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Occupational Fine Particulate Matter Exposure and its Associated Effects on the Cardiovascular System: A Systematic Review

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OCCUPATIONAL FINE PARTICULATE MATTER EXPOSURE AND ITS ASSOCIATED EFFECTS ON THE CARDIOVASCULAR SYSTEM: A SYSTEMATIC REVIEW

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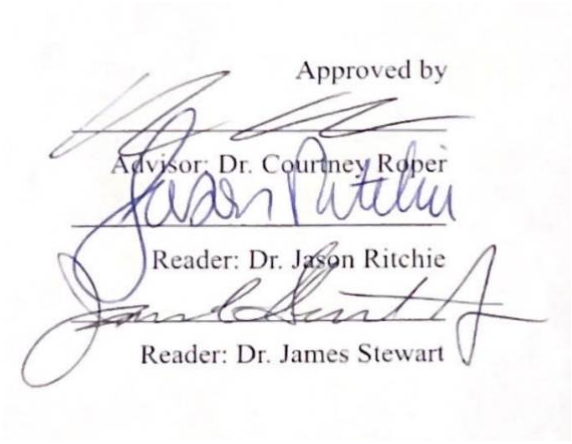

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ABSTRACT

Fine particulate matter ($PM_{2.5}$), the solid and liquid portion of air pollution under 2.5 microns in diameter, has been shown to cause numerous negative effects on the body. These inhalable particles are often researched for their effects on the respiratory system in outdoor settings, however systemic health impacts have been observed following inhalation of $PM_{2.5}$. Additionally, exposures to $PM_{2.5}$ can occur in occupational settings but are less frequently studied compared to outdoors. This literature review seeks to identify studies that determined associations between inhaled $PM_{2.5}$ and the resulting cardiovascular effects in occupational settings. We conducted a search of literature studying $PM_{2.5}$ exposures and cardiovascular outcomes. We analyzed 31 articles pertaining to key cardiovascular effects of $PM_{2.5}$ exposure in occupational settings, finding associations in 93.5% of cases. Because limited literature focuses on cardiovascular endpoints of $PM_{2.5}$ specifically in occupational settings, it is important that additional research is conducted in order to more fully comprehend the direct effects that $PM_{2.5}$ has on the cardiovascular system and other related cardiovascular risk factors.

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LIST OF ABBREVIATIONS

Fine Particulate Matter ($PM_{2.5}$)

Particulate Matter (PM)

Environmental Protection Agency (EPA)

Did Not Specify (DNS)

Confidence Interval (CI)

Standard Deviation (SD)

Respiratory Rate (RR)

Standard Deviation of Number of N (SDNN)

Total Pollution (TP)

Mitochondrial DNA (mtDNA)

Heart Rate Variability (HRV)

Heart Rate (HR)

Cardiovascular (CV)

Cardiovascular Disease (CVD)

Cardiopulmonary Disease (CPD)

Alternating/Direct Current (AC/DC)

Cardio-Ankle Vascular Index (CAVI)

Electrocardiogram (ECG)

Exhaled Nitric Oxide (eNO)

Volatile Organic Compounds (VOCs)

C-Reactive Protein (CRP)

Ischemic Heart Disease (IHD)

Blood Pressure (BP)

1.0 Introduction

According to the World Health Organization, air pollution accounts for an estimated total of 4.2 million deaths each year. Defined as unwanted, unhealthy chemical particles and substances in the atmosphere, these particles are classified into either primary or secondary categories.[1] Whereas primary pollutants are emitted straight into the air, secondary ones are resultant from the primary pollutants themselves. Examples of primary pollutants could be carbon monoxide, nitrogen oxide, and sulfur oxide, whereas a common secondary pollution could be ozone produced from nitrogen oxide reactions when VOCs, volatile organic compounds that easily evaporate at room temperature into the surrounding air, react with the nitrogen oxide and sunlight to create smog.[2] The composition of air pollution consists of a mixture of solid and gaseous particles in air. Most air pollutants arise from energy usage and production, often from fossil fuels that are burned and release pollutants as a byproduct into the atmosphere.[3] As a result, the constant buildup of these particles results in increased likelihoods of health detriments to humans, as well as adding to other existential problems such as climate change. Common effects of long-term exposure of air pollutants include respiratory illness, heart disease, and nerve damage, large problems that are dangerously prevalent today.

Under the Clean Air Act, the EPA has defined air pollution as one of six criteria, being classified as either ground-level ozone, particulate matter, lead, carbon monoxide, sulfur dioxide, or nitrogen dioxide.[4] Of the particulate matter criteria, pollution is categorized as either being coarse (PM_{10}) or fine ($PM_{2.5}$). Whereas PM_{10} is confined to pollution of equal to or less than ten

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[1] Daly and Zannetti, "An Introduction to Air Pollution – Definitions, Classifications, and History."
[2] "What Causes Smog?"
[3] June 22 and Turrentine, "Air Pollution."
[4] US EPA, "Criteria Air Pollutants."

micrometers in diameter, $PM_{2.5}$ has a diameter of 2.5 micrometers or less. For the purpose of this study, $PM_{2.5}$ was the only PM size analyzed.

