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Whitley Cagle

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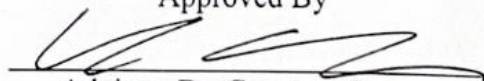
THE EFFECTS OF SHORT-TERM INDOOR AND PERSONAL EXPOSURE TO FINE  
PARTICULATE MATTER (PM<sub>2.5</sub>) ON CARDIOVASCULAR HEALTH: A REVIEW

By  
Whitley Ann Cagle

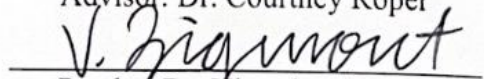
A thesis submitted to the faculty of The University of Mississippi in partial fulfillment of the  
requirements of the Sally McDonnell Barksdale Honors College.

Oxford, MS  
May 2022

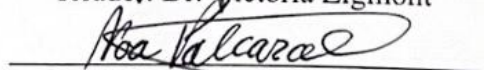
Approved By



Advisor: Dr. Courtney Roper



Reader: Dr. Victoria Zigmont



Reader: Dr. Noa Valcárcel

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## ABSTRACT

Fine Particulate Matter (PM<sub>2.5</sub>), a component of air pollution, has known effects on human health. However, there is limited research surrounding the specific cardiovascular impacts associated with these small particles entering into the bloodstream. This literature review aims to make associations between indoor and personal exposures to PM<sub>2.5</sub> and cardiovascular health. An online search was conducted to obtain scholarly articles using various key terms, and the articles were filtered through based on specific selection criteria. In the final review, we focused on 37 research articles which discussed PM<sub>2.5</sub> and its effects on the human cardiovascular system. Positive associations between PM<sub>2.5</sub> exposures and adverse cardiovascular health outcomes were found in 86% of the included studies. While indoor/personal exposure studies are not as prevalent as studies involving ambient (outdoor) air pollution, it is evident that conducting further research would provide greater insight into the complex cardiovascular health consequences associated with exposure to PM<sub>2.5</sub>.

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## LIST OF ABBREVIATIONS

PM	Particulate Matter
PM <sub>2.5</sub>	Fine Particulate Matter
WHO	World Health Organization
EPA	Environmental Protection Agency
CO	Carbon Monoxide
O <sub>3</sub>	Ozone
NAAQS	National Ambient Air Quality Standards
AHA	American Heart Association
CVD	Cardiovascular Disease
MI	Myocardial Infarction
IAQ	Indoor Air Quality
OSHA	Occupational Safety and Health Administration
BP	Blood Pressure
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
CIMT	Carotid Intima-Media Thickness
ECG/EKG	Electrocardiogram
RHI	Reactive Hyperemia Index
CRP	C-Reactive Protein Test for Heart Disease
MVF	Macrovascular Function
RH-PAT	Reactive Hyperemia-Peripheral Arterial Tonometry
BC	Black Carbon
CAM	Cardiac Autonomic Modulation
BAD	Brachial Artery Diameter
CHD	Coronary Heart Disease
COPD	Chronic Obstructive Pulmonary Disease



LE	Low-Efficiency (Filtration)
HE	High-Efficiency (Filtration)
AIP	Atherogenic Index of Plasma
ANS	Autonomic Nervous System
CNS	Central Nervous System

## **1 Introduction**

Each year, air pollution is responsible for the deaths of an estimated seven million people worldwide. According to data from the World Health Organization (WHO), nine out of ten people consistently breathe air that contains pollutants at levels that exceed WHO guideline limits. Low- and middle-income countries suffer most substantially from poor air quality due to lack of necessary resources and technology to combat or reduce pollution emissions. Air pollution itself comes in many forms, and can be both ambient (outdoor) and indoor (WHO, *Air Pollution*). Common pollutants found in the air include gaseous pollutants such as carbon monoxide (CO) and ozone (O<sub>3</sub>), and particles suspended in the air known as particulate matter (PM) (Du et al.). Examples of air pollutants range from smog over cities from vehicle exhaust and manufacturing plants to smoke inside the home from cooking, heating, or burning candles or incense. Household exposure to smoke from cookstoves and fuels accounts for 3.8 million of the 7 million air pollution-related annual deaths. These premature deaths occur often as a result of stroke, heart disease, chronic obstructive pulmonary disease, lung cancer, and acute respiratory infections brought on by exposure to air pollution (WHO, *Air Pollution*).

### **1.1 Fine Particulate Matter**

Fine particulate matter (PM<sub>2.5</sub>) is a special air pollutant that presents a unique and detrimental risk to human health. The Environmental Protection Agency (EPA) defines particulate matter (PM) in general as a mixture of solid particles and liquid droplets found in the air. The EPA separates particulate matter into categories based on size due to the fact that different sized particles affect the human body differently. Fine particulate matter (PM<sub>2.5</sub>) are inhalable particles measuring 2.5 micrometers (µm) in diameter and smaller. The composition of

particulate matter can include different combinations of hundreds of different chemicals emitted from a multitude of sources. Complex chemical reactions of air pollutants such as sulfur dioxide and nitrous oxides emitted from industrial plants and automobiles react in the atmosphere to form PM<sub>2.5</sub> (OAR US EPA, “Particulate Matter (PM) Basics”). The primary source of PM<sub>2.5</sub> is human combustion of fossil fuels from industry, automobiles, power generation, etc., but other relevant sources include biomass burning, heating, cooking, and other indoor activities (Brook, Rajagopalan, et al.). The National Ambient Air Quality Standards (NAAQS) set by the EPA for particulate matter pollution specifies a maximum amount of particulate matter that can be present in outdoor air. Currently, the standards for PM<sub>2.5</sub> exposure are less than 12 µg/m<sup>3</sup> annually and less than 35 µg/m<sup>3</sup> in a 24-hour period (OAR US EPA, “Particulate Matter (PM) Basics”).

## **1.2 Health Endpoints from PM<sub>2.5</sub> Exposures**

Fine particulate matter (PM<sub>2.5</sub>), because it measures less than or equal to 2.5 µm in diameter, is not only easily inhaled, but also has the ability to lodge deeply into the lungs as far as the alveoli and even enter into the bloodstream. Because of this property, fine particulate matter poses the most serious risk to human health of any other form of particulate matter found in the air. Numerous scientific studies have linked PM<sub>2.5</sub> to a multitude of serious health problems including nonfatal heart attacks, aggravated asthma, irregular heart beat (arrhythmia), decreased lung function, and even premature death in individuals with pre-existing conditions such as lung or heart disease (OAR US EPA, *Health and Environmental Effects of Particulate Matter (PM)*). Furthermore, the WHO estimates that PM<sub>2.5</sub> contributes to approximately 800,000 premature deaths per year, making it the 13th leading cause of mortality worldwide (WHO, *Air Pollution*). Both short-term and long-term exposures to PM<sub>2.5</sub> have adverse effects on human health. Long-term exposures, experienced by individuals residing in areas with high levels of

particle pollution, have been linked to a number of health problems such as reduced lung function, chronic bronchitis, and premature death. Short-term exposures (hours or days) to PM<sub>2.5</sub> typically aggravate existing lung disease, triggering asthma attacks and acute bronchitis. Furthermore, in individuals with pre-existing cardiovascular disease (CVD), short-term PM<sub>2.5</sub> exposures have been shown to trigger both fatal and nonfatal heart attacks and arrhythmias. However, PM<sub>2.5</sub> exposure can also impact healthy individuals with no history of cardiovascular or respiratory disease or dysfunction. The symptoms experienced by healthy individuals, although they are usually temporary, can include eye, nose, and throat irritation; coughing; phlegm; chest tightness; and shortness of breath. All ages are vulnerable to the negative health effects brought on by exposure to PM<sub>2.5</sub>. While older adults are at increased risk and are more likely to become hospitalized due to the possibility of undiagnosed lung or heart disease aggravated by PM<sub>2.5</sub>, young, healthy children are also faced with immense health risks. Because their lungs are still developing and they spend more time engaging in high levels of activity, young children are extremely susceptible to developing asthma or other respiratory diseases when PM<sub>2.5</sub> levels are high in their environment (OAR US EPA, *Health and Environmental Effects of Particulate Matter (PM)*).

Because PM<sub>2.5</sub> has a small enough aerodynamic diameter to enter and travel through the bloodstream, the negative impacts of the toxins carried on the surface area of these particles on the cardiovascular system are substantial. In fact, the cardiovascular health consequences of air pollution generally exceed health consequences within the respiratory system, despite the fact that particulate matter enters the lungs first via inhalation (Brook, Rajagopalan, et al.). However, knowledge of the cardiovascular impacts of particulate matter exposures is limited. The American Heart Association (AHA) released its first statement regarding air pollution and

cardiovascular disease in 2004, providing researchers and medical professionals with the baseline knowledge that air pollution is in fact linked to cardiovascular morbidity and mortality. The aim of this statement was to raise awareness of the severity of this issue and encourage further research (Brook, Rajagopalan, et al.). Since the release of this statement by the AHA, more research has been conducted to better understand pollution-induced cardiovascular risks. Numerous clinical studies have shown that particulate matter is strongly associated with increased risk for cardiovascular disease such as myocardial infarction (MI), cardiac arrhythmias, ischemic stroke, vascular dysfunction, hypertension and atherosclerosis (Du et al.). More highly-exposed areas of the population face an even greater risk. Because they are exposed to high levels of PM long-term (months to years), the risk for cardiovascular morbidity and mortality is significantly increased, which ultimately reduces life expectancy by several months to even a few years. Furthermore, PM pollution is particularly dangerous due to the fact that it has the ability to continuously and inconspicuously enhance acute cardiovascular risk for as long as the exposure lasts, both making existing cardiovascular disease significantly more severe over time and increasing the risk of developing cardiovascular disease in the future (Brook, Rajagopalan, et al.).

