Vulnerable Populations: Fetal and Infant Health Outcomes of Wildfire Smoke

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This white paper was completed for a graduate-level class at the University of Montana to assist the efforts of Climate Smart Missoula. We hope this work proves useful to those looking into the effects of wildfire smoke on pregnant women, fetuses and infants. For questions or comments, email info@climatesmartmissoula.org.

This paper is organized as follows:

INTRODUCTION

As the climate changes, wildfire smoke events are becoming more common. Decades of fire suppression management decisions and hotter, drier summers have led to devastating wildfires, and therefore wildfire smoke, that can become a major public health crisis far from the combustion source. Fires in California at the beginning of the century seem to have brought the issue to light and spurred an influx of wildfire smoke public health research in the United States. Significant gaps remain in this relatively new field of research however. Some of these gaps can be filled in through corollary air pollution studies but the subject warrants much additional research.

Thus far, clear evidence supports a significant relationship between wildfire smoke and several health outcomes. Recent meta-analysis studies conducted by Reid et al. (2016) and Cascio (2017) both conclude that a strong association exists between all-cause mortality and wildfire smoke (1336, 588). These studies also find significant evidence that wildfire smoke induces pulmonary stress such as exacerbation of asthma and chronic obstructive pulmonary disease (COPD). Evidence also suggests that smoke events can increase instances of lower respiratory tract infections like bronchitis and pneumonia (Reid et al., 2016, p. 1336-1338; Cascio, 2017, 588). Less clear are the relationships between smoke and cardiovascular diseases, but new research seems to be amassing some evidence for a connection. Forsberg et al. (2012) claim, "...studies are now linking airborne exposure to finely sized particulate matter ($PM_{2.5}$) with cardiovascular conditions and increased cardiovascular-associated mortality" (p. 99). Both Reid et al. (2016) and Cascio (2017) draw more hesitant conclusions citing inconclusive evidence for a relationship (Reid et al., 2012, p. 1338-1339; Cascio, 2017, 588-589). Despite newer research, study results remain too mixed to make a confident claim of association between wildfire smoke and cardiovascular health.

Study of these health outcomes help identify vulnerable populations as those particularly sensitive to pulmonary and cardiovascular stress including those with pre-existing pulmonary and cardiovascular conditions, children, and the elderly. Pregnant women, fetuses, and infants are also often identified as vulnerable populations, but with little explanation. Cascio (2017) writes, "Susceptible populations probably include people with pre-existing respiratory disease, middle-aged and older adults, children, pregnant women and fetuses" (p. 588). The study's only other mention of pregnant women and fetuses comes tacked onto a paragraph largely about cardiovascular outcomes and cites only one study linking smoke to low birth weight (589). Reid et al. (2016) similarly keeps discussion brief by confining their scope to studies of birth weight outcomes and concluding, "Corroborative evidence suggests that wildfire smoke exposure

effects on birth outcomes are plausible" (1339). So should infants and pregnant women be considered a vulnerable population? Significant research gaps need to be addressed to definitively answer, but corollary studies of ambient air pollution, and of fine particulate matter measuring less than 2.5 micrometers ($PM_{2.5}$) specifically, suggest there are significant reasons to be concerned.

RESEARCH GAPS AND CHALLENGES

Beyond specific perinatal research gaps for varied health outcomes, geographies or etc, the category of pregnant mothers and infants is in itself a major research gap for smoke studies. In their guide for nurse practitioners, Forsberg et al. (2012) suggest that health professions think of the vulnerabilities of pregnant women to wildfire smoke in the same way as cigarette smoke because, "There are no known studies of the effects of wildfire smoke on pregnant women…" (104). Search terms such as "wildfire smoke and infants" generates few relevant results beyond birth weight studies. As a major component of both wildfire smoke and ambient urban air pollution, looking at $PM_{2.5}$ can help broaden the scope, but research is still limited.

