furnaces, and New Residential Masonry Heaters [79 FR 6329].

UPHE is the largest civic organization of health care professionals in Utah, and perhaps the Western United States, with over 300 members whose expertise includes virtually every medical specialty and related disciplines like biology, genetics, atmospheric modeling and chemistry, toxicology, ecology, and engineering.

The health affects of air pollution represent the core of our expertise and therefore our engagement in public policy. Air pollution in general is increasingly recognized as a systemic health threat, impairing the functioning of virtually every organ system, and related to the same broad spectrum of disease outcomes as cigarette smoke. Because of this, UPHE considers the proposed EPA rule regulating wood burning devices to be grossly inadequate.

Burning trash in backyard incinerators has long been prohibited in most urban areas and the justification is obvious. Operating a vehicle with excessive emissions has long been prohibited through emissions inspections. We have accepted the rationale that even though it may be cheaper for the owner of an older, more polluting vehicle to continue to operate that vehicle, for the public good, those vehicles have to be cleaned up or retired. Why should home heating devices be any different?

Many years ago we adopted a societal norm that no one should be involuntarily subjected to second hand cigarette smoke because of the inherent public health consequences and the infringement on the rights of nonsmokers to avoid exposure.

Routine wood burning should not be allowed for exactly the same philosophical, aesthetic and public health reasons as prohibition of cigarette smoking in public venues, backyard trash incineration, and excessive vehicle emissions. The smoke from wood stoves, boilers and fireplaces creeps onto adjacent property and into nearby homes affecting the quality of life and health of neighbors. Cheap heat or pleasant ambiance for a resident burning wood is accomplished at the expense of nearby neighbors and the community at large, just like second hand cigarette smoke.

Wood smoke is uniquely toxic among all types of community air pollution as detailed by the information below.

1. Wood smoke is a surprisingly large contributor to individual and community pollution exposure. It creates hot spots of pollution and "local victims."

"The largest single source of outdoor fine particles (PM2.5) entering into our homes in many American cities is our neighbor's fireplace or wood stove. ....only a few hours of wood burning in a single home at night can raise fine particle concentrations in dozens of surrounding homes throughout the neighborhood and cause concentrations of PAHs (polycyclic aromatic hydrocarbons)—one of the most toxic compounds of air pollution—higher than 2,000 ng/m3." (Dr. Wayne Ott, Stanford University, Feb. 1, 1998). Background concentrations of PAHs should be close to zero.

Unlike most other sources of pollution, home wood burning emissions are released directly into the area where people spend most of their time at an elevation that does not promote dispersion. Studies from California (1) show that **within a single square kilometer of a residential area, concentrations of wood smoke can vary as much as 2,500 times. Highest measured concentrations were up to 100 times higher than the community average**. Indoor concentrations were found to average 75% as high as outdoor concentrations. A single wood-burning household can envelope adjacent and downwind homes with a primary PM 0.1 (the most dangerous subset of PM2.5) plume. This demonstrates how significant the creation of "local victims" is in assessing the true extent of the health impacts of wood burning. We don't prohibit smoking on air planes or in public buildings because of what that does to community PM2.5 levels. We do so because of the public health consequences to those in the immediate area. The same consideration and protection should apply for neighbors in the issue of wood burning to prevent "local victims."

But wood burning also has an enormous impact on community wide pollution levels. Source apportionment studies have estimated that wood/biomass combustion contribute 10-40% of the fine particle concentrations (PM2.5) in large cities such as Seattle, Phoenix, Beijing, Prague and Helsinki (2,3,4,5). In Pierce County, Washington, 53% of PM2.5 comes from wood. A study in Los Angeles showed that in the winter, residential wood combustion contributed 30% of primary organic aerosols, (probably the most important mass component of particulate pollution) more than motor vehicle exhaust which contributed 21% (6,7). In Fresno, Calif. wood smoke contributed on average 41% of organic carbon and approximately18% of total PM2.5 mass (8).