### **1.3 Indoor vs. Personal Exposures**

According to the Environmental Protection Agency (EPA), the average person spends about 90% of their time indoors, whether that is in their homes, at work or at school, or doing other activities involved in day-to-day life. Furthermore, individuals with increased sensitivity to the dangers associated with indoor air pollution such as young children, the elderly, and people with pre-existing cardiovascular and respiratory problems typically spend even more time within their places of residence (ORD US EPA). Indoor Air Quality (IAQ) is defined by the EPA as the

quality of the air within or surrounding buildings, especially as it relates to and affects the health and comfort of those living and/or working within them (OAR US EPA, *Indoor Particulate Matter*). Multiple sources are responsible for generating indoor particulate matter. Combustion activities within the home such as burning of candles or incense; use of fireplaces, space heaters, or kerosene heaters; and cigarette smoking all contribute substantially to levels of particulate matter within residences (OAR US EPA, *Indoor Particulate Matter*). Another major contributor of household air pollution is cooking with polluting fuels, also known as “dirty” fuels. These dirty fuels include coal, kerosene, and biomass such as wood, charcoal, crop residues, and animal manure (Rosário Filho et al.). Furthermore, the health consequences brought on by the release of these pollutants into the air are enhanced indoors by factors such as poor ventilation as well as high temperatures and levels of humidity. The Occupational Health and Safety Administration (OHSА) outlines strict standards regarding proper ventilation requirements within places of work in order to reduce levels of particulate matter and protect the health and safety of employees. However, it is much more difficult to regulate the indoor air quality within individual homes, and no clear air quality guidelines exist for many indoor environments (OAR US EPA, *Indoor Particulate Matter*).

Personal particulate matter exposures, as opposed to indoor exposures, directly follow the habits of an individual, rather than the level of PM exposure only when they are in their home or in a certain room. Because individuals spend a significant portion of their time indoors, personal exposures are heavily weighted by indoor PM concentrations. Personal monitoring of PM exposure involves monitors worn on the body of the study participant for a set period of time. This method, because the monitors move with the study participant rather than remaining stationary, provides a more holistic measurement of the substances in the air that individuals are

exposed to while carrying out the activities of daily life. Additionally, personal PM monitoring primarily measures the concentration of particulate matter near the breathing zone of the study participant, allowing for a more accurate estimate of how much PM is actually entering the body during a given period of time. However, there are some disadvantages associated with personal exposure monitoring. For example, personal monitoring requires a much higher level of participant compliance and participation than stationary indoor monitoring in order to ensure accurate measurements (Chow et al.).

#### **1.4 Study Overview**

For this study, we reviewed articles that evaluated associations between indoor and/or personal exposure to fine particulate matter and various cardiovascular health implications. I hypothesize that significant positive associations will be found between PM<sub>2.5</sub> and adverse cardiovascular effects. The aim of this study is to demonstrate the importance of further research surrounding indoor and personal concentrations of PM<sub>2.5</sub> and its impact on human cardiovascular health in order to improve public health efforts.

## **2 Methods**

### **2.1 Search Databases**

A systematic review of published and peer-reviewed articles and research studies was conducted primarily using Google Scholar and The University of Mississippi Library's One Search tool. Databases searched included PubMed(NIH), Embase (Elsevier), and Scopus (Elsevier). The aim of the search strategy was to locate studies that linked fine particulate matter (PM<sub>2.5</sub>) and its constituents to cardiovascular health consequences. Keywords included: cardiovascular, fine particulate matter, and environmental exposure. Initial searches resulted in a total of 1,349 articles following the removal of duplicates and incomplete text (i.e. only abstracts available).

### **2.2 Selection Criteria**

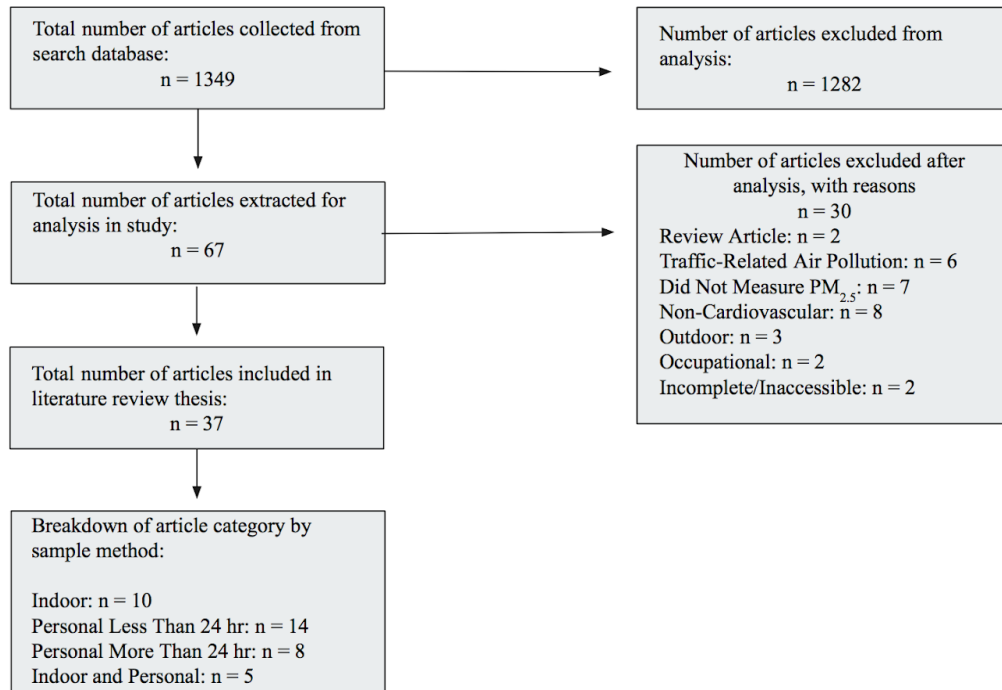
Following the initial search, selections were made using the selection criteria described in detail below. For the purposes of this review, there were no time, language, or geographical limitations. All individuals regardless of sex, age, and co-morbidities were included. Exclusions included occupational PM<sub>2.5</sub> exposures and outdoor exposure studies such as studies involving traffic-related air pollution exposures (TRAPs). Exposures to PM<sub>2.5</sub> included both short-term exposures (hours to days) and long-term exposures (months to years). All studies included in this review discussed personal and/or indoor PM<sub>2.5</sub> concentrations and made associations to human cardiovascular health consequences. This included observational studies such as cohort, cross-sectional, and clinical studies, but excluded review articles, working papers, non-peer reviewed studies, and books/book chapters. Cardiovascular endpoints included mortality and morbidity associated with cardiac events (i.e. hypertension, myocardial infarction (MI), heart rate variability (HRV), atherosclerosis, and other ischemic heart disease).



### **2.3 Article Selection & Data Extraction**

Articles were filtered through using the title and abstract to determine if they meet the eligibility criteria for this project (i.e. assessment of cardiovascular health effects and PM<sub>2.5</sub> concentrations measured for a set period of time using indoor and/or personal monitors). This process resulted in a total of 67 articles to be further screened for final use in this review. Following this initial screening, data was extracted from each article and consolidated into an Excel spreadsheet. Data extracted included: article title, authors, study location, time during which the study took place, number of participants, age range of participants, sex of participants, pre-existing conditions of participants (if applicable), exclusions, monitor placement, sampling period, specific sources, other pollutants measured (if applicable), health endpoint(s) assessed, statistical methods, interventions (if applicable), average PM<sub>2.5</sub> concentration, health associations, results, and conclusions. After data extraction, the studies were organized into three main categories based on sample collection: indoor sampler studies, personal (worn) sampler studies, and studies involving both indoor and personal samplers. Then, the studies involving only personal samplers were further divided into two additional categories based on sample duration, one group containing studies in which samples were taken for 24 hours or less, and the other containing studies in which samples were recorded for more than 24 hours. Any studies that did not clearly fit within these categories were excluded from the final analysis.

Figure 1: Schematic Diagram of Article Selection Process



### 3 Results

After narrowing down the search process, a total of 67 articles were reviewed. Following the screening process, 37 articles were selected for the final analysis. Of these, 10 were indoor exposure studies, 22 were personal exposure studies, and 5 were studies which considered both indoor and personal fine particulate matter (PM<sub>2.5</sub>) exposures simultaneously. The eliminated articles were excluded from use in the final review for various reasons. For example, 6 articles were excluded because they observed the effects of traffic-related (outdoor) air pollution on PM<sub>2.5</sub> exposure and cardiovascular health, 3 articles were excluded because they modeled and/or estimated PM<sub>2.5</sub> exposure as opposed to measuring it directly, and 2 were excluded for incompleteness (i.e. no observations or results included, not peer-reviewed, etc.). The remaining studies were excluded if they focused exclusively on other, non-cardiovascular PM<sub>2.5</sub>-associated health consequences such as respiratory complications and low birth weight/premature birth in newborns.

#### 3.1 Indoor PM<sub>2.5</sub> Associations with Cardiovascular Health

*Table 1* presents the articles that studied indoor exposures to PM<sub>2.5</sub> and made associations to human cardiovascular health. The indoor air pollution studies were conducted in both rural areas such as Nangong County in the Hebei Province of North China and the city of Puno in Peru, and urban areas such as British Columbia, Canada; Shanghai and Beijing, China; and Copenhagen, Denmark. A majority of the studies excluded both smokers and smoking households. Only 4 studies (studies by Pang, Caravedo, Pan, and Dong et. al.) included participants who smoked or lived with smokers. Sampling periods of the included indoor studies ranged from 2 hours to 7 consecutive days, demonstrating the cardiovascular effects of indoor PM<sub>2.5</sub> exposures ranging from 2 hours to 2 weeks. Of the 10 indoor studies, 8 continuously

monitored the PM<sub>2.5</sub> concentration in the main activity room of the home, typically the living room, and 2 monitored kitchen exposures during the process of actively cooking. The study by Dong et. al. presented in Table 1 is a survey of 18,484 households across China. This study associated a higher mortality risk with indoor and ambient (outdoor/environmental) PM<sub>2.5</sub> exposure combined compared with ambient exposure alone, emphasizing the detrimental health effects indoor air pollution has the potential to induce (Dong et al.).