The lack of research about wildfire smoke and mothers, fetuses, and infants can largely be attributed to challenges of methodology. All studies of wildfire smoke and health have few obstacles in common. First, studies are often necessarily retrospective as prediction of a smoke event can be difficult. These retrospective studies then usually must rely on health outcome data (i.e. hospitalization records) that provide a limited picture of possible consequences. From the other end, study results can be mixed because of difficulty assigning accurate measures of exposure and great heterogeneity in doing so across studies. Some studies assign exposure by the nearest monitor, some adjust these numbers from self-reported behaviors, others use satellite imagery and air quality monitoring reports to model exposure, and a few use source-oriented models that look at point of combustion (Reid et al., 2016, 1335; Laurent et al., 2016, 472). Finally, the rural nature of many smoke events mean air quality monitoring stations may be sparse and the available study populations too small to generate "adequate statistical power" (Reid et al., 2016, 1334). These difficulties only cover issues with health and wildfire smoke generally. Outside of wildfire, the field of air pollution and reproductive health comes with a whole host of additional issues.

In 2007 a number of epidemiologists and other health scientists convened to discuss the methodological issues of studying air quality and reproductive health outcomes. Woodruff et al. (2008) summarized the workshop findings and identified four areas of concern. The first concern was for confounding and effect-measure modification challenges. They looked at questions such as should, and how can, a study control for confounding variables like race and class. For example, is maternal hypertension an extraneous factor that should be controlled for or is hypertension exacerbated by smoke and therefore an essential component for understanding risk to fetal and infant health? "Essentially, there is difficulty in distinguishing between the possible direct effects of air pollution on fetal growth and the possible effects of air pollution on other pregnancy factors, which in turn can be independent risk factors of fetal growth restriction and/or make pregnancies more susceptible to air pollution," writes Woodruff et al. (2008, p. 314). The group's second point of concern addressed difficulties with defining exposure citing similar issues to those associated with wildfire smoke studies (p. 315). Relatedly, multiple studies have attempted to pinpoint exposure windows, or in other words, at what point during a pregnancy the fetus is at risk. Cross-study comparisons suffer from inconsistent definitions of gestational windows and uncertainty about exact points of conception. The inconsistencies inhibit researchers' ability to conclude whether the fetus is more at risk during the first or last trimester of a pregnancy (p. 316-317). Finally, ambient air pollution is made up of multiple compounds that are difficult to isolate. Woodruff et al. write that they are concerned about the, "...variability in the types of air pollutants evaluated and which individual pollutants or combination of pollutants are identified as the pollutant(s) associated with the perinatal outcome" (p. 317).

This final concern has considerable consequence for comparison of ambient urban pollution studies with wildfire smoke studies. Even if studies look at the shared $PM_{2.5}$ pollutant, the difference in source of

pollution introduces another set of variables that could render comparison inadequate to address gaps in wildfire smoke research. The term particulate matter does not refer to a uniform substance, but rather is a size assignment for a variety of substance. Particulate matter can be as simple as elemental carbon to more complex metals, oils, and etc. (Steingraber, 2010, 176). The effect of these differences is unknown. More research into wildfire smoke related health outcomes is clearly needed as are advances in methodology. In the meantime, some educated leaps and guesses should be made to act proactively on this issue. Knowledge of human biological processes and studies of the general effects of $PM_{2.5}$ provide enough evidence to raise concern.

WHAT WE KNOW

In wildfire smoke particulate matter presents at the highest concentration compared to other pollutants such as carbon dioxide, monoxide, hydrocarbons, and hazardous organic compounds. These high concentrations make it the pollutant of most concern for researchers. Of particulate matter, the fine particulates—those 2.5 microns and smaller—are most concerning as they *cannot* be filtered out in the nose or upper airways and so reach and lodge in the respiratory system (Forsberg et al., 2008, 99-100).

The EPA air quality standards for $PM_{2.5}$ currently sit at 12.0 $\mu g/m^3$ averaged for the year and 35 $\mu g/m^3$ averaged over a 24-hour period (NAAQS Table). A few more numbers to put these standards and the wildfire smoke issue in some perspective. On December 4, 2018 (day of writing) in Missoula, Montana, monitoring recorded a clear, healthy air day of 8.6$\mu g/m^3$ (DEQ Air Monitoring Data). The 48-hour $PM_{2.5}$ average for Beijing, China on the same day was 95$\mu g/m^3$ which they classify as moderate air quality but would be classified as unhealthy by the Montana Department of Environmental Quality (Beijing Air Pollution: Real-time Air Quality Index). During the 2017 wildfires in Missoula, monitors recorded a 144.5$\mu g/m^3$ average on September 4, 2017—one of the most noticeably hazy days of the wildfires. The air quality hazard peaked at a one-hour average of 471.3$\mu g/m^3$ between 6:00 and 7:00am that morning (DEQ Air Monitoring Data).