1.1 Fine Particulate Matter

Fine Particulate matter ($PM_{2.5}$) is defined as any suspended particle that exists under a size of 2.5 um in aerodynamic cross-length.[5] These particles are, by definition of the United States Environmental Protection Agency (EPA), inhalable under any circumstance.[6] General examples of $PM_{2.5}$ are normally dust, pollen, and spores. $PM_{2.5}$ can also be categorized as an aerosol pollutant, or particles that have a liquid component to their makeup.[6] Common examples of $PM_{2.5}$ as aerosols are dust, sea salts and volcanic ash, and combustion byproducts from coal and auto emissions.

$PM_{2.5}$ can result from a number of different sources, with common ones being from construction sites, unpaved roads, fields, smokestacks, or fires.[7] Combustion exclusively accounts for most $PM_{2.5}$ production, as the five U.S. cities that contribute the most fine pollutants reside in California, one of the largest users of coal plants and natural gas factories.[9] Other potential sources are from nuclear plants and auto plants that give pollutants off as a byproduct from energy production and manufacturing. Previous research has found that the highest amounts of particulate matter are produced by metal industries (Pb, Zn, Fe, Mn), crustal/soil particles (Ca, Si), motor vehicle traffic (EC, NO_2), coal combustion (As, Se), oil combustion (V, Ni), salt particles (Na, Cl), and Biomass burning (K).[8]

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[5] Dockery and Pope, “Acute Respiratory Effects of Particulate Air Pollution.”
[6] US EPA, “Particulate Matter (PM) Basics,” April 19, 2016.
[6] US EPA, “Particulate Matter (PM) Basics,” April 19, 2016.
[7] US EPA, “Particulate Matter (PM) Basics,” April 19, 2016.
[9] “The Particulars of PM 2.5.”
[8] Thurston, Ito, and Lall, “A Source Apportionment of U.S. Fine Particulate Matter Air Pollution.” [11]
“Fine Particle Pollution.”

According to a revision in 2012 from the EPA and dictated through the Clean Air Act, the daily standard for primary and secondary $PM_{2.5}$ is no greater than 35 ug/m^3, alongside an annual average of no greater than 12 ug/m^3.[11] However, it should be noted that these standards exist only for outdoor air quality measures, and are separate from indoor and occupational standards. Similarly, the World Health Organization air quality guideline released in 2021 recommended no greater than 5 ug/m^3 annually, with an hourly suggestion of 15 ug/m^3 or less. Yet again, this release was only a suggestion and has no enforceability in real-time.

1.2 Health Endpoints of $PM_{2.5}$ Exposures

$PM_{2.5}$ is one of the most dangerous environmental health problems for human health systems. Due to its incredibly small size, $PM_{2.5}$ is able to infiltrate deep into lungs as well as enter the bloodstream, eventually traversing through the blood, and often affecting the entire body's functional ability by late stages.[9] This can result in lung disease, heart attack, and other disruption diseases such as arrhythmia and asthma, and is most often seen in the immunosuppressed, elderly, and early aged children.[5] There is also evidence to suggest a disruption of the endocrine system, as metabolic disease likelihood has shown to increase with increasing amounts of $PM_{2.5}$ intake.[10] In addition to internal system impairments, external problems arise through potential deficit of visual capability in the brain due to factors like obstruction of view and cloudedness.[5]

Cardiovascular Effects of $PM_{2.5}$

Researchers Hamanaka and Mutlu detail that acute and chronic exposure to $PM_{2.5}$ contribute to an increased risk of death from cardiovascular events including ischemic heart

[9] US EPA, "Health and Environmental Effects of Particulate Matter (PM)."
[10] Hamanaka and Mutlu, "Particulate Matter Air Pollution."

disease, heart failure, and thrombotic stroke[11]. Additionally, the two suggest that long and short-term studies indicate a correlation of $PM_{2.5}$ with increased risk of myocardial infarction and cerebrovascular disease, alongside a positive correlation between $PM_{2.5}$ exposure and blood pressure resulting from arteriolar constriction, a process more precisely defined as arteriolar narrowing. When a person is exposed to $PM_{2.5}$, research shows that inhaled particles can cause massive damage in the bloodstream, leading to inflammation of blood vessels that can cause blockages and clots, and thus resulting in cardiovascular events such as strokes or hypertension[12]. Increased exposure has also been shown to decrease the functioning ability of cells in the body, allowing for cardiovascular disease to become more prominent while lowering the immune system's ability to fight other cardiovascular effects from $PM_{2.5}$[13].