The studies by Pang et. al. and Caravedo et. al. were intervention studies that took place in rural communities/villages where homes are primarily heated by burning fossil fuels such as coal and/or biomass fuels. In both studies, participants were divided into a coal/biomass fuel-burning group and an intervention clean fuel-burning group. The results of both studies showed a significant difference in exposure levels between the two groups after 24 hours of continuous monitoring. The results of Pang et. al.'s study showed that participants in the coal-burning group were exposed to a range of 77.0-112.0 µg/m<sup>3</sup> of PM<sub>2.5</sub>, while participants in the intervention group were exposed to a range of only 12.0-15.0 µg/m<sup>3</sup> (Pang et al.). Furthermore, in Caravedo et. al.'s study, participants in the biomass fuel-burning group were exposed to an average concentration of 178 µg/m<sup>3</sup>, while participants in the intervention group were exposed to an average concentration of only 27 µg/m<sup>3</sup> in a 24-hour span (Caravedo, Painschab, et al.). Pang et. al. defined carotid intima-media thickness (CIMT) of greater than 1.5 mm (normal thickness for adults ranges from 0.7 to 0.8 mm) or focal wall thickening that protruded into the lumen of the heart more than 0.5 mm (more than 50%) as atherosclerosis (Pang et al.). Atherosclerosis is a condition in which abnormal thickening or hardening of the inner lining of an artery is present, usually due to a buildup of plaque along the vessel wall, resulting in inhibition of the natural flow of blood. Atherosclerosis is a preliminary condition that often goes undiagnosed, but can

lead to more serious conditions later in life. For example, inhibition of blood flow to the brain can result in stroke, lack of blood flow to the heart can result in heart attack, and reduced blood flow to the limbs can result in severe pain and tissue death (Johns Hopkins Medicine). Among the 426 participants in the coal-burning group, 60.8% suffered from carotid atherosclerosis, while 52.8% of the 326 intervention participants displayed the same symptoms (Pang et al.). The results of this study indicate that long-term exposure to indoor smoky coal combustion for heating has the potential to significantly increase the risk of developing atherosclerosis and systemic inflammation, particularly in rural communities.

Both the study by Gabdrashova et. al. and the study by Cole-Hunter et. al. sampled PM<sub>2.5</sub> in kitchens for a short period of only 2 hours (or during the time it took to cook meat on a stove from start to finish). These extremely short-term exposure studies of only a few hours display the immediate effects of PM<sub>2.5</sub> on cardiovascular health. Gabdrashova et. al.'s results show that, during cook time, the 33 participants were exposed to an average of 17 µg/m<sup>3</sup> of PM<sub>2.5</sub> (Gabdrashova et al.). Blood pressure (BP) and heart rate (HR) measurements were taken immediately before cooking and again after cooking. Mean systolic blood pressure (SBP), which is the pressure at which the heart ejects blood into the arteries, values were 105.0 +/- 3.2 mmHg before cooking and increased slightly to 108.0 +/- 3.1 mmHg after cooking. While both of these values are within normal SBP range of 90-120 mmHg (Sheps), this slight increase displays how rapidly the effects of PM<sub>2.5</sub> exposure can occur. However, the study also notes that this slight increase in SBP as well as a decrease in HR among participants could be due to the act of cooking itself. Other factors beyond particle exposure such as heat stress from the stove, standing and moving for a period of time, anxiety, and lack of food and drink could also be responsible for the changes in BP and HR among the participants. This study made associations to SBP, but

did not observe any significant changes in diastolic blood pressure (DBP) (Gabdrashova et al.). When SBP increases but DBP remains the same, there is a risk of developing a condition known as Isolated Systolic Hypertension (ISH). Likewise, a controlled study at Colorado State University completed by Cole-Hunter et. al. observed the impact of no more than 2 hours of stove use on cardiac function by using continuous electrocardiogram (ECG/EKG) monitoring to determine heart rate variability (HRV), which is a fluctuation in the time intervals between adjacent heart beats (Shaffer and Ginsberg). The results of this study show that not only can very short-term exposure to PM<sub>2.5</sub> trigger an increase in HRV, but the increase in HRV often occurs immediately following exposure (Cole-Hunter et al.). While low HRV is often less favorable than high HRV, rapid increases in HRV above a certain threshold is typically a sign of abnormal parasympathetic nervous system activity (the body attempting to recover from the excess stress brought on by PM<sub>2.5</sub> inhalation), which is also known as an imbalance in Cardiac Autonomic Modulation (CAM) (Shaffer and Ginsberg).

Four of the studies presented in Table 1 continuously monitored indoor air for more than 48 hours and implemented air filter interventions. Allen, Chen, Bräuner, and Li et. al.'s studies demonstrated how implementing the use of air filters such as HEPA filters and air purifiers can decrease concentrations of indoor PM<sub>2.5</sub> and, thus, benefit overall cardiovascular health. The results of Allen's study showed a 60% reduction in indoor PM<sub>2.5</sub> due to utilization of HEPA filters for seven consecutive days. Furthermore, HEPA filtration was associated with both a 9.4% increase in Reactive Hyperemia Index (RHI) as well as a 32.6% decrease in C-Reactive Protein (CRP). Both RHI and CRP are predictors of cardiovascular risk. Low RHI values are typically associated with cardiovascular risk factors such as obesity, smoking, and diabetes, and a high CRP content can be an indicator of the presence of heart disease (Allen et al.). Bräuner et. al.

observed improved macrovascular function (MVF), which is the function of any of the large (macro) blood vessels in the body such as the coronary arteries and the aorta, in study participants as a result of 48 hours of air purification. MVF was measured in this study using reactive hyperemia-peripheral arterial tonometry (RH-PAT). Results of this study showed an improved MVF of 8.1% in the cohort of 41 healthy, non-smoking, elderly individuals (Bräuner et al.). In their study, Chen et. al. observed a significant decrease in average PM<sub>2.5</sub> exposure from 48.6 µg/m<sup>3</sup> without indoor air filtration to only 8.6 µg/m<sup>3</sup> with continuous indoor air filtration (Chen et al.). Likewise, the results of Li et. al.'s study also demonstrated a significant decrease in average exposure to PM<sub>2.5</sub> from 53.1 µg/m<sup>3</sup> in the control group to 24.3 µg/m<sup>3</sup> in the purification group, further demonstrating the positive impact of air filtration on indoor PM<sub>2.5</sub> exposure (H. Li et al.).

The results of the indoor-only exposure studies in this review demonstrated significant reductions in indoor air pollution exposure as well as the health benefits associated with indoor air filtration, as well as the acute, almost instantaneous nature of the effects of only a few hours of direct exposure to PM<sub>2.5</sub> during the process of cooking. According to these studies, indoor air filtration devices, by reducing the average amount of PM<sub>2.5</sub> and other pollutants inhaled into the body, decreased the prevalence of atherosclerotic plaque and heart disease, and improved overall macrovascular function in study participants. While the kitchen studies included in this section provide valuable insight into the amount of PM<sub>2.5</sub> exposure that occurs during cooking and the possible cardiovascular health effects that can arise, they also have some limitations. For example, the cardiovascular effects such as elevated BP experienced by these participants can also be attributed to other factors

Table #1: *Sampler Placement: Indoors*

Study		Participants			Sampling Period	Average Conc. PM <sub>2.5</sub> (ug/m3)	Results & Cardiovascular Health Associations	Reference
Location	Duration	Number	Age	Exclusions				
British Columbia, Canada	Nov 2008 - Apr 2009	45	19+	Smoking	7 days	Filter off: 11.2 +/- 6.1 Filter on: 4.6 +/- 2.6 (filters reduced by 60%)	Filtration improved endothelial function and decreased inflammatory biomarkers. Filters increased RHI by 9.4% and decreased CRP by 32.6%	(Allen et al.)
Shanghai, China	Nov - Dec 2015	55	18-22	Smoking, living with smokers	2 weeks	Unpurified air: 46.8 (9.1) Purified air: 8.6 (4.0)	PM-associated effects on cardiopulmonary function persisted even at low PM <sub>2.5</sub> concentrations, especially on HRV indices.	(Chen et al.)
Not specified	Not specified	33	18-51	Smoking, drug use, respiratory disease	During cook time	17	HR reduced after cooking. Reductions significant after 90 min. Associated with increase in SBP.	(Gabdrashova et al.)
Nangong County, Hebei Province, North China	Nov 2018 - Jan 2019	752	25-80	Heart/kidney disease, stroke, diabetes	24 hours	Coal-burning: 87.0 (77-112) Control: 17.5 (12-15)	Long-term exposure to coal combustion increases risk of atherosclerosis and inflammation	(Pang et al.)
Copenhagen, Denmark	Not specified	41	60-75	Smoking	48 hours	Filter off: 12.6 (11.2-14.1) Filter on: 4.7 (3.9-5.7)	Reduction by filtration improved macrovascular function	(Bräuner et al.)
Puno, Peru	Feb - Dec 2011	519	35+	None	24 hours	Biomass fuel: 178 Clean fuel: 27	Found no associations between exposure to biomass fuel smoke and right ventricular pressure/volume overload nor pulmonary hypertension.	(Caravedo, Painschab, et al.)
Shanghai, China	Nov - Dec 2015	55	18-22	Smoking; allergic, respiratory, cardiac disease	48 hours	Real purification: 24.3 Sham purification: 53.1	Higher PM <sub>2.5</sub> induces activations of the hypothalamus-pituitary-adrenal and sympathetic-adrenal-medullary axes, adding mechanistic insights into the health outcomes of PM <sub>2.5</sub> exposure.	(H. Li et al.)
Fort Collins, CO (Colorado State University)	Oct 2016 - Jan 2018	48	20-36	Smoking	2 hours	Control: 0; Petroleum: 10; Gasifier: 35; Fan Rocket: 100; Rocket elbow: 250; Three-stone fire: 500	An increase in HRV can occur immediately following exposure to low or "improved" levels of cookstove-emitted PM <sub>2.5</sub> among healthy adults	(Cole-Hunter et al.)
Beijing, China	Nov 2015 - May 2016	43	58-81	Respiratory/cardiac disease, lung cancer	48 hours	58.01 +/- 52.82	Indoor PM <sub>2.5</sub> is associated with altered cardiac function in COPD patients.	(Pan et al.)
36 Chinese cities	Nov 2015 - May 2016	18,484	n/a	None	24 hours	Nationwide average: 40	Higher mortality risk attributed to indoor and ambient PM <sub>2.5</sub> exposure compared with ambient exposure alone	(Dong et al.)