There is no reason to think that Utah's largest cities would be much different. In fact the magnitude of the problem in the Salt Lake City area was further identified by an important study published in June, 2013 (9) suggesting that smoke from fireplaces, wood stoves and cooking grills was responsible for as much direct PM2.5 as vehicles when the PM2.5 was above 20 ug/m3. This further speaks to the inadequacy of the current wood burn control program in minimizing the problem.

Furthermore, **the emissions from modern combustion appliances for wood logs may increase ten-fold if they are not operated appropriately** (10).

The EPA itself estimates that **a single fireplace operating for an hour, burning 10 pounds of wood, will generate more PAHs than 130,000 cigarettes** (11). There are a few studies suggesting that particulate pollution in wood smoke from wildfires is much more toxic to lung macrophages than an equivalent concentration of similar sized particulate pollution found in typical urban smog (12,13,14,15).

According to the California Air Resources Board the inhalable particle pollution from one woodstove is equivalent to the amount emitted from 3,000 gas furnaces producing the same amount of heat per unit. While so called EPA certified wood stoves may be cleaner, they still cannot begin to approach the emissions level of a natural gas furnace.

Woodsmoke is not just an outdoor problem. The particles are very small (ultrafine), ranging from .2 microns at the start of the burn period to .05 microns as the burn cycle progresses. Particles of this size behave like gases. There is no practical way to prevent wood smoke pollution from seeping into nearby homes. The extremely small size of the particles results in the particles remaining suspended in the atmosphere for long periods making a disproportionate contribution to airshed pollution. Stagnant conditions and winter temperature inversions result in wood smoke hanging close to the ground, easily penetrating homes and buildings.

A study by the University of Washington showed that **50 to 70 percent of the outdoor levels of wood smoke were found in nearby homes that were not burning wood.**

EPA did a similar study in Boise, Idaho, with similar results (16). A study in Vancouver reported that woodsmoke particles are 7 times more likely to be breathed into our lungs than the average PM2.5 particle in Vancouver's air (17,18).

2. Wood smoke is even more toxic than other particulate pollution

Wood smoke is an extremely toxic, public health hazard. It contains over 200 chemicals and compound groups. The emissions are almost entirely in the inhalable size range (19). **Components of wood smoke are very similar to those in cigarette smoke**. They include particulate matter, carbon monoxide, formadehyde, sulfur dioxide, nitrogen oxides, dioxins, and polycyclic aromatic hydrocarbons (PAHs) (20). Furthermore, like with cigarettes, those who are doing the wood burning, are the most victimized by the pollution generated.

A report by Environment and Human Health, Inc., The Health Effects of Wood Smoke, cites medical research that indicates wood smoke interferes with lung development in children and increases a child's risk for serious lower respiratory infections like bronchitis and pneumonia (21). Wood smoke exposure can depress the immune system and damage the pulmonary epithelium (22) and increase arterial stiffness (23)

The very small size of the particulate emissions and high levels of PAH from wood smoke may account for its excessive toxicity compared to fossil fuel generated PM. Ultrafine particles are more potent in inducing inflammatory responses than fine particles (24,25,26,27). Wood smoke produces high levels of free radicals, DNA damage as well as inflammatory and oxidative stress responses in gene expression in cultured human cells (28).

The EPA estimates that the lifetime cancer risk from wood stove smoke is twelve times greater than that from an equal volume of second hand tobacco smoke. (The Health Effects of Wood Smoke, Washington State Department of Ecology). Burning two cords of wood produces the same amount of mutagenic particles as driving 13 gasoline powered cars 10,000 miles each at 20 miles/gallon or driving 2 diesel powered cars 10,000 miles each @ 30 miles/gallon (29).

Wood smoke particles have been reported to induce DNA damage in vitro in human monocytic and epithelial cell lines and in a murine macrophage cell line. Particles from three different combustion appliances (old boiler, modern boiler and pellets boiler) with varying content of organic carbon showed a similar genotoxic potency (30). However, particles from poor combustion, i.e. low temperature wood boilers, seem to have greater

effects on both cytotoxicity and DNA damage than particles from more complete combustion conditions.