1.3 Occupational Exposures

Occupational exposures are defined as any presence of hazardous materials within the workplace that may affect the performance or overall risk of loss of function in said workplace.[14]More specifically, occupational exposures involve direct intake from a direct source within the work setting, and tests on these exposure types collect data from the person at the site or from the site itself. Examples of studies to these exposures often include high exposure to silica dust, asbestos, and welding fumes, and can be either in indoor or outdoor sites and environments.. Sampling of these events is commonly conducted through the use of personal monitors, devices directly attached to the subjects that continuously collect and measure $PM_{2.5}$

[11] Hayes et al., "PM2.5 Air Pollution and Cause-Specific Cardiovascular Disease Mortality."
[12] Wyatt et al., "Low Levels of Fine Particulate Matter Increase Vascular Damage and Reduce Pulmonary Function in Young Healthy Adults."
[13] Wyatt et al., "Low Levels of Fine Particulate Matter Increase Vascular Damage and Reduce Pulmonary Function in Young Healthy Adults."
[14] "Workplace Pollution | Environmental Pollution Centers."

exposure levels by analyzing contamination in the air as it filters through the device. Another frequently used sampling method uses devices that are instead at a stationary location, collecting $PM_{2.5}$ concentration and generalizing for larger areas. At this time, the Occupational Safety and Health Administration (OSHA) has set permissible exposure limits (PELs), eight-hour weighted average values of concentrations that should never be exceeded in order to keep from hazardous environments and toxic potential, on certain compounds.[15] However, most of these PELs are for specific gasses, and both the EPA and OSHA currently have no established PEL or guideline for $PM_{2.5}$ in the workplace, leading to potentially dangerous settings.

1.4 Current Study

In this study, a literature review was conducted by analyzing and comparing $PM_{2.5}$ exposure instances in an occupational setting with the cardiovascular health effects that were resultant. . We hypothesize that increased exposures to inhalable $PM_{2.5}$ will be positively associated with cardiovascular symptoms, as well as an overall increased risk of cardiovascular disease. By conducting this literature review, we attempt to show the potential dangers of $PM_{2.5}$ in occupational settings while providing insight for future studies that seek to study the associations between $PM_{2.5}$ and cardiovascular health.

2. Methods

Our research strategy consisted of both published and unpublished studies from an initial search from PubMed (NIH) in order to identify usable material. The reference list of all collected articles also included Embase (Elsevier) and Scopus (Elsevier). The search base for grey literature included Google Scholar, ClinicalTrials.gov, and Cochrane Central Register of Controlled Trials (Wiley). No time, language, or geographical limits were applied to the

[15] Spear and Selvin, "OSHA's Permissible Exposure Limits."

retrievals. We refined searches that linked $PM_{2.5}$ exposure in an occupational setting to cardiovascular health effects. The papers were collected using the keywords and index terms “cardiovascular”, “fine particulate matter”, “environmental exposure”, “occupational”, “heart rate variability”, “heart disease”, “hypertension”, “ and “systemic review” in order to specifically concentrate on world studies pertaining to exposure concentrations below current EPA standards. Additional focus was applied to studies that measured cardiovascular endpoints from $PM_{2.5}$ for analysis. Cardiovascular endpoints included mortality and morbidity associated with any cardiac events including hypertension, myocardial infarction, heart rate variability, atherosclerosis, and other ischemic heart disease. All studies including $PM_{2.5}$ concentrations below EPA standards with associations to human cardiovascular health endpoints were used, including all observational studies but excluding editorials, working papers, conference proceedings, reviews, and books. Papers were restricted to occupational experiments, excluding all indoor and ambient air exposures. Additional exclusion criteria included papers using any PM other than $PM_{2.5}$ (i.e. PM_{10}) and any papers measuring $PM_{2.5}$ with lacking total average concentrations. Data extraction included the author, title of work, location and date of experimentation, number of participants, average age, sex, any preexisting conditions of participants, exclusions from testing, monitor placement for measurement, sampling periods, any specific sources of $PM_{2.5}$, other pollutants measured, method of measurement, statistical methods and adjustments, any interventions used, average $PM_{2.5}$ concentration (ug/m^3), health associations and results, and conclusions. Extraction of said data allowed for efficient and definitive results to be categorized and associations to be further analyzed.