*Abbreviations:* PM = Particulate Matter, PM<sub>2.5</sub> = Fine Particulate Matter, CVD = Cardiovascular Disease, HR = Heart Rate, SBP = Systolic Blood Pressure, COPD = Chronic Obstructive Pulmonary Disease, RHI = Reactive Hyperemia Index, CRP = C-Reactive Protein (Test for Heart Disease).



### **3.2 Under 24 Hour Personal PM<sub>2.5</sub> Exposure Associations with Cardiovascular Health**

With 14 total, studies focused on personal PM<sub>2.5</sub> monitoring for less than 24 hours comprise the majority of this review. As mentioned previously in Section 1.3, personal air pollution monitoring consists of the sampler being worn on the body of the study participant (i.e. attached to clothing near the breathing zone or on the outside of a backpack) for a set period of time in order to obtain an accurate, holistic measurement of the amount of PM<sub>2.5</sub> that individual is likely exposed to while completing the tasks of day-to-day life. These studies were also conducted in both rural and urban areas across the globe. Rural areas included multiple Guatemalan villages as well as Puno, Peru, and urban areas include Mexico City, Mexico; Beijing, China; and Detroit, Michigan. These studies, which analyzed the cardiovascular health consequences associated with 24 hours or fewer of personal exposure to PM<sub>2.5</sub>, are presented in *Table 2.1*.

Both studies by McCracken et. al. as well as the study by Huang et. al. focused solely on female participants, particularly healthy, adult (over the age of 25) females who served as the primary cook in their household during the time of the study. McCracken et. al. conducted two separate studies in several villages in Guatemala, particularly agricultural, indigenous populations who primarily use open fires within the home for cooking. In both studies, an improved wood stove called a *plancha* was introduced to half of the participants, while the other half continued with the use of open fires. Plancha cookstoves include a chimney component which ventilates particle emissions from the kitchen and, thus, reduces the overall amount of particulate matter that the user is exposed to. Researchers observed a more than 50% reduction in average PM<sub>2.5</sub> concentration from 264 µg/m<sup>3</sup> in the open fire group to 102 µg/m<sup>3</sup> in the intervention group (McCracken, Smith, D, et al.). Similarly, in the second study by McCracken, improved

cookstove intervention reduced PM<sub>2.5</sub> concentrations from an average of 266 µg/m<sup>3</sup> in the open fire group to an average of 102 µg/m<sup>3</sup> in the intervention group (McCracken, Smith, Stone, et al.). The results of the first study observed reductions in SBP by 3.7 mmHg and DBP by 3.0 mmHg in women who used the improved cookstove and concluded that the chimney stove intervention reduces overall BP (McCracken, Smith, D, et al.). Likewise, the second study presented by McCracken et. al. concluded that the improved cookstove intervention also reduced the occurrence of nonspecific ST-segment depression in EKG readings, suggesting that excess exposure to woodsmoke affects ventricular repolarization, thus affecting overall cardiac function (McCracken, Smith, Stone, et al.). Furthermore, the study by Huang et. al., which took place in the metropolitan area of Taipei, Taiwan, associated PM<sub>2.5</sub> exposures with decreased SDNN (Standard Deviation of N-N intervals) as well as decreased r-MSSD (Root mean square of successive R-R interval differences), which are both indicative of changes in HRV, among housewives, particularly following activities such as stir-frying food, cleaning with detergent, and burning incense (Huang et al.).

Studies by Li, Brook, Cárdenas, and Riojas-Rodríguez et. al. associated personal exposure to air pollution specifically in urban/metropolitan areas with cardiovascular health consequences. Li et. al.'s study, located in Beijing, China, continuously monitored personal exposure to PM<sub>2.5</sub> as well as black carbon (BC), which is a component of PM<sub>2.5</sub> commonly referred to as soot, and determined their combined effects on HRV indices. Both HRV and HR were monitored using 24-hour electrocardiography (ECG/EKG). The personal exposure levels were listed as 28.05 µg/m<sup>3</sup> of PM<sub>2.5</sub> and 1.80 µg/m<sup>3</sup> of BC (L. Li et al.). The conclusions of this study as well as Cardenas et. al.'s study in Mexico City, Mexico further demonstrated the drastic effects of PM<sub>2.5</sub> exposures in urban areas on HRV. Furthermore, Hampel's study consisting of 5

participants in Augsburg, Germany observed very rapid changes in HRV within only 30 minutes of PM<sub>2.5</sub> exposure, demonstrating how quickly the negative effects of exposure on HRV are experienced (Hampel et al.). A second study completed in Mexico City, Mexico narrowed down the participant pool by only including individuals who had been diagnosed with ischemic heart disease for more than six months. In this high-risk population, researchers observed a diminished HRV response in conjunction with increasing exposure to PM<sub>2.5</sub> (Riojas-Rodríguez et al.). A low HRV can indicate domination of the sympathetic nervous system and can increase vulnerability to stress and susceptibility to disease (Shaffer and Ginsberg). Folino et. al. demonstrated a similar autonomic nervous system dysregulation in patients who have had myocardial infarction (MI), which is the lack of adequate blood flow to the heart muscle commonly referred to as a heart attack. They also documented more severe arrhythmias in patients who had the highest average personal PM<sub>2.5</sub> exposures (Folino et al.).

Two studies were completed in both the rural and urban areas of Puno, Peru in order to make an accurate comparison of the average personal PM<sub>2.5</sub> exposure levels in each area. In both of these studies, the participants labeled “rural” had a long-term history of exposure to biomass fuels, while the participants labeled “urban” had a long-term history of exposure to clean fuels due to greater accessibility of safer methods of heating and cooking within the home in more developed areas. Painschab’s study consisted of 276 total participants, and Caravedo et. al.’s consisted of 456 participants. The results of both studies document a less than 25 µg/m<sup>3</sup> of personal PM<sub>2.5</sub> exposure for the urban participant group and a greater than 250 µg/m<sup>3</sup> of personal PM<sub>2.5</sub> exposure for the rural participant group. According to Painschab’s results, the significantly increased exposure to PM<sub>2.5</sub> in Puno’s rural communities compared with the urban areas was associated with a number of cardiovascular health effects. First, mean CIMT measurements were

significantly higher in the rural group than in the urban group, and atherosclerotic plaques in the carotid artery were present in 26% of the rural participants and only 14% of the urban participants. The study also mentions that the level of atherosclerotic plaque in the carotid arteries of these participants was similar in magnitude to people who regularly smoke tobacco. Additionally, the rural group displayed higher average systolic and diastolic blood pressures than the urban group, with the average BP in the rural group measuring 118/75 (SBP/DBP) compared to 111/71 in the urban group (Painschab et al.). Caravedo et. al. expanded on the results of Painschab's study by analyzing the prevalence of biomarkers specifically associated with presence of endothelial inflammation in urban vs. rural participants in Puno. The results of this show that levels of the biomarkers ICAM-1 and VCAM-1 (Intercellular Adhesion Molecule 1 and Vascular Cell Adhesion Molecule 1), which are indicative of inflammatory disease, were consistently elevated in rural participants compared to urban clean fuel-users. The levels of ICAM-1 are listed as 330.9 ng/mL in the rural group and 302.3 ng/mL in the urban group and the levels of VCAM-1 were 403.3 ng/mL in the rural group and 361.8 ng/mL in the urban group (Caravedo, Herrera, et al.).

A study completed in various locations across China showed associations between the carbon components of PM<sub>2.5</sub> and atherosclerosis. Fan et. al. measured personal exposure to PM<sub>2.5</sub> and included the amounts total carbon, organic carbon, and elemental carbon components of PM<sub>2.5</sub> in their analyses. The average amount of personal PM<sub>2.5</sub> exposure in this study is listed as 164.75 µg/m<sup>3</sup>, consisting of 53.86 µg/m<sup>3</sup> total carbon, 44.93 µg/m<sup>3</sup> organic carbon, and 9.46 µg/m<sup>3</sup> elemental carbon. Their analysis showed significant positive correlations between total carbon, organic carbon, and elemental carbon and atherogenic index of plasma (AIP), which is a measurement of the presence of atherosclerosis (Fan et al.).

Two studies conducted in the United States made associations between personal PM<sub>2.5</sub> exposure and elevated HR. First, a study in Harrisburg, Pennsylvania concluded that increases in concentration of PM<sub>2.5</sub> were significantly associated with elevations in HR, as well as HRV implications. These cardiovascular effects suggest that increased personal PM<sub>2.5</sub> exposures can result in imbalanced CAM, which is typically characterized by decreased parasympathetic nervous system modulation and increased sympathetic nervous system modulation. Furthermore, the results of He et. al.'s study suggests that the effects on CAM are acute and occur within 1-6 hours of exposure (He et al.). A second study completed in six neighborhoods in Detroit, Michigan also explored the instantaneous cardiovascular effects of personal PM<sub>2.5</sub> exposure on HR, among other cardiovascular health endpoints. While PM<sub>2.5</sub> was personally monitored for 24 hours 5 separate times within each participating Detroit home, the exposure data was not included in this study. Cardiovascular examinations were conducted within each participant's home following at the end of each 24-hour period where BP, HR, brachial artery diameter (BAD), and endothelial function were measured. The findings of this study suggest that slight increases in PM<sub>2.5</sub> exposure result in small increases in HR as well as slight trends toward endothelial inflammation (Brook, Shin, et al.).