Biological Mechanisms for Adverse Effects of Smoke

Regardless of their source and make-up, $PM_{2.5}$ of all types causes concern because of how the human body reacts to the foreign matter. Forsberg et al. (2008) warn nurse practitioners that, "...inhalation of increased concentrations of $PM_{2.5}$ affects health by such mechanisms as increased oxidative stress, systemic inflammation, endothelial dysfunction [circulation], increased blood viscosity, proatherothrombosis [clotting], hypertension, and autonomic [nervous system] dysfunction of the heart" (p. 103). $PM_{2.5}$ can cause pulmonary stress directly through their presence in the lungs, but the ultra-fine particles can also enter the bloodstream through the alveoli and be carried to other systems (Forsberg et al., 2008, p. 100; Liu et al., 2016, p. 3278).

Studies that find associations between particulate matter and adverse effects on fetal development hypothesize that $PM_{2.5}$ in the bloodstream sparks a series of biological processes that affect placenta and fetal development. Liu et al. (2016) write that once $PM_{2.5}$ enters the bloodstream, "The particles are recognized as foreign matter, the local immune response is activated, and pro-inflammatory cytokines are released" (p. 3278-3279). Prolonged systemic inflammation affects a host of other processes and can decrease nutrient and oxygen supply to the fetus (p. 3279). These processes provide a basis for hypothesis and explanations of the link between fetal and infant health outcomes with $PM_{2.5}$ pollutants in wildfire smoke.

Studied Health Outcomes

Studies of ambient air pollution and infant and fetal health outcomes thus far have found evidence for lowered birth weights, changed placental development, adult cardiovascular effects, compromised immune and lung function in adolescence, and risk of fetal death resulting in stillbirth (Stieb et al., 2012; Liu et al., 2016; Tanwar et al., 2017; Black et al., 2017; DeFranco et al., 2015). The studies covering birth

outcomes like low birth weight and stillbirths use public human health data while systems studies that look at the placenta or later developmental outcomes use animal subjects. The heterogeneity of test subjects, differences in developmental study period, and varied sources of exposure make comparison of these studies difficult. However, when taken as a whole, they begin to suggest that mothers and public health workers should take precautions to protect against the negative health outcomes of wildfire smoke.

Most of these studies relate the $PM_{2.5}$ component of general ambient air pollution to health outcomes rather than wildfire smoke specifically. Since they focus on $PM_{2.5}$ some comparisons can be made, but should be understood as such. Casting doubt on comparability of air pollution and wildfire smoke studies, at least one study attempting to understand what components of air pollution drive risk of low birth weight did not observe a correlation between $PM_{2.5}$ and low birth weight. They even found that $PM_{2.5}$ from woodsmoke was inversely related (households with $PM_{2.5}$ from woodsmoke had higher than average birth weights) although they attribute this to possible uncontrolled confounding factors such as positive healthy environments associated with woodstove homes like access to outdoor recreation. The authors of the study go on to acknowledge that their findings are in opposition to other major studies and meta-analysis (Laurent et al., 2016, 476). Despite this study, the bulk of evidence suggests that $PM_{2.5}$ is a major component of pollution contributing to health outcomes. As discussed, the hypothesized biological pathways for health outcomes function similarly no matter the source of $PM_{2.5}$ because all foreign matter can trigger the body's inflammatory responses. For the studies described below comparability between $PM_{2.5}$ in urban pollution and wildfire smoke is assumed in order to describe a more complete picture of what the *possible* effects of a major smoke event can be on fetuses and infants.