3.0 Results

After completing the search process, 64 articles were reviewed (out of 1,349 articles that were found). After all papers were analyzed, 31 articles fit the inclusion criteria and were included in the study. Each article was then grouped based on the occupation that it pertained to, with number of studies: Construction/Maintenance Workers (n=4), Boiler Workers (n=10), Mail/Truck/Taxi Drivers (n=5), Commuters (n=5), or Other (n=7). The remaining 33 articles were excluded due to either a lack of necessary information pertaining to $PM_{2.5}$ data, or for various other reasons. Regarding $PM_{2.5}$ data extraction, the article needed to have a total average $PM_{2.5}$ concentration, although two papers were included that had interquartile ranges with an additional total range of collection. Other exclusion reasons included papers that lacked any mention of $PM_{2.5}$, papers that failed to experiment within an occupational setting, papers that had no health associations or results pertaining to $PM_{2.5}$ that was measured, or papers that were literature reviews themselves (*Figure 1*). It should be noted that 4 papers were excluded from analysis due to limitations on accessibility, and therefore were no longer eligible for review.

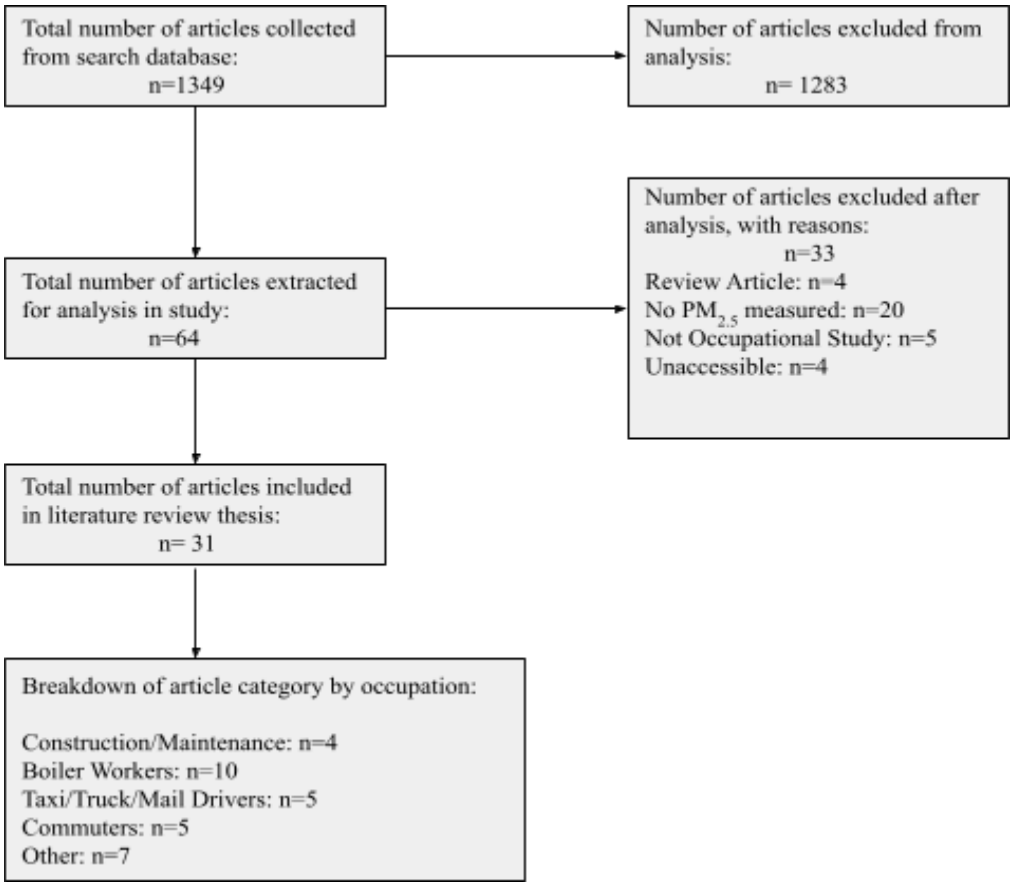

Figure 1: Systematic Review of total collection, inclusion, and exclusion criteria tree.

3.1 Occupational $PM_{2.5}$ Associations in Construction/Maintenance Workers

Table 1 displays the articles that tested $PM_{2.5}$ exposure and cardiovascular health associations on workers at construction sites and highway maintenance roads. The locations of these studies were all inside of the United States, either in Boston, Baltimore, or on an unidentified highway. The number of participants for each was between 18 and 57, with one study (Eninger, R. M. et. al.) having only 5 closely examined participants. All 4 studies had participants that averaged around 40 years old, and all subjects were male. Two studies excluded any cardiovascular disease or devices such as pacemakers and stunts, and two studies (Magari,