The personal exposure studies where PM<sub>2.5</sub> was monitored for 24 hours or less made associations between fine particulate matter and a multitude of cardiovascular health implications including elevated BP, changes in HR and HRV, autonomic nervous system dysregulation, MI, and arrhythmias. Additionally, these studies demonstrate the catastrophic effect of PM<sub>2.5</sub> in rural areas with limited access to resources that reduce exposure. Because individuals living in rural areas experience drastically higher levels of indoor air pollution exposure, they are more likely to have higher BP, CIMT, presence of atherosclerotic plaque, and

presence of inflammatory disease. Furthermore, participants in rural areas using improved cookstoves instead of open fires for cooking experienced improved BP, improved ventricular repolarization (cardiac function), and normalized HRV indices.

Table 2.1: Sampler Placement: Personal for 24 or Fewer Hours

Study		Participants				Average Conc. PM <sub>2.5</sub> (ug/m <sup>3</sup> )	Results & Cardiovascular Health Associations	Reference
Location	Duration	Age	Number	Sex	Exclusions			
23 villages in San Marcos, Guatemala	2003 - 2005	38+	Chimney intervention - 49 open fire - 71	F	None	Control: 264 Intervention: 102	SBP and DBP were lower among women who cooked with the stove instead of open fire; chimney stove intervention reduces BP	( <a href="#">McCracken, Smith, D, et al.</a> )
Puno, Peru	Not specified	35+	Clean fuel - 122 Biomass fuel - 154	B	None	Rural: >250 Urban: <25	Exposure to biomass fuels is associated with increased CIMT and higher prevalence of carotid artery atherosclerotic plaque and higher BP	( <a href="#">Painschab et al.</a> )
Puno, Peru	Not specified	35+	Clean fuel - 228 Biomass fuel - 228	B	None	Rural: >250 Urban: <25	Specific biomarkers related to endothelial inflammation were positively associated with exposure to biomass fuels	( <a href="#">Caravedo, Herrera, et al.</a> )
Beijing, China	Dec 2017 - Jun 2018	18-49	97	B	Smoking, drinking alcohol, cardiopulmonary dysfunction, hypertension, diabetes	28.05 (SD 31.98)	Short-term PM <sub>2.5</sub> exposure associated with HRV. Obese adults showed greater HRV changes than normal-weight adults	( <a href="#">L. Li et al.</a> )
China	Dec, 2011	60+	112	B	CVD	164.75 +/- 110.67	The carbon components of PM <sub>2.5</sub> are risk factors of atherogenic index of plasma	( <a href="#">Fan et al.</a> )
6 neighborhoods in Detroit, Michigan	3 years	18+	51	B	Smoking	Not included	Increases in PM <sub>2.5</sub> exposure during daily activity associated with small increases in HR and trends toward endothelial cell dysfunction.	( <a href="#">Brook, Shin, et al.</a> )
Taipei, Taiwan	Feb - Jun, 2014	20-35	72	B	Smoking, disease	37.3	PM <sub>2.5</sub> associated with increases in N7-MeG and 8-oxodG indicative of systemic inflammation and oxidative stress.	( <a href="#">Lai et al.</a> )
Augsburg, Germany	Feb-Mar, 2008	Avg. 46	5	B	None	13.2 (SD 36.8)	Very rapid changes in HRV observed within 30 min of PM <sub>2.5</sub> exposure	( <a href="#">Hampel et al.</a> )
Padua, Italy	June 2006 - May 2007	Avg. 63	39	B	Individuals who have not been diagnosed with MI for more than 6 months.	Summer: 33.9 +/- 12.7 (SD 39) Winter: 62.1 +/- 27.9 (SD 36) Spring: 30.8 +/- 14 (SD 30)	Exposure to PM <sub>2.5</sub> associated with autonomic dysregulation in patients with MI. More severe arrhythmias occurred at highest exposures. Decrease in HRV.	( <a href="#">Folino et al.</a> )
Harrisburg, PA	Nov 2007 - June 2009	45+	106	B	Smoking, severe cardiac problems	13.66 (SD 11.77)	Increase in PM <sub>2.5</sub> concentration significantly associated with elevated HR	( <a href="#">He et al.</a> )

Guatemala (agricultural, indigenous population)	Oct 2002 - Nov 2004	38-84	Intervention - 49 Control - 70	F	None	Control: 266 Intervention: 102	Stove intervention reduced occurrence of ST- segment depression, suggesting that wood smoke exposures affect ventricular repolarization	( <a href="#">McCracken, Smith, Stone, et al.</a> )
Taipei (metro area)	2010 - 2012	25-64	50	F	Non-housewives, individuals who are not the primary cook of their household	23.5 (SD 19.4)	Indoor PM <sub>2.5</sub> exposures associated with decreased SDNN and RMSSD among housewives	( <a href="#">Huang et al.</a> )
Mexico City, Mexico	Not specified	20-40	52	B	Cardiovascular, pulmonary, neurological, or endocrine disease. Taking medication.	10.8	Increase of PM <sub>2.5</sub> air pollution associated with HRV	( <a href="#">Cárdenas et al.</a> )
Mexico City, Mexico	Dec 2001 - Apr 2002	25-75	30	B	Individuals who have not been diagnosed with ischemic heart disease for more than 6 months, diabetics, smokers	46.8	For this high-risk population, alteration of cardiac autonomic regulation was significantly associated with personal PM <sub>2.5</sub> exposures.	( <a href="#">Riojas- Rodríguez et al.</a> )

*Abbreviations:* M = Male, F = Female, B = Both (Male and Female), PM<sub>2.5</sub> = Fine Particulate Matter, CVD = Cardiovascular Disease, MI = Myocardial Infarction, SD = Standard Deviation, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, BP = Blood Pressure, CIMT = Carotid Intima-Media Thickness, HRV = Heart Rate Variability, HR = Heart Rate, SDNN = Standard Deviation of N-N Intervals, RMSSD = Root Mean Square of Successive Differences between heart beats, N7-MeG = Methylguanine, 8-oxodG = Deoxyguanosine.



### 3.3 Over 24 Hour Personal PM<sub>2.5</sub> Exposure Associations with Cardiovascular Health

The eight total studies which contained research in which personal PM<sub>2.5</sub> exposures were continuously measured for more than 24 hours are presented in *Table 2.2*. Of these, two studies were conducted in Chinese cities (i.e. Guangzhou, Weinan, and Beijing), two were conducted in Boston, two in Taiwan, one in Denmark, and one in Poland. Five of the eight studies excluded smokers and individuals who lived with smokers. In this section, personal PM<sub>2.5</sub> monitoring durations ranged from 48 hours to 5 consecutive days. While having participants wear personal monitors for long periods of time provides a more accurate measurement of the amount of PM<sub>2.5</sub> most people are likely exposed to while carrying out the normal activities of daily life, a limitation of this review is that there are no studies currently available in which long-term (months to years) personal exposure measurements are taken.

First, two studies completed in Boston, Massachusetts by Lee et. al. provide more evidence for the implications of fine particulate matter inhalation on HRV indices. After a year-long study involving measurements of markers of systemic inflammation and oxidative stress (i.e. blood and urine samples), researchers were able to conclude that individuals with excessive oxidative damage and systemic inflammation are more susceptible to dangerous cardiovascular effects associated with exposure to PM<sub>2.5</sub>. Significantly stronger effects on both HRV and HR were observed in study participants who expressed elevated levels of biomarkers of oxidative stress and systemic inflammation, further suggesting negative impacts on CAM (Lee, Eum, Fang, et al.). Oxidative stress is defined as an imbalance between the number of free radicals and the amount of antioxidants within the body, which often leads to cell and tissue damage. Oxidative stress is a process that occurs naturally in the body and contributes to aging, but outside sources such as excess exposure to environmental radiation and pollutants can both

enhance the effects of oxidative stress and cause them to appear earlier in life (Eske). A second more specialized study which excluded participants with overt heart disease was undergone by the same team of researchers during the same year. The results of this study suggest that the effects of PM<sub>2.5</sub> on CAM occur almost immediately following exposure. Lee et. al. observed rapid, significant changes in HRV within 2.5 hours of exposure to more than 12 µg/m<sup>3</sup> of PM<sub>2.5</sub>, with the most significant changes recorded at the 25-minute mark. They also noted that the changes in daytime HRV were relatively insignificant in comparison to changes in nocturnal HRV (Lee, Eum, Rodrigues, et al.). Similarly, the results of a study in Taiwan also supports that the impacts in HRV and overall CAM occur very rapidly following exposure. HRV increased immediately following exposure to an average concentration of 12.6 µg/m<sup>3</sup> of PM<sub>2.5</sub> with impacts lasting as long as 4.5 to 5 hours (Lung et al.). A second study conducted in northern Taiwan excluded both smokers and individuals with any history of cardiovascular disease (CVD). Tsou and colleagues observed increased HRV and HR following exposure to an average concentration of 13.7 µg/m<sup>3</sup> of PM<sub>2.5</sub>, suggesting that adverse cardiovascular effects can occur among healthy adults with no history of smoking or CVD (Tsou et al.).

Both the studies by Zhao et. al. and M. Liu et. al. in different Chinese cities also associated personal exposure to fine particulate matter with notable increases in both systolic and diastolic blood pressure. M. Liu and colleagues, however, also made associations between short-term exposure to PM<sub>2.5</sub> and mean arterial pressure (MAP), prehypertension, and hypertension (high BP) in children aged 4-12 years. Evidence has shown that slightly elevated BP in childhood is more likely to facilitate the development of hypertension in adulthood. According to the results of this study, the highest elevation in SBP measured 0.27%, the highest elevation in

DBP measured 0.57%, and the highest elevation in MAP measured 0.45% for every 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$  (M. Liu et al.).

Researchers in Copenhagen, Denmark studied the effects of personal exposure to fine particulate matter on the blood compartment. Sørensen and colleagues accomplished this by measuring red blood cell (RBC) and platelet counts in the 50 male and female participants. The results of this study show positive associations between personal exposure to  $\text{PM}_{2.5}$  and RBC concentrations in women, but there was no significant relationship for the male participants. This evidence suggests that  $\text{PM}_{2.5}$  does have an effect on the blood compartment, and this may be involved in the atherosclerotic process due to the fact that increased RBC concentration in the blood can significantly affect blood viscosity (i.e. blood's ability to flow in blood vessels) (Sørensen et al.).