Low birth weight (LBW) studies comprise the bulk of perinatal health and smoke research. Birth weight concerns health professionals because LBW weakens system functions making it more difficult for infants to fight infection and overcome other health complications. “Preterm birth and low birth weight are well-known to be associated with increased neonatal morbidity and mortality as well as possible increased morbidity in adulthood,” summarizes Stieb et al. (2012, p. 101). A meta-analysis of ambient air pollution, low birth weight, and preterm birth by Stieb et al. (2012) looked at various components of ambient air pollution and attempted to pool statistics to make estimates of exposure on the number of grams decrease in birth weight. Looking at 61 studies of low birth weight and preterm births, Stieb et al. (2012) found that PM_{10} (particles bigger than $PM_{2.5}$) exposure of 20 $\mu g/m^3$ is associated with a -16.8g change in birth weight. For $PM_{2.5}$, 10$\mu g/m^3$ (well below national standard of 35$\mu g/m^3$) exposure is associated with a -23.4g change in birth weight (p. 107). The analysis also looked at carbon monoxide—another substance found in wildfire smoke. They found 1ppm exposure associated with -11.4g decrease in birth weight (p.107). Besides these dose-response type estimates, the study also pooled “odds ratios,” or in other words, they looked at the odds of a low birth weight outcome for a certain exposure level. An odds ratio above one means there is a correlation between exposure and greater instances of low birth weight. At their respective exposure levels, PM_{10} had an odds ratio of 1.1, $PM_{2.5}$ odds ratio was 1.05, and carbon monoxide at 1.07 (p. 107). Stieb et al. (2012) conclude that, “...pooled estimates of effects are generally indicative of associations between CO, NO_2, PM and pregnancy outcome…” (p. 110). “Generally indicative” seems like a fairly weak statement and casts no light as to whether the effects are of great enough magnitude to be particularly concerning for infant health.

The Stieb et al. (2012) meta-analysis also attempted to determine risks by trimester, but heterogenous results across studies left this question unanswered. In this case, wildfire smoke studies may actually be able to cast light on this gap because of their temporal nature. An often-cited study by Holstius et al. (2012) looked at birth weight outcomes surrounding the 2003 California wildfires. They were able to compare trimesters because of exposure differences across gestational age during the time of the wildfire. At the height of the wildfires, $PM_{2.5}$ concentrations reached 90 $\mu g/m^3$ and remained around 75 $\mu g/m^3$ even under light smoke conditions (2-3 times higher than the national 24-hour standard) (Holstius et al., 2012, p. 1340). This massive study of almost 900,000 births found a 6.1g decrease in birth weight across all trimesters for infants exposed to wildfire smoke in utero versus non-exposed fetuses. The largest observed

effect of 9.7g decrease came in the second trimester (p. 1342). A study in Brazil of mothers exposed to biomass burning also looked at trimesters and divided exposure concentrations by quartile. They found a significant association between the highest quartile of exposure and the 2nd and 3rd trimesters where odds ratios were 1.51 and 1.5 respectively (Candido da Silva et al., 2014, p. 3-5). In more general terms, "...an increase in approximately 50% of LBW occurred when the fourth quartile was compared with the first quartile range of PM2.5 and CO" (Candido da Silva et al., 2014, p. 4). Despite these findings, neither study raises alarms nor makes strong prescriptions for public health action with Holstius et al. (2012) writing, "This study indicates that maternal exposure to wildfire events may result in modestly lower infant birth weight. A small decline in birth weight is unlikely to have clinical relevance for individual infants, and there is debate about whether a small shift in the population distribution of birth weight has broader health implications" (p. 1344). LBW studies generally agree that $PM_{2.5}$ has some effect on infant development, but are inconclusive about the public health implications.

In contrast to LBW, studies of stillbirth risk have large public health implications, but more mixed results about whether a correlation exists with $PM_{2.5}$. Pearce et al. (2010) studied births over a 30-year span (1962-1992) in Newcastle upon Tyne, UK. The study found no correlation between particulate matter and stillbirth, but looked exclusively at black smoke pollution records. Black smoke is associated with PM_4 rather than $PM_{2.5}$ and the larger size could mean better biological filtering capacity to protect against the pollution (p.121). DeFranco et al. (2015) conducted a similar mass study in rural and urban Ohio looking at births over a 5-year span. They found no correlation of stillbirth with chronic low levels of $PM_{2.5}$ exposure (average of 13 $\mu g/m^3$ or just above the national yearly standard), but did find a significant correlation between high exposure (defined as 16.22 $\mu g/m^3$) during the 3rd trimester (p. 5). They conclude, "...we found that pregnant women exposed to high levels of $PM_{2.5}$ during the third trimester of pregnancy had a 42% increased risk of stillbirth" (p. 7). A 42% increased risk for a 16.22 $\mu g/m^3$ exposure level is significant in light of the much higher averages associated with wildfire smoke events. This raises serious alarm bells and suggests stillbirth risk should be studied in greater depth to shed light on inconsistent results.