et. al.) included all participants assuming they had a calculable average $PM_{2.5}$ concentration. Two studies (Magari, et. al; Eninger. R. M. et al.) identified participants with existing smoking statuses or cardiovascular conditions including hypertension, bronchitis, and heart disease. Three studies used a personal monitor to record $PM_{2.5}$ concentration, and one study used monitors that were instead placed on the toolboxes of the workers. (Eninger. R. M. et. al.). Sampling periods ranged from 4-24hr monitoring, taking either total averages or at specific intervals. For the study in Boston (Magari et. al.), 4hr and 9hr $PM_{2.5}$ average concentrations were 69.7 (93.5) ug/m^3 and 15.9 (24.1) ug/m^3, respectively. The study associated $PM_{2.5}$ exposure with decreased respiratory rate (RR), observed decreases were 2.66% for every 1000 ug/ mg^3 increase in $PM_{2.5}$ over the duration of the study. A slowed RR can result in low blood oxygen, increased levels of acidity in the blood, or even complete respiratory failure, making it a likely potential precursor to further impacting cardiovascular disease (CVD).[16] In another study (Megari et. al.), construction workers were exposed to average $PM_{2.5}$ concentrations of 116.0 ug/m^3. The study collected specific metals such as vanadium and lead, found significant associations between exposure to airborne metals and alterations in cardiac autonomic function, a regulator of blood pressure and heart rate (HR)[17]. In this study, HR instead increased in the standard deviation from the normal number (SDNN) of 11.30ms and 3.98ms for every 1ug/m^3 increase of exposure. In the study by Meier, R. et. al., $PM_{2.5}$ was collected during work and after work for maintenance workers, with average concentrations of 65.7 (69.9) ug/m^3 and 22.9 (19.5) ug/m^3, respectively. The study associated increased heart rate variability (HRV) with increased $PM_{2.5}$ levels, including additional variability due to work noise as well. HRV occurs when the time between heart beats is inconsistent, and

[16] ("Bradypnea: Causes, Symptoms, and Treatment" 2017)
[17] (Hägglund et al. 2012)

has been previously associated with higher risk of future cardiovascular and mental issues.[18] In the last study, 5 participants were monitored six times to find their total average $PM_{2.5}$ concentrations of 64.6 (48.3) ug/m^3. Of the five participants, four had insignificant associations. One of the subjects, age 63, showed a significant positive association between $PM_{2.5}$ exposure and RR after each 4hr exposure assessment. Opposed to decreased HR, increased rates (tachycardia) can result in chest pain, shortness of breath (SOB), fainting, or more serious illnesses such as heart failure or strokes.[19] Overall, there were four studies, three of which showed associations between increased $PM_{2.5}$ levels and increased HR, and one which showed a decrease in respiratory rate.

[18] ("Heart Rate Variability (HRV): What It Is and How You Can Track It" n.d.)
[19] ("Tachycardia - Symptoms and Causes" n.d.)

***Table 1**: Construction/Maintenance Workers and $PM_{2.5}$ Exposure*

Study Design		Participants				Sampling Period	Monitor Placement	Avg. $PM_{2.5}$ Conc. ug/m^3 ± (sd)	Results and Cardiovascular Associations	Reference
Location	**Time**	**Number**	**Mean Age/ Gender**	**Exclusions**	**Existing Conditions**					
Boston, US	DNS	57 (33 4hr avg.) (24 9hr avg.)	38.2, Male	Those working without a calculable average.	Not Specified	4hr or 9hr moving average over 24hr.	Personal	4hr- 69.7 (93.5) 9hr- 15.9 (24.1)	2.66% decrease (95% CI, −3.75% to −1.58%) in the 5-minute SD of normal RR intervals (SDNN) for every 1 mg/m^3 increase	(Magari et al. 2001)
Construction sites in US	DNS	39	38.3, Male	None	Hypertension (n=7) Chronic bronchitis (n=8) smokers	24hr personal collection	Personal	116.0 (161)	Statistically significant mean increases in the SDNN index of 11.30 msec and 3.98 msec for every µg/m3 increase in the lead and vanadium concentrations. Suggests an association between exposure to airborne metals and significant alterations in cardiac autonomic function.	(Magari et al. 2002b)
Highway Maintenance Roads in US	Days between May 2010- Feb 2012	18	46.0, Male	Hypertension, Cardiopulmonary health problems, acute allergies, diabetes, obesity.	Had hypertension (were treated).	From work till morning after (>15 hours)	Personal	During Work- 65.7 (69.9) After Work- 22.9 (19.5)	$PM_{2.5}$ and work noise were associated with markers of increased heart rate variability, and with increased high-frequency and low-frequency power.	(Meier et al. 2014)
Baltimore, Maryland in maintenance facilities	Dec. 2001 March 2002	5	36, 38, 63, 28, 45 (Not Means), Male	Pacemaker Usage, beta/calcium blocker usage, other night employment.	Smokers, Heart Disease, Hypertension	8hrs a day (3 workers) 4 hrs a day (2 workers)	On top of the participant's toolbox	64.7 (48.3)	Subject 3 showed significant positive association with $PM_{2.5}$ 4-hour exposure for SDNN (regression coefficient = 2.55, p = 0.013) and TP (regression coefficient = 967.4, p = 0.012)	(Eninger and Rosenthal 2004)