Free radicals produced from wood smoke are chemically active for twenty minutes. In contrast tobacco smoke free radicals are chemically active for thirty seconds. **Wood smoke free radicals may attack our bodies cells up to forty times longer once inhaled** (31). Animal toxicology studies show that wood smoke exposure can disrupt cellular membranes, depress macrophage activity, destroy ciliated and secretory respiratory epithelial cells and cause aberrations in biochemical enzyme levels (32).

Wood burning is also the largest source of PAHs into the urban environment, and the third largest source of dioxin exposure in the US (33). Dioxins are one of the most intensely toxic compounds to which humans are ever exposed. Dioxins, and many of the other chemicals in wood smoke, are exactly the type of chemicals that the American College of Obstetricians and Gynecologists, and the American Society for Reproductive Medicine addressed in a prepared statement last fall, stating,

"Reducing exposure to toxic environmental agents is a critical area of intervention for obstetricians, gynecologists, and other reproductive health care professionals. Patient exposure to toxic environmental chemicals and other stressors is ubiquitous, and preconception and prenatal exposure to toxic environmental agents can have a profound and lasting effect on reproductive health across the life course. Prenatal exposure to certain chemicals has been documented to increase the risk of cancer in childhood…[we] join leading scientists and other clinical practitioners in calling for timely action to identify and reduce exposure to toxic environmental agents while addressing the consequences of such exposure (34)."

Dioxins fall into the broad category of endocrine disruptor chemicals. The Endocrine Society, internal medicine specialists in diseases of the pancreas, thyroid, adrenal and pituitary glands and hormone dysfunction, issued this public statement four years ago,

"Even infinitesimally low levels of exposure indeed, any level of exposure at all, may cause endocrine or reproductive abnormalities, particularly if exposure occurs during a critical developmental window. Surprisingly, low doses may even exert more potent effects than higher doses (35)."

Many chemicals like dioxins have been shown to not only impair the health of those exposed, but to also impair the health of subsequent generations who are not exposed, through epigenetic changes (36).

3. Inversion season is not the only time we are at risk from wood smoke

Medical research is well established that air pollution emitted into the community airshed when levels are relatively low has as much, or even greater health impacts than when PM or ozone concentrations are higher. In fact plotting a curve correlating sudden cardiac death (the signature outcome of PM exposure), vs. concentration of PM, yields a curve whose steepest part is at the lowest doses (37). In other words **eliminating wood burning in circumstances that already meet the NAAQS may be even more important in protecting public health**. This is not factored into NAAQS, but it is nonetheless an important consideration in regulating the creation of wood smoke.

4. Wood smoke is a large community economic liability and does not have a favorable carbon footprint

The Bay Area Air Quality Management District estimates that more than $1billion of medical expenses are caused by burning wood smoke in the Bay Area, including this calculation--**one wood fire can cost your next door neighbor an average of $40 in medical expenses.**

Given the overwhelming scientific consensus regarding a burgeoning, primarily human caused climate crisis, it is also important to consider the carbon footprint of wood burning. A 2010 study concluded that the amount of carbon released per unit of energy produced is actually greater for wood than it is for fossil fuels. **It is a common misconception that burning wood is carbon neutral**. Considering the entire carbon life cycle of wood, burning releases carbon now when we can least afford to do so, carbon that would have otherwise been stored for decades or perhaps centuries. While sustainable forestry practices can help repay that "carbon debt," those benefits don't accrue until the distant future, too late to be of much help. As a result of this study the state of Massachusetts changed its renewable portfolio standard to exclude biomass projects with long carbon payback periods.

Summary

We believe that through the Clean Air Act, the EPA has the authority, indeed the obligation, to make rules regarding wood burning devices more strict than the current proposal. Indeed, if the EPA makes a weak ruling like the current proposal, it virtually sanctifies the continued exposure of hundreds of millions of people to unnecessary, adverse health outcomes. The fact that these wood burning devices exist, that

companies make a profit manufacturing them, and that many people like to use them for reasons such as cost, convenience, or ambiance, is no excuse for the EPA not to fulfill its obligation. Frankly, the EPA cannot both protect wood burning manufacturers and simultaneously protect public health, and it is clearly mandated to do the latter.