The mixed results of low birth weight studies and stillbirths bring up questions of what biological processes may or may not be occurring to produce these results. Liu et al. (2015) set out to answer this question by looking at the effects of $PM_{2.5}$ exposure on placenta development. Size and blood flow of the placenta can affect nutrient and oxygen availability causing stunted fetal development (p. 3275). The study exposed 10 newly pregnant rats to a cumulative 30 $\mu g/m^3$ of $PM_{2.5}$ delivered intravenously. Researchers determined the dosage by comparing breaths per minute of rats to humans and adjusting accordingly from the average $PM_{2.5}$ levels of 85.9 $\mu g/m^3$ found in Beijing (p. 3278). Two of the exposed rats and one control rat aborted leaving a relatively small sample size (p. 3276). Dissection of the placenta led to a number of findings including high white blood cell counts indicating infection in 7 of 8 exposed rats, thrombus (clotting) in 5 of 8, fibrin (scarring) in 3 of 8, and several other infection related outcomes in 2 of 8 rats (p. 277). The increased inflammation dues to infection and other findings can all contribute to decreased nutrient and oxygen supply to the fetus and therefore cause longer term health effects (p. 3279). From these findings Liu et al. conclude simply, "Pregnant women should avoid $PM_{2.5}$ exposure" (2015, p. 3279).

Beyond immediate infant outcomes, a few studies look at the longer-term effects of in utero or early life exposure to $PM_{2.5}$. One study, Tanwar et al. (2017), looked at adult cardiovascular health after in utero exposure to particulate matter. Researchers exposed pregnant mice to air with an average $PM_{2.5}$ concentration of 73.61 $\mu g/m^3$ every day of the pregnancy for 6 hours at a time (p. 2). Exposure here differs from Liu et al. (2015) by method (air versus direct injection) and particulate matter level. Tanwar et al. (2017) did not discuss exposure decisions in-depth and so the effects of these differences on outcome remain unclear. At 12 weeks of age the mice were studied for various markers of cardiovascular disease and other epigenetic changes. They found several of these markers including: left ventricle remodeling, impaired hemodynamics (blood flow), impaired cardiomyocyte function (muscular cells in the heart that aid contraction and expansion), inflammatory and profibrotic (scarring) response, increased collagen deposition (proteins commonly found in ligaments and tendons which cause a sort of stiffening effect in

heart muscles which also impairs contractive function), and altered "baseline expression and remodeling of calcium homeostatic proteins" (a calcium imbalance?) (p. 4-5). Interestingly, these findings applied largely to male rather than female offspring as females tend to have better "cardioprotective mechanisms" (p. 10). Tanwar et al. summarize the findings in their conclusion, "Acute and chronic $PM_{2.5}$ exposure impairs vascular reactivity and induces systemic inflammation, contributing to various manifestations of CVD [cardiovascular disease]" (2017, p. 8).