Terms:
DNS- Did Not Specify; CI- Confidence Interval; SD- Standard Deviation; RR- Respiratory Rate; SDNN- Standard Deviation of Number of *N* (*N* being any beat); "Personal"- attached to the participant or moving within 1 meter of breathing area; TP- Total Pollution

3.2 Occupational $PM_{2.5}$ Associations in Boiler Workers

Table 2 is an outlook of the collection of studies conducted on boiler workers and their cardiovascular associations. Analyzing 10 articles all between the years of 1996 and 2012, most noted locations were in the United States in Massachusetts. Although a number of articles did not identify the location of the study (n=5), it is likely that many were also in Massachusetts due to multiple studies having been conducted by the same research team. Each study ranged from 2072 participants, all of which were male save for one article (Fan, T. et. al.). The studies excluded factors such as diabetes, irregular heartbeat patterns, CVD, hypertension, and any who took medications. One study by Cavallari, et. al. and one by Fang. S. C. et. al. had no stated exclusions from participation, and one study excluded those who were nonsmokers living among smokers (Magari, S. R. et al.). Similarly to the previous studies, all but two articles measured $PM_{2.5}$ using personal monitors that were attached to the person or somewhere within 1m of the participants breathing zone. The other two studies collected pollutants by placing a stationary monitor at the work site (Byun, H. M. et al. and Fan, T. et. al.).

Five conducted studies had associations between $PM_{2.5}$ exposure and HRV. In a study by Byun, H. M. et. al., boiler workers were sampled either on welding or non-welding days for five hours, collecting average $PM_{2.5}$ concentrations of 38.0 ug/m^3 and 15.0 ug/m^3, respectively (no standard deviations were given). The study concluded that mitochondrial DNA (mtDNA) methylation, a process that occurs with factors such as aging and smoking, showed a negative association with $PM_{2.5}$ levels, further suggesting that higher blood mtDNA allowed for higher susceptibility of risk of decreased HRV. In another study, workers were monitored for around five hours during a working day, averaging 43 (34.0) ug/m^3 in $PM_{2.5}$ concentrations (Fan, T., et.

al.). Results showed an acute decline of HRV when exposure to metal-rich $PM_{2.5}$ increased. In one study by Cavallari, et. al., workers were monitored for 24 hours over a span of seven years, collecting mean $PM_{2.5}$ concentrations of 73.0 (50.0) ug/m^3 and also observing an inverse relationship between HRV and increased $PM_{2.5}$ exposure. Another study by Cavallari and his team monitored a number of participants during varying amounts of time while working, averaging 5.33 hours. The results suggested a decline in HRV for 14 hours following PM exposure, although 12 participants were smokers and another 6 hypertensive. Additionally, the study suggested cardiovascular autonomic responses and delayed responses as $PM_{2.5}$ exposure increased. The last study associated with HRV collected mean $PM_{2.5}$ concentrations every four hours for 24 hours, averaging 1.92 (1.22) ug/m^3. From the data, they concluded that $PM_{2.5}$ may affect HRV and HR measures based on an individual's CAD profile, or likelihood of becoming ill with coronary artery disease. It should be noted that an unspecified number of participants were smokers or had a recent history of smoking.

The other five studies conducted all had CV associations with $PM_{2.5}$ exposure, but either focused on general cardiovascular (CV) health, heart rate, broader arrhythmogenic effects, or heartbeat electrical output. For CV health, one study (Cavallari, et. al.) measured participants for over 31 periods of one working and one non working day, collecting a median $PM_{2.5}$ concentration of 650 ug/m^3. The results of this study supported cardiotoxicity of $PM_{2.5}$ metal exposure and impact on cardiovascular health. Two other studies focused on the relationship between HR itself and $PM_{2.5}$ exposure. One study (Magari, S. R. et al.) monitored smokers and nonsmokers for one 24 hour period, collecting mean $PM_{2.5}$ concentrations of 96 (158) ug/m^3. For nonsmokers, they found a 1.9% increase in HR after the period. Another study (Fang, S. C. et al.) split monitoring sessions into welding and non welding shifts, gathering mean $PM_{2.5}$