Finally, a study in Krakow, Poland studied the effects of personal  $\text{PM}_{2.5}$  exposure on Krakow residents in their second trimester of pregnancy. The measured 48-hour average for  $\text{PM}_{2.5}$  personal exposure was 33.6 (standard deviation 32.135)  $\mu\text{g}/\text{m}^3$ . Researchers observed an average increase in SBP by 6.1 mmHg (this measurement was even higher for women with excessive gestational weight gain). Associations to DBP were also made, but they were not as significant (Jedrychowski et al.). This study concluded that exposure to fine particulate matter during pregnancy can be associated with increased maternal BP, demonstrating an associated prohypertensive effect. Gestational hypertension can potentially lead to many maternal morbidities such as preeclampsia (a pregnancy complication characterized by high BP and organ system damage), cesarean delivery, abruptio placentae (abruption of the placenta), and renal dysfunction, as well as perinatal morbidities such as preterm birth and restriction of fetal growth (Jedrychowski et al.).

Personal exposure measurements lasting longer than 24 hours provide even stronger evidence for the cardiovascular impacts of PM<sub>2.5</sub> exposure. In this section, various researchers associated PM<sub>2.5</sub> with significant changes in HRV, increased oxidative stress and systemic inflammation, increased overall BP and maternal BP, and decreased blood viscosity. Additionally, one study consisting of young children associated childhood exposures to PM<sub>2.5</sub> with increased MAP, prehypertension, and hypertension.

Table 2.2: Sampler Placement: Personal for More Than 24 hours

Study		Participants				Average Conc. PM <sub>2.5</sub> (ug/m <sup>3</sup> )	Results & Cardiovascular Health Associations	Reference
Location	Duration	Age	Number	Sex	Exclusions			
Guangzhou (South China) & Weinan (North China)	G: Oct 2018 - May 2019. W: Jun 2019 - Dec 2019	4-12	268	B	None	Range: 32.95-35.34	Short-term PM <sub>2.5</sub> exposure is associated with increased SBP, DBP, MAP, and prevalence of prehypertension and hypertension in children.	(M. Liu et al.)
Northern Taiwan	Fall 2018 & Spring 2019	40-75	35	B	Smokers, history of CVD	13.7 +/- 11.4	Adverse associations between PM <sub>2.5</sub> and increased HRV and HR among healthy adults.	(Tsou et al.)
Boston, MA	Mar - Aug, 2004	Avg. 44	21	B	Overt heart disease	Low: <12 Medium: 12-24 High: >= 24	Rapid changes in nocturnal HRV occurred within 2.5 hours of exposure, suggesting immediate cardiac autonomic effects	(Lee, Eum, Rodrigues, et al.)
Boston, MA	2004	Avg. 44	21	B	None	29.8 +/- 77.7	Individuals with excessive oxidative damage and systemic inflammation are more susceptible to adverse cardiac effects of particulate air pollution	(Lee, Eum, Fang, et al.)
Taiwan	Not specified	20-65	36	B	Smoking	12.6 +/- 8.9	HRV increased after exposure to PM <sub>2.5</sub> with impacts lasting for 4.5-5 hrs.	(Lung et al.)
Beijing, China	Feb - Jul 2012	35-75	65	B	Smoking	64.2 (SD 36.9)	Exposure to PM <sub>2.5</sub> and BC associated with increases in SBP and DBP, as well as an increase in HRV	(Zhao et al.)
Copenhagen, Denmark	Nov 1999 - Aug 2000	20-33	50	B	Smoking	16.1	Particulate air pollution has measurable effects in the blood compartment, causing an increased RBC count, affecting blood viscosity and contributing to atherosclerosis	(Sørensen et al.)
Krakow, Poland	Not specified	18-35	431	F	Women not in the 2nd trimester of pregnancy, smoking, drug use, HIV, chronic disease.	33.6 (SD 32.135)	Exposure to PM <sub>2.5</sub> during pregnancy associated with increased maternal BP	(Jędrychowski et al.)

Abbreviations: M = Male, F = Female, B = Both (Male and Female), PM<sub>2.5</sub> = Fine Particulate Matter, CVD = Cardiovascular Disease, HIV = Human Immunodeficiency Virus, SD = Standard Deviation, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, BP = Blood Pressure, MAP = Mean Arterial Pressure, HRV = Heart Rate Variability, BC = Black Carbon, RBC = Red Blood Cell

### 3.4 Simultaneous Indoor and Personal Sampling Studies

Studies in which both indoor and personal PM<sub>2.5</sub> exposure measurements were taken simultaneously are presented in *Table 3*. Gathering both indoor and personal PM<sub>2.5</sub> exposure data from the same participants in the same location allows researchers to draw valuable conclusions regarding how much of the total PM<sub>2.5</sub> individuals are exposed to in daily life is associated with indoor sources. Simultaneous indoor and personal sampling studies consist of five total studies. All but one of these studies excluded smokers and those living with smokers from participating. These studies were also conducted in both rural and urban areas across the globe, including Honduras, Taipei, The Netherlands, Finland, and The United States. However, the study conducted in The Netherlands and Finland did not make any significant associations to cardiovascular health (de Hartog et al.). Particle measurement durations in this section range from 24 hours to 10 days.

Two studies in this group implemented filter interventions and compared the PM<sub>2.5</sub> concentrations with and without air filtration. First, a study conducted in Detroit, Michigan by Morishita et. al. compared exposure concentrations after 72 hours of “sham” filtration (the filter is in the room, but it is turned off), low-efficiency (LE) filtration, and high-efficiency (HE) filtration. With sham filtration, the average concentration of indoor PM<sub>2.5</sub> measured 15.5 µg/m<sup>3</sup>. LE filtration for 72 hours reduced exposure to 10.9 µg/m<sup>3</sup>, and HE filtration for the same amount of time further reduced exposure to 7.4 µg/m<sup>3</sup> (Morishita et al.). A similar study conducted in Taipei documented the amount of PM<sub>2.5</sub> in filtered vs. unfiltered air. The 24-hour average concentration of PM<sub>2.5</sub> before filtration was listed as 21.4 µg/m<sup>3</sup>, and this average was reduced to 12.8 µg/m<sup>3</sup> after filtration (Chuang et al.). In their study, Morishita et. al. observed the effects of air filtration on brachial blood pressure in senior citizens living in a low-income residential

home. They were able to conclude that any level of filtration for three days resulted in reduced diastolic and systolic blood pressures in senior citizens. According to their results, both low-efficiency and high-efficiency filtration for three days yielded an average decrease of 3.2 mmHg in brachial SBP and an average decrease of 1.5 mmHg in brachial DBP. These results show that any level of air filtration could potentially be cardioprotective against personal exposures to fine particulate matter (Morishita et al.). Furthermore, the results of the Taipei study by Chuang et al. likewise demonstrated higher SBP and DBP in participants with sham filtration. Additionally, sham filtration participants displayed higher levels of biological markers indicative of systemic inflammation and oxidative stress (i.e. 8-oxodG (deoxyguanosine) and N7-MeG (methylguanine)). This study also demonstrates the potential cardiovascular health benefits associated with implementing long-term air filtration devices within the home (Chuang et al.).

Researchers in Honduras conducted an indoor and personal exposure study involving only non-pregnant, non-smoking women aged 25 to 56 who serve as the primary cooks in their household. This study implemented the use of a stove intervention in order to gauge how much PM<sub>2.5</sub> women are exposed to when using traditional open-fire stoves for cooking. This study utilized *Justa* stoves, which were designed to release one-third of the PM<sub>2.5</sub> typically emitted by a traditional open three-stone fire. Personal PM<sub>2.5</sub> was measured by having each woman wear a device for 24 hours, while indoor measurements were taken simultaneously using a stationary monitor located in the kitchen near the stove. For the traditional open-fire stove, the average personal exposure was documented as 126 µg/m<sup>3</sup> and indoor exposure was documented as 360 µg/m<sup>3</sup>. The *Justa* stove significantly improved these exposure results, reducing the average personal exposure to 66 µg/m<sup>3</sup> and the average indoor exposure to 137 µg/m<sup>3</sup> (Young et al.). While the exposure averages after the stove intervention are significantly lower than the

exposure associated with open-fire cookstoves, these improved measurements are still at a level that poses a severe threat to human health. This study further demonstrates how much more significantly rural, underdeveloped areas are negatively impacted by air pollution.

Finally, a study conducted in Seattle, Washington by L. Liu and colleagues focused on the average indoor and personal PM<sub>2.5</sub> exposures of vulnerable populations in Seattle. The participants in this study consisted of 34 elderly patients diagnosed with Chronic Obstructive Pulmonary Disease (COPD), 27 elderly patients diagnosed with Coronary Heart Disease (CHD), 28 elderly healthy individuals (i.e. no signs or symptoms of cardiorespiratory disease), and 19 children (aged 6 to 13) diagnosed with asthma. COPD, CHD, and asthma are all significantly associated with vulnerability to the harmful effects of exposure to fine particulate matter. The results of this study showed that the children with asthma experienced the highest level of personal exposure and the second highest level of indoor exposure (by only 0.3 µg/m<sup>3</sup>) (L.-J. S. Liu et al.). The results of this study suggest that children and teenagers are likely exposed to higher levels of PM<sub>2.5</sub> than adults.

Studies involving both indoor and personal exposure monitoring provide the most holistic measurement of PM<sub>2.5</sub> exposure because monitoring both allows for determination of how much of the total PM<sub>2.5</sub> an individual is exposed to during a given time is from indoor sources. A limitation of this review is that a very small percentage of the studies included were simultaneous indoor and personal monitoring studies. The studies included in this section observed vulnerable populations such as the elderly, people living in rural communities, and individuals with diseases such as COPD, CHD, and asthma. Both stove intervention and air filtration intervention studies saw significant reductions in indoor PM<sub>2.5</sub> exposure as well as



improved cardiovascular health (i.e. improved BP, lower prevalence of inflammation and oxidative stress).