It is long overdue that we consider subjecting one's neighbors to the high pollution consequences of wood burning devices is as much of an anachronism as allowing cigarette smoking on air planes. The medical science demands that the EPA act aggressively to curtail, as much as is legally possible, this serious public health menace.

Sincerely,

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References

1. Thatcher, T. & Kirchstetter, T. (2011). Assessing Near-Field Exposures from Distributed Residential Wood Smoke Combustion Sources. Report prepared for the California Air Resources Board.

2. Wu CF, Larson TV, Wu SY, Williamson J, Westberg HH, Liu LJ: Source apportionment of PM(2.5) and selected hazardous air pollutants in Seattle. Sci Total Environ 2007, 386:42-52.

3. Song Y, Tang X, Xie S, Zhang Y, Wei Y, Zhang M, Zeng L, Lu S: Source apportionment of PM2.5 in Beijing in 2004. J Hazard Mat 2007, 146:124-130.

4. Lewis CW, Norris GA, Conner TL, Henry RC: Source apportionment of Phoenix PM2.5 aerosol with the Unmix receptor model. J Air Waste Manag Assoc 2003, 53:325-338.

5. Saarikoski SK, Sillanpää M, Saarnio KM, Hillamo RE, Pennanen AS, Salonen RO: Impact of biomass combustion on urban fine particulate matter in central and northern Europe. Water Air Soil Pollut 2008, 191:265-277.

6. Rogge, W. F., L. M. Hildemann, M. A. Mazurek, G. R. Cass, and B. R. T. Simoneit. (1991) Sources of fine organic aerosol, 1, Charbroilers and meat cooking operations, Environ. Sci. Technol., 25, 1112–1125.

7. Rogge, W. F., L. M. Hildemann, M. A. Mazurek, G. R. Cass,and B. R. T. Simoneit. (1998) Sources of fine organic aerosol, 9, Pine, oak and synthetic log combustion in residential fireplaces, Environ. Sci. Technol., 32, 13–22.

8. Gorin, C, J. Collett, and P. Herckes. (2006) Wood Smoke Contribution to Winter Aerosol in Fresno, CA. Journal of the Air and Waste Management Association 56: 1584-1590 (quote on p. 1584).

9. K.E. Kelly, R. Kotchenruther, R. Kuprov, G.D. Silcox, Receptor model source attributions for Utah's Salt Lake City airshed and the impacts of wintertime secondary ammonium nitrate and ammonium chloride aerosol. Journal of the Air & Waste Management Association, 63:5, 575-590.

10. Nussbaumer T, Klippel N, Johansson L: Survey on measurements and emission factors on particulate matter from biomass combustion in IEA countries [abstract]. [http://www.verenum.ch/Publikationen/Biomass-Conf9.2.pdf] website 16th Eurpoean Biomass Conference and Exhibition, 2.- 6.June Valencia, Spain 2008.

11. Sacramento Metropolitan Air Quality Management District – Agenda, page 5. http://airquality.org/bod/2005 MarParticulateMatterSB656Briefing.pdf

12. Franzi LM, Bratt JM, Williams KM, Last JA. Why is particulate matter produced by wildfires toxic to lung macrophages? Toxicol Appl Pharmacol. 2011 Sep 16. [Epub ahead of print].

13. Environmental Health Perspectives, California Wildfires of 2008: Coarse and Fine Particulate Matter Toxicity, 2009, Vol. 117 (6):893-897.

14. Environmental Health Perspectives: Oxidative Punch of Wildfires, 117:A58, February, 2009.

15. Migliaccio CT, Kobos E, King QO, Porter V, Jessop F, Ward T. Adverse effects of wood smoke PM(2.5) exposure on macrophage functions. Inhal Toxicol. 2013 Feb; 25(2):67-76. doi: 10.3109/08958378.2012.756086.