concentrations of 88.7 (54.2) and 20.2 (37.9) ug/m^3, respectively. Although four workers had hypertension, the results showed an inverse relationship between $PM_{2.5}$ and SDNN of HR, suggesting that HR was lowered instead of being raised. Regarding arrhythmogenic associations, one study monitored participants for anywhere from 5-90 hours during work days and non-work days throughout one of four years from 1999, 2003, 2004, and 2006 (Cavallari, et. al.). After collecting an average of 274 (754) ug/m^3 over the entire year, the data suggested that individuals who lack underlying CVD are susceptible to arrhythmogenic effects of particle exposures. Arrhythmogenic effects can be any of the conditions that are associated with heart beat patterns, being either increased heart rate, decreased heart rate, or heart rate variability, and can cause fatigue, light-headedness, sweating, fainting, and anxiety[20]. Lastly, another study measured exposure for 4-6 hours five times over a two year period, collecting mean $PM_{2.5}$ concentrations of 47.0 (30.0) ug/m^3 (Umukoro, P. E. et. al.). The results suggested that metal-rich $PM_{2.5}$ was associated with a reduction in cardiac AC and DC, lasting for up to 60 minutes after exposure was depleted. AC and DC currents control when the heart contracts to pump blood out of the heart and to the rest of the body, and current variability is associated with atrial fibrillation (AF), a condition closely linked to strokes and heart failure[24]. In all, 10 studies were analyzed with associations between increased $PM_{2.5}$ exposures and decreased HRV (n=3), varied change in HRV (n=2), decreased HR (n=1), increased HR (n=1), increased cardiotoxicity (n=1), arrhythmogenic effects on the heart (n=1), or decreased AC and DC current output (n=1).

[20] ("Symptoms, Diagnosis and Monitoring of Arrhythmia | American Heart Association" n.d.)
[24] (Rienstra et al. 2012)

Table 2: *Boiler Workers and $PM_{2.5}$ Exposure*

Study Design		Participants				Sampling Period	Monitor Placement	Avg. $PM_{2.5}$ Conc. ug/m^3 ± (sd)	Results and Cardiovascular Associations	Reference
Location	**Time**	**Number**	**Mean Age/ Gender**	**Exclusions**	**Existing Conditions**					
Local Boilers in US	Jan 2007-June 2012	48	<30 (10) 30-40 (10) 40-50 (9) 50-60 (11) >60 (5), Male	Cardiovascular Disease, alcohol consumption	None	6 cycles over period. Sampled after no work and after 5hrs of work	Background ambient environment	38.0 on welding day 15.0 on non-welding day	mtDNA methylation showed a negative association with $PM_{2.5}$ exposure levels. Results suggest that persons with higher blood mtDNA methylation levels were more susceptible to the adverse PM effect on HRV measures.	(Byun et al. 2016)
Quincy, Massachusetts at Boilermaker Union local 29	Jan 2010-June 2012	66	NA, Male and Female	Cardiovascular Disease	None	During the work day for avg. of 5hrs.	Personal and in surrounding work area	43 (34.0)	Results show the acute decline of HRV following the exposure of metal-rich welding $PM_{2.5}$ and support evidence of a short-term cardiac response to boiler exposure.	(Fan et al. 2014)
Local Boiler Site, US	1996-2006	36	41.0, Male	Cardiovascular Disease, Hypertension	Hypertension and CV disease (participation was removed)	24hr non working monitor 8-hr time-weighted averages calculated over 7 years.	Personal	73.0 (50.0) mean with a range of 4.0-270	Observed a consistent inverse exposure-response relationship, with a decrease in all HRV measures with increased $PM_{2.5}$ exposure	(Cavallari et al. 2007)
Massachusetts Boiler Sites	Jan. 25-Feb 8, 2003	26	34, Male	Diabetes	Some Smokers	24 hrs monitoring. 4hr real-time avgs.	Personal	1.92 (1.22)	$PM_{2.5}$ may affect HRV and HR measures based on an individual's CAD profile.	(Chen et al. 2006)
Unspecified Local Welding School	1999-2006	26	43, Male	Medications	Potential Diabetes	31 Periods of 1 working and 1 non-working day.	Personal	650 (median only)	Results support the cardiotoxicity of $PM_{2.5}$ metal exposures, specifically manganese, on cardiovascular health	(Cavallari, Eisen, et al. 2008)