The final study that raises concerns about $PM_{2.5}$ exposure's effect on developmental health may also be the most relevant. While other studies focus on in utero exposure, Black et al. (2017) look at the effects of infant exposure on development and adolescent health outcomes. This study also uniquely studies wildfire smoke directly. During Northern California wildfires in 2008, outdoor housed infant rhesus macaque monkeys were exposed to elevated levels of $PM_{2.5}$ due to ambient wildfire smoke. The infant monkeys spent a total of 10 days above the 35 $\mu g/m^3$ national air quality standard with a maximum recorded concentration of 78 $\mu g/m^3$ (p. 659-660). Monkeys born the following year in 2009 were never exposed to particulate matter above air quality standards and so provided a comparison cohort to study various pulmonary issues. The study had two important findings: First, early life exposure correlated with "attenuation of PBMC responses to TLR ligands" or, in other words, immune system overreaction led to inflammation of airways. Second, they found reduced lung function with markers such as, "significantly reduced inspiratory capacity, residual volume, vital capacity, and functional residual capacity per unit of body weight" (p. 660-661). So the adolescent monkeys suffered from the dual impacts of reduced lung function and altered immune and inflammatory responses that can exacerbate pulmonary conditions like asthma. Like Tanwar et al.'s (2017) cardiovascular study, Black et al. (2017) found differences according sex but this time with greater effects attributed to females (p. 662). Black et al. (2017) conclude by tempering the public health implications of their conclusions, "Our findings from a cohort of nonhuman primates exposed to prolonged ambient wildfire smoke suggest that children who underwent similar exposures as infants may exhibit a similar health profile, with the important caveat that animals in our study were exclusively housed outdoors throughout the assessment period" (p. 663). This is an important caveat, but an acute exposure reaching a 144.5$\mu g/m^3$ average on a day during the 2017 Montana wildfires is well above the maximum exposure level of 78 $\mu g/m^3$ infant monkeys.

Effects of high-level acute versus low-level chronic exposure to $PM_{2.5}$ on human health is just another complexity that current research cannot yet answer. Obviously, much more research is needed to definitely understand the consequences of wildfire smoke. However, this question and others like it are somewhat irrelevant to a more general question of whether pregnant mothers, fetuses, and infants should be considered vulnerable populations in need of significant public health resources to protect. To answer this question, this paper looks at a variety of studies. While this prevents much analysis of the individual merits of each study, taken as a whole, this collection of studies begin to make a strong general connection between the $PM_{2.5}$ component of wildfire smoke negative fetal and infant health outcomes. Given this, measures should be taken to protect pregnant mothers and infants against wildfire smoke. But how?

PROTECTING AGAINST SMOKE

Lead by the British Columbia Centre for Disease Control and California public health offices, research, educational campaigns, and other efforts are underway to find the most effective ways of protecting against wildfire smoke. There are two main methods of protection: creating clean indoor air with filters and using filtration masks. The first is cited as greatly preferable to the latter.

$PM_{2.5}$ is too fine to be filtered through traditional surgical masks and so requires more advanced filtering. According to meta-analysis by the BC Centre for Disease Control (2014), testing of wet and dry handkerchiefs allowed 70-907% penetration of particulate matter while basic dust masks performed only marginally better with 60-90% penetration. "These types of masks may actually be detrimental, giving the wearers a false sense of security and encouraging them to increase their physical activity and time outdoors," conclude the report's author (BC Centre, 2014, 3). The report breaks effectively air purifying

masks into air-supplying and air-purifying. Air-supplying masks require significant training and require transport of a tank rendering them ineffective as a public health measure. Air-purifying masks however may be useful in the event the wearer must be outside.

A N95 mask is the most commonly recommended. The number 95 refers to the efficiency of filtration for 0.3μg particulate matter (BC Centre, 2014, 6). This size filtration is sufficient for particle pollutants in woodsmoke and so could be an effective measure of protection for pregnant mothers. However, public health officials recommend indoor air filtration over mask for good reason. Masks can protect only against particulate matter. Other wildfire smoke pollutants require different chemical filtration techniques. A combination mask can be used to protect against multiple pollutants, but prior knowledge of what chemical pollutants may be present is necessary to ensure efficacy. In addition, masks can cause a certain level of discomfort by making it difficult to breath and accumulating heat and humidity inside the mask. Finally, the effectiveness of a N95 mask is almost entirely dependent on proper fit that creates a seal. Masks do not fit persons with facial hair—and more importantly for this discussion, they do not fit children or infants (BC Centre, 2014, 7-8).

Creating healthy indoor air requires some kind of fine particulate filtration. Clean indoor air can be created through tightly sealed spaces combined with portable air filters or high rated air-conditioning filtration systems that recirculate indoor air. Figure 1 looks at the filtration efficiency of several classifications of air filters.