16. New Hampshire Department of Environmental Services – Air Resources http://www.des.state.nh.us/ard/smoke.htm

17. Intake Fraction of Urban Wood Smoke, Ries et al.. Envir Sci Tech, 2009

18. Wood Smoke Brochure. Vol. 113, No. 4, April 2005 http://www.burningissues.org

19. Environmental Impact of Residential Wood Combustion Emissions and Its Implications, John A. Cooper, APCA Journal, Vol.30 No.8, August 1980

20. Minnesota Pollution Control Agency http://www.pca.state.mn.us/air/woodsmoke/healtheffects.html

21. Washington State Department of Ecology; Air Quality Program http://www.nwcleanair.org/pdf/aqPrograms/woodHeating/woodSmokeandYourHealth.pdf

22. Unosson J, Blomberg A, Sandström T, Muala A, Boman C, Nyström R, Westerholm R, Mills NL, Newby DE, Langrish JP, Bosson JA. Exposure to wood smoke increases arterial stiffness and decreases heart rate variability in humans. Part Fibre Toxicol. 2013 Jun 6;10(1):20. [Epub ahead of print]

23. American Lung Association – Air Quality http://www.lungusa.org/site/pp.asp?c=dvLUK9O0E&b=23354

24. Brown DM, Stone V, Findlay P, MacNee W, Donaldson K: Increased inflammation and intracellular calcium caused by ultrafine carbon black is independent of transition metals or other soluble components. Occup Environ Med 2000, 57:685-691.

25. Murphy SAM, Berube KA, Richards RJ: Bioreactivity of carbon black and diesel exhaust particles to primary Clara and type II epithelial cell cultures. Occup Environ Med 1999, 56:813-819.

26. Höhr D, Steinfartz Y, Schins RPF, Knaapen AM, Martra G, Fubini B, Borm PJA: The surface area rather than the surface coating determines the acute inflammatory response after instillation of fine and ultrafine TiO2 in the rat. Int J Hyg Environ Health 2002, 205:239-244.

27. Monteiller C, Tran L, MacNee W, Faux S, Jones A, Miller B, Donaldson K: The pro-inflammatory effects of low-toxicity low-solubility particles, nanoparticles and fine particles, on epithelial cells in vitro: the role of surface area. Occup Environ Med 2007, 64:609-615.

28. Danielsen PH, Møller P, Jensen KA, Sharma AK, Wallin H, Bossi R, Autrup H, Mølhave L, Ravanat JL, Briedé JJ, de Kok TM, Loft S. Oxidative stress, DNA damage, and inflammation induced by ambient air and wood smoke particulate matter in human A549 and THP-1 cell lines. Chem Res Toxicol. 2011 Feb 18;24(2):168-84. Epub 2011 Jan 14.

29. Dr. Joellen Lewtas, Contribution of Source Emissions of the Mutagenicity of Ambient Urban Air Particles, U.S. EPA, #91-131.6, 1991)

30. Karlsson HL, Ljungman AG, Lindbom J, Möller L: Comparison of genotoxic and inflammatory effects of particles generated by wood combustion, a road simulator and collected from street and subway. Toxicol Lett 2006, 165:203-211.

31. Lachocki, Pryor, et al, Persistent Free Radicals in Wood smoke, Louisiana State University, Free Radical Biology & Medicine Vol.12, 1992)

32. Timothy V. Larson and Jane Q. Koenig. A Summary Of Emissions Characterization And Noncancer Respiratory Effects Of Wood Smoke, U.S.EPA-453/R-93-036, Dec. 1993

33. EPA 1994, Loretta Ucelli spokeswoman, Washington Post

34. http://www.acog.org/Resources_And_Publications/Committee_Opinions/Committee_on_Health_Care_for_Underserved_Women/Exposure_to_Toxic_Environmental_Agents

35. https://www.endocrine.org/endocrine-press/scientific-statements

36. Schmidt C. Uncertain Inheritance: Transgenerational Effects of Environmental Exposures. Environ Health Perspect; DOI:10.1289/ehp.121-A298

37. Peters, A. Air Quality and Cardiovascular Health: Smoke and Pollution Matter. Circulation. 2009: 120:924-927