Study Design		Participants				Sampling Period	Monitor Placement	Avg. $PM_{2.5}$ Conc. ug/m^3 ± (sd)	Results and Cardiovascular Associations	Reference
Location	**Time**	**Number**	**Mean Age/ Gender**	**Exclusions**	**Existing Conditions**					
Unspecified Local Welding School	1999-2006	36	40, Male	None	12 Smokers 6 Hypertensive	Over a work shift of mean length 5.33 hr	Personal	112 (76)	Suggests declines in HRV for up to 14 hours following PM exposure and a multiphase cardiovascular autonomic response with immediate (2 hrs) and delayed (9-13 hrs) responses.	(Cavallari, Fang, et al. 2008)
Local Unspecified Boiler Site	2006	23	40.0, Male	None	4 Hypertensive	36-day collection of both wielding and non-wielding days.	Personal	Wielding Shift - 88.7 (54.2) Non-wielding- 20.2 (37.9)	Observed an inverse association between the 1-hour $PM_{2.5}$ and 5-minute SDNN	(Fang et al. 2009)
Local Boiler Site, US	1996-2006	36	41.0, Male	Cardiovascular Disease, Hypertension	Hypertension and CV disease (participation was removed)	24hr non working monitor 8-hr time-weighted averages over 7 years.	Personal	73.0 (50.0) mean with a range of 4.0-270 ug/m^3	Observed a consistent inverse exposure- response relationship, with a decrease in all HRV measures with increased $PM_{2.5}$ exposure	(Cavallari et al. 2007)
Not Specified	June to August 1999	20	42, Male	Nonsmokers living with smokers	9 Smokers	One 24 hr period.	Personal	96 (158)	No mean increase in HR associated with an increase in 100 ug/m^3 in the 3hr moving avg. $PM_{2.5}$ for smokers. For nonsmokers, a 1.9% increase in HR was observed	(Magari et al. 2002a)
Local Boiler Union, US	1999, 2003, 2004, 2006	72	38, Male	High Ectopic Beat Averages, Cardiovascular Disease	Not Specified	5-90 hrs during work days and non-work days throughout the year.	Personal	274 (754) over the entire period.	Suggests that individuals with no known underlying cardiovascular disease are susceptible to the arrhythmogenic effects of particle exposures	(Cavallari et al. 2016)
Quincy, Massachusetts Boiler Union	Jan. 2010-June 2012	48	40, Male	Prior Heart Problems and Any Welding 2 weeks Prior to Testing	5 participants had Heart Problems (were excluded)	4-6hrs data repeated five times over the sampling period	Personal	7.0 (30)	Metal-rich occupational PM2.5 exposure is associated with a reduction in cardiac AC and DC lasting up to 1 h after exposure has ceased	(Umukoro et al. 2016)

Terms:
mtDNA- Mitochondrial DNA; HRV- Heart Rate Variability; CAD- Coronary Artery Disease; HR- Heart Rate; PM- Particulate Matter; CV- Cardiovascular; AC- Alternating Current; DC- Direct Current

3.3 Occupational $PM_{2.5}$ Associations in Mail, Truck, and Taxi Drivers

Table 3 presents the studies that pertained to mail, taxi, and truck drivers and their cardiovascular associations with $PM_{2.5}$ exposure. The mail delivery studies (n=2) took place in Taiwan, the taxi driver studies (n=2) were conducted in Beijing, and the singular truck driver study took place across the entirety of the United States. Each study had between 11 and 17 participants, save for one article that analyzed 54,319 drivers (Hart, J. et. al.). The average age of each study stood at 36 years old, all testing on males except for two studies (Wu. S, et. al; Deng, F. et. al.) that tested men and women. Common exclusion criteria were cardiovascular disease, smokers, alcoholics, those taking medication, and obesity. One study (Deng, F. et. al) excluded all data collected while the participant was outside of the taxi, and one study (Hart, J. E. et. al.) excluded long-haul drivers who carried loads for two or more days. One study (Wu, C. F. et. al.) had five participants with a history of smoking, while the other four noted no preexisting conditions. Sampling periods ranged from 5-12 hr periods, with one study measuring annual average exposure to $PM_{2.5}$ (Hart. J. E. et. al.)

3.3a Mail Delivery Endpoints and Associations

Both mail delivery studies were conducted from February to March of 2007. In one article by Wu, C. F. et. al., participants wore personal monitors for 5-6 consecutive working day hours, collecting average $PM_{2.5}$ levels of 68.2 ug/m^3. The study found that an increase in personal exposure to ozone with particulate matter of diameter between 1.0 and 2.5ug was associated with a 4.8% and 2.5% increase in cardio-ankle vascular index (CAVI), respectively. CAVI is an index that is used to determine the overall stiffness of the arteries in the body, and has been linked to

arteriosclerosis and other diseases that can lead the heart to pump blood less efficiently[21]. The other study by Wu, C. F. et. al. used the same sampling method and period as before, collecting an average $PM_{2.5}$ concentration of 12.7 (6.2) ug/m^3. Participants in both studies were monitored for $PM_{2.5}$ using a personal cascade impactor sampler (PCIS) and for CAVI using a transportable monitoring machine (Vasera VS-1000). The results of the study suggested that an increase in personal exposure to $PM_{2.5}$ was associated with a 3.28% increase in CAVI.

3.3b Taxi Driver Endpoints and Associations

Both studies were conducted during the Beijing 2008 olympics. In one study (Wu, S. et