Table 3: Sampler Placement: Both Indoor and Personal

Study		Participants				Sampling Period	Average Conc. PM <sub>2.5</sub> (ug/m3)	Results & Cardiovascular Health Associations	Reference
Location	Duration	Age	Number	Sex	Exclusions				
Detroit, MI	Oct 2014 - Nov 2016	Avg. 67	40	B	Smoking	72 hr per filtration type	Sham filtration: 15.5 (SD 10.9) LE Filtration: 10.9 (SD 7.4) HE Filtration: 7.4 (SD 3.3)	Any filtration for 3 days decreased brachial SBP and DBP. The use of portable air filtration systems is potentially cardioprotective against exposure to PM <sub>2.5</sub>	(Morishita et al.)
Honduras	Not specified	25-56	147	F	Smoking, Pregnant	24 hr	Traditional stove: Personal 126, Kitchen 360 Improved Stove: Personal 66, Kitchen 137	Women with traditional stoves had a 2.8 mmHg higher SBP than women with improved stoves.	(Young et al.)
Taipei	2013-2014	30-65	200 (100 air filtration, 100 control)	B	History of smoking and related CVDs: arrhythmia, hypertension, diabetes, coronary artery disease	24 hr (12 times)	Filtered air: 12.8 +/- 7.4 Unfiltered air: 21.4 +/- 14.5	Air pollution exposure is associated with elevated BP, systemic inflammation, and oxidative stress.	(Chuang et al.)
Amsterdam, The Netherlands & Helsinki, Finland	6-8 months in each location	50	Amsterdam: 37 Helsinki: 47	B	Smoking, individuals without physician-diagnosed CHD	24 hr prior to clinic visit; visits biweekly for 6-8 weeks.	Amsterdam: Indoor 14.9, Personal 15.3 Helsinki: Indoor 10.2, Personal 10.0:	Because personal and indoor measurements were only taken during the 24 hr preceding the clinic visit, no significant associations to HRV were made.	(De Hartog et al.)
Seattle, WA	Oct 1999 - May 2001	Elderly: >65 Children: 6-13	108: 34 with COPD, 27 with CHD, 28 healthy (all elderly), and 19 children with asthma	B	None	10 consecutive days	PERSONAL: COPD 10.5, CHD 10.8, Healthy 9.3, Asthmatic 13.3 INDOOR: COPD 8.5, CHD 9.5, Healthy 7.4, Asthmatic 9.2	Average personal PM <sub>2.5</sub> exposures among sensitive subpopulations in Seattle were similar to mean outdoor PM <sub>2.5</sub> concentration.	(L.-J. S. Liu et al.)

Abbreviations: M = Male, F = Female, B = Both (Male & Female), PM<sub>2.5</sub> = Fine Particulate Matter, CVD = Cardiovascular Disease, CHD = Coronary Heart Disease, SD = Standard Deviation, COPD = Chronic Obstructive Pulmonary Disease, SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure, BP = Blood Pressure, mmHG = Millimeters of Mercury, HRV = Heart Rate Variability

## 4 Discussion

For this project, articles were reviewed that associated exposure to fine particulate matter with one or more cardiovascular health endpoints. The majority of existing research involving the impacts of PM<sub>2.5</sub> on human health focus primarily on respiratory diseases and dysfunctions with limited research regarding the associated cardiovascular implications. Additionally, studies involving PM<sub>2.5</sub> exposure monitoring typically prioritize ambient (outdoor) air pollution exposures. Very few studies take indoor exposures into account despite the many indoor sources of particulate matter (i.e. cooking, burning candles/incense, etc.). The aim of our study was to analyze research articles that assessed cardiovascular health following some amount of exposure to particulate matter. The results of this study suggest that more research focused on the effects of PM<sub>2.5</sub> exposure on the cardiovascular system could provide a better understanding of how air pollution specifically impacts cardiovascular morbidity and mortality. Direct positive associations of PM<sub>2.5</sub> exposures with cardiovascular health outcomes were found in 89% of the studies included in our final review.

Of the 4 studies that did not directly correlate PM<sub>2.5</sub> with negative impacts on cardiovascular health, only 2 did not find any significant associations. The remaining 2 focused primarily on providing mechanistic insights into how the components of PM<sub>2.5</sub> impact cardiovascular function and emphasized the importance of including indoor air pollution in research rather than focusing solely on ambient exposures. Li et. al.'s study, for example, provides mechanistic insights into the health outcomes associated with exposure to PM<sub>2.5</sub>. This study suggests that the human central nervous system (CNS) responds to changes in PM exposure, but has many limitations and requires more research in order to draw any significant conclusions. For example, researchers failed to draw blood samples upon participant enrollment

into the study, thus making it impossible to determine and eliminate normal variations in CNS activity. Additionally, size-fractionated PM data was not collected, so researchers were unable to attribute their findings solely to PM<sub>2.5</sub> (H. Li et al.).

#### **4.1 Exposure Levels in Low- & Middle- Income Countries**

Of the 37 studies included in this review, 7 took place in low- to middle-income countries, particularly within agricultural, indigenous populations. All seven studies involved either an improved cookstove intervention or indoor heating system intervention. Of these seven, two were direct comparisons of the PM<sub>2.5</sub> exposure differences between the rural and urban areas of Puno, Peru. Almost 3 billion people across the globe rely on burning of biomass fuels within the home for cooking and heating (Ezzati). Activities such as cooking with open flames and burning biomass fuels within the home without proper ventilation causes immense amounts of exposure to PM<sub>2.5</sub> and other dangerous air pollutants such as black carbon (BC), a main component of PM<sub>2.5</sub> that is linked to a multitude of human health complications such as respiratory disease, cardiovascular disease, various cancers, and birth defects (US EPA, *Black Carbon Research and Future Strategies*).

The three cookstove intervention studies, which solely focused on female participants who identified as the primary cook of the family, each demonstrated how effectively an improved stove for cooking within the home can limit PM<sub>2.5</sub> exposures in rural areas as well as how drastically the indoor pollutants experienced by women in these areas impact their health. Two studies discussed in Section 3.2, monitored personal PM<sub>2.5</sub> exposures for 24 hours or less, each study took place in indigenous villages across Guatemala. Without the use of improved stoves for cooking provided by the researchers, the participants in these two studies were exposed to a combined average of 265 µg/m<sup>3</sup> of PM<sub>2.5</sub> during the sampling period (McCracken et

al.). The third study of this nature, discussed previously in Section 3.4, took place in Honduras and measured both indoor and personal exposures for 24 hours. With traditional stove use (i.e. open fires), the 24-hour personal exposure average is listed as 126  $\mu\text{g}/\text{m}^3$ , and the average indoor  $\text{PM}_{2.5}$  concentration in the kitchen is listed as 360  $\mu\text{g}/\text{m}^3$  (Young et al.). The Indoor Air Hygiene Institute considers fine particulate matter exposure levels to be dangerous to human health after they reach more than 35  $\mu\text{g}/\text{m}^3$  in a 24-hour period. Further, long-term exposure to  $\text{PM}_{2.5}$  levels above 50  $\mu\text{g}/\text{m}^3$  can lead to serious respiratory and cardiovascular complications and premature mortality (Indoor Air Hygiene Institute). The ambient  $\text{PM}_{2.5}$  guidelines established by the World Health Organization are even lower, recommending an annual mean exposure of 5  $\mu\text{g}/\text{m}^3$  and a 24-hour mean exposure of 15  $\mu\text{g}/\text{m}^3$  (WHO, *Ambient (Outdoor) Air Pollution*). According to the WHO, burning wood, dung, or coal in indoor stoves is responsible for nearly 2 million deaths worldwide (US EPA, *Black Carbon Research and Future Strategies*). Statistics like this emphasize the importance of furthering indoor air pollution research.

Furthermore, two of the seven low- and middle-income intervention studies examined the impact of using coal and biomass fuel-burning methods for indoor heating on exposure to  $\text{PM}_{2.5}$  and the associated cardiovascular risk. Both studies, discussed in Section 3.1, measured  $\text{PM}_{2.5}$  concentrations in the primary activity room of the home for 24 hours. Pang et. al.'s study in Nangong County of the Hebei Province in North China recorded an indoor exposure average of 87.0  $\mu\text{g}/\text{m}^3$ , with exposures ranging from 77 to 120  $\mu\text{g}/\text{m}^3$  when the participants were using traditional coal-burning methods for indoor heating their homes (Pang et al.). And, Caravedo et. al.'s study in Puno, Peru recorded an indoor exposure average of 178  $\mu\text{g}/\text{m}^3$  of  $\text{PM}_{2.5}$  when the study participants were using biomass fuel-burning methods for indoor heating (Caravedo, Painschab, et al.). Needless to say, these 24-hour indoor exposure averages are also significantly

higher than the recommendations listed by both the Indoor Air Hygiene Institute and the Environmental Protection Agency, and are cause for concern regarding a multitude of adverse health effects, including cardiovascular morbidity and mortality.

#### **4.2 Effect of PM<sub>2.5</sub> on Heart Rate & Heart Rate Variability**

A total of 15 studies included in this review observed significant effects of fine particulate matter on changes in heart rate and/or heart rate variability. Of these studies, 1 associated PM<sub>2.5</sub> with a decrease in HR and 3 associated PM<sub>2.5</sub> with increases in HR. A majority of the studies included in this review measured HR in some capacity, but only 4 mentioned significant associations between PM<sub>2.5</sub> and abnormal fluctuations in HR. Studies by Brook, He, and Tsou et. al. each associated short-term indoor or personal exposure to PM<sub>2.5</sub> with an increase in HR. An increase in HR is expected following exposure to PM<sub>2.5</sub> because it is one of the ways the human body responds to stress. Foreign particles inhaled into the body may initiate the body's stress response, but also may cause inflammation in the lungs as well as swelling of the airways, both contributing to abnormal increases in resting heart rate (Breecher). The study completed by Gabdrashova et. al., however, observed a reduction in HR following exposure to PM<sub>2.5</sub> during the process of cooking meat on a stovetop. This study found significant reductions in HR 90 minutes after cooking, and this is likely due to the fact that the study participants had had ample time to sit down and relax, causing their HR to return to normal. There are multiple stress factors involved in this study (i.e cooking over a hot stove, being observed by researchers, being in an unfamiliar environment such as a laboratory, etc.) that may have contributed to a significant increase in HR during the experiment followed by a drop in HR after relaxing for a 90-minute period. Other environmental stress factors aside from air pollution can contribute to fluctuations in HR and should be considered when interpreting observed HR results.