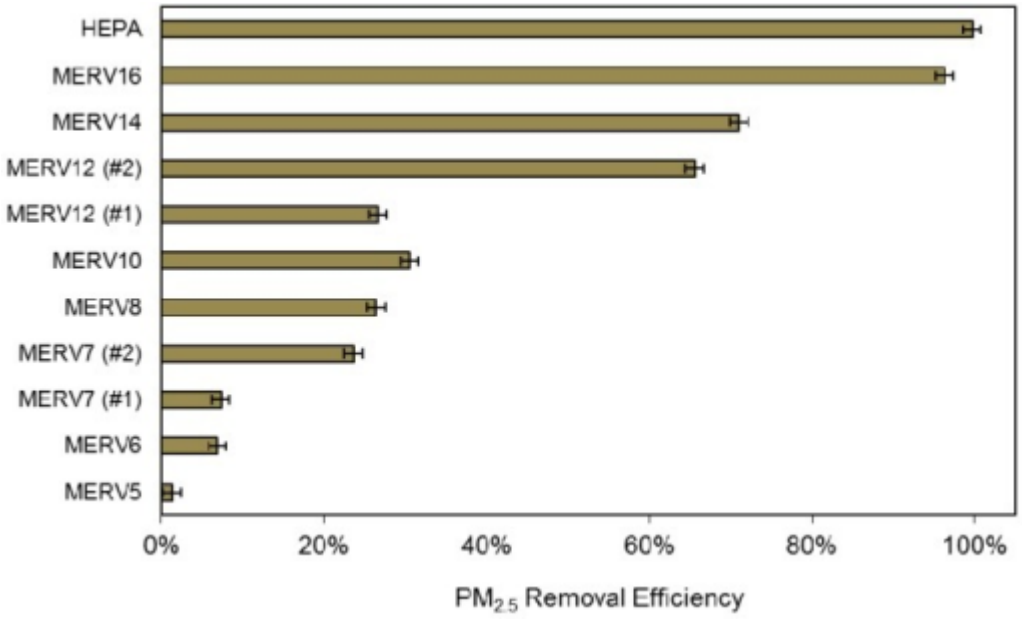

Figure 1: Zhao, Azimi, and Stephens, 2015

Minimum Efficiency Reporting Values (MERV ratings) can be found on most facility air conditioning units. Higher rated MERV units are less energy efficient however so it's recommended that higher efficiency filtration be swapped in during poor air quality events only. For portable units, high-efficiency particulate air (HEPA) filters are generally recommended. While Zhao, Azimi, & Stephens (2015) show nearly 100% removal efficiency, meta-analysis by Barn et al. find that with in-home testing reduction of $PM_{2.5}$ concentrations vary from 32-88% reduction depending on testing method (2016, 3). Even with mixed results, indoor air filtration can provide some level of protection for mothers and infants at relatively low cost. Portable HEPA filters comes with a room size recommendation that usually covers a typical single residential room. Portable units help with ease of transfer form a general living space to sleeping quarters at night, but multiple units may be needed. Basic HEPA units run near $100 which can be prohibitive for

some households. Therefore, identification of households with pregnant mothers and infants as vulnerable populations becomes important for public health interventions that provide filters.

NEXT STEPS

Challenging methodology and a lack of population specific research creates uncertainty around the specific impacts of wildfire smoke pollution on mothers, fetuses, and infants, but the existing body of research indicates health concerns are warranted. Some wildfire smoke affected geographies already work to protect these populations, but they may sometimes be secondary intervention priorities behind seniors and people with pre-existing conditions. So far this has been the case in Missoula, Montana where Climate Smart Missoula and the county health department have already distributed filters to homebound seniors in 2017. More recent efforts are underway to target other populations. In 2018 Climate Smart began assessment of school filtration capabilities and needs as well as distributed filters to nurseries throughout the county. The group would also like to begin efforts to reach pregnant and new mothers. This paper serves as background research for future educational campaigning which may include materials added to a general wildfire smoke safety informational page (montanawildfiresmoke.org, run by Climate Smart Missoula) as well as informational pamphlets directed to health care providers and distributed directly to mothers through OBGYN facilities, Dulas, and other health services.

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