As for heart rate variability, 4 studies made associations between PM<sub>2.5</sub> exposure and increases in HRV, 5 studies made associations between increases in HRV changes and increased levels of PM<sub>2.5</sub> exposure, and one study found diminished HRV responses in patients diagnosed with ischemic heart disease. HRV, which is the fluctuation of time between each adjacent heartbeat, is a result of the two opposing branches of the autonomic nervous system, the parasympathetic branch and the sympathetic branch, working against one another to regulate heart rate. The sympathetic branch, which responds to stress, increases heart rate, while the parasympathetic branch works to decrease heart rate and return the body to its relaxed, normal state (Harvard Health Publishing). HRV is a very complex measurement, changing constantly in individuals and varying from individual to individual. Age, sex, normal resting HR, and overall health and lifestyle can all influence an individual's HRV (Shaffer and Ginsberg). Each of these factors, as well as other factors such as the clinical method of HRV measurement, could be responsible for differing results among studies.

#### **4.3 Effect of PM<sub>2.5</sub> on Systolic and Diastolic Blood Pressure**

Nine studies included in this review associated PM<sub>2.5</sub> with increases in blood pressure. To break this down further, 6 studies found increases in both systolic and diastolic pressures, 2 found increases in systolic pressure but no significant changes in diastolic pressure, and 1 observed increases in overall maternal blood pressure. Furthermore, all air filtration studies involving HEPA filters and/or other types of indoor air filtration included in this review found reductions in average blood pressures after use. There are multiple possibilities that may explain why these studies observed increases in BP following exposure to PM<sub>2.5</sub>. One probable mechanism presented by Brook et. al. suggests autonomic nervous system imbalance in favor of the sympathetic branch. Their research demonstrated a “blunting” of cardiovascular

parasympathetic tone in response to inhaled particulate matter pollution, leading to domination of sympathetic tone which increases BP as well as HR (Brook, Urch, et al.). Another team of researchers, however, attribute increases in BP in response to PM<sub>2.5</sub> exposure to endothelial cell dysfunction contributing to vasoconstriction, which is the narrowing of blood vessels.

Vasoconstriction decreases the available space through which blood can flow, thus increasing the resistance or force of blood flow which increases blood pressure (Fiordelisi et al.). A subsequent increase in overall BP in response to exposure to PM<sub>2.5</sub> is expected, however the exact mechanism is not fully understood.

#### **4.4 PM<sub>2.5</sub> Associations with Atherosclerosis**

Next, a total of 8 studies included in this review found associations between fine particulate matter and atherosclerosis, defined in Section 3.1 as abnormal thickening or hardening of the blood vessel wall resulting in disruption of the normal flow of blood. Of these eight research articles, one found an increase in the presence of atherosclerotic plaque, one found an increase in Carotid Intima-Media Thickness (CIMT), three found increases in endothelial inflammation, two found increases in the presence of Atherogenic Index of Plasma (AIP), and one found an increase in red blood cell count (i.e. an increase in blood thickness resulting in slowing of blood flow) as a result of exposure to PM<sub>2.5</sub>. Studies suggest that particulate matter exposures (PM<sub>10</sub>, PM<sub>2.5</sub>, and Ultrafine Particles (UFPs)) both long- and short-term inhibit arterial endothelium-dependent vasodilation, thus promoting vasoconstriction and contributing to the progression of atherosclerosis (Fiordelisi et al.). According to a journal by the American Heart Association, a majority of the PM<sub>2.5</sub>-associated deaths occur as a result of atherosclerotic cardiovascular disease (ASCVD), which is responsible for stroke and myocardial infarction. PM<sub>2.5</sub> exposure induces multiple mechanistic pathways which each contribute to the progression



of atherosclerosis and ASCVD. The most studied of these effects include endothelial cell dysfunction, tissue inflammation, thrombosis (blockage of vessels by blood clots), vasoconstriction, and secondary tissue damage (which induces plaque instability/sensitivity) (Bevan et al.).

#### **4.5 Limitations and Deficiencies of The Available Literature**

First, a limitation of this review is that the only studies available demonstrate the effects of short-term (hours to weeks) PM<sub>2.5</sub> exposures on cardiovascular health, there are no studies currently available which directly continuously monitor indoor or personal levels of PM<sub>2.5</sub> long-term (months to years) and make associations to cardiovascular health. This is likely due to a lack of participants willing to wear a personal monitor or leave a stationary indoor monitor in their home for an extended period of time. Some studies which estimate long-term air pollution exposure and/or use prediction modeling methods to determine likely long-term exposures exist, however they are not as accurate as direct measurement studies would be. Long-term personal and indoor PM<sub>2.5</sub> monitoring would provide a more accurate estimate of the total amount of PM<sub>2.5</sub> the average person is exposed to during their lifetime as well as a better understanding of the long-term effects of PM<sub>2.5</sub> exposure.

A second limitation of the literature available for this project involves the locations in which these studies were conducted. **Figure 2** is a map demonstrating the share of deaths from indoor air pollution in 2019, with the highest percentages of deaths occurring in the darkest-colored areas. These substantially-affected areas include most prominently the lowest-income areas of sub-Saharan Africa as well as some Middle Eastern countries. However, none of the studies included in this review were conducted in the areas that are harmed the most by the effects of indoor air pollution.

Finally, another limitation of this review is the lack of studies focused on children and childhood exposures to particulate matter pollution, despite children experiencing the highest levels of indoor PM<sub>2.5</sub> exposures due to the fact that they, on average, spend the majority of their time indoors, close to ground level where there is a high concentration of harmful air pollutants. Additionally, children breathe at a more rapid pace than adults, so they absorb more pollutants into their lungs and, thus, into their bloodstream. According to the WHO, 93% of the world's children (under the age of 15) breathe polluted air that puts their health and development at risk (WHO, *More than 90% of the World's Children Breathe Toxic Air Every Day*). Of the 37 total studies included in this review, only two included participants younger than 18 years of age. Further, one of the two studies only included a group of 19 children who had been diagnosed with asthma, providing insight into only a small, specific demographic. There are many reasons for the lack of studies involving children, but a main reason is the legal and ethical implications associated with doing research on children under the age of 18 who are unable to give informed consent. However, more research involving the health impacts of PM<sub>2.5</sub> on newborns and young children would undoubtedly provide greater insight into the unique air pollution-related dangers faced by children across the world, but particularly in low- and middle- income countries with limited access to air purification resources.

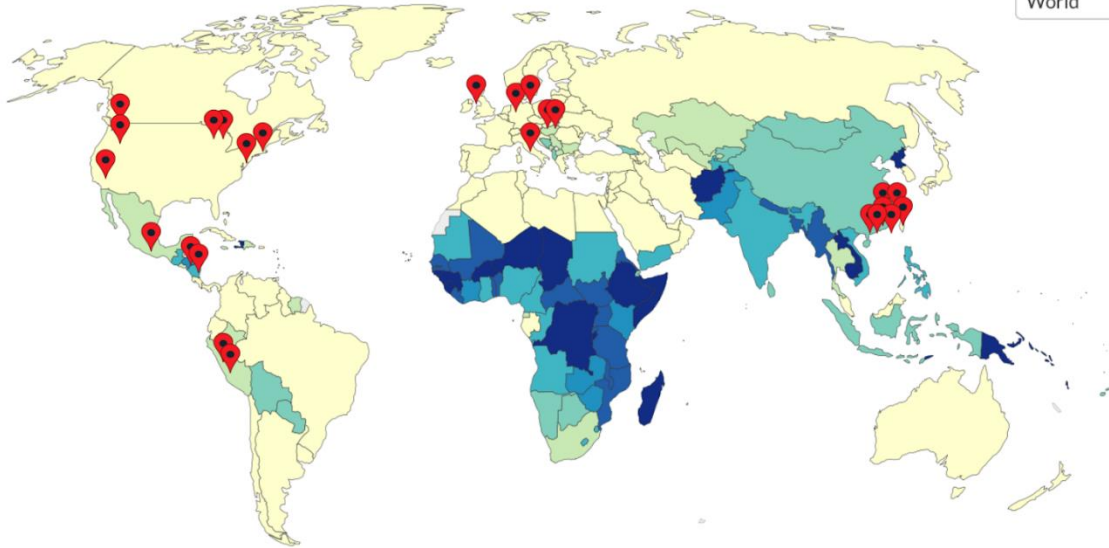
Figure 2: Map of Study Locations & Share of Deaths from Indoor Air Pollution in 2019

### Share of deaths from indoor air pollution, 2019

Share of deaths, from any cause, which are attributed to indoor air pollution – from burning solid fuels – as a risk factor.

Our World  
in Data

World



Roser, Max. <https://ourworldindata.org/energy-poverty-air-pollution>.

**Key:** Dark Blue Areas = highest share of deaths from indoor air pollution exposure  
Red pins = study locations

## **5 Conclusion**

Almost all of the studies included in this review made direct positive associations between short-term indoor and/or personal exposures to PM<sub>2.5</sub> and one or more cardiovascular health consequences. The findings of this review demonstrate the importance of further studying how indoor and personal exposures to PM<sub>2.5</sub> impact human cardiovascular health. More research on the health implications associated with indoor and personal exposures to PM<sub>2.5</sub> could improve public health and safety efforts across the globe, rather than focusing research on ambient air pollution alone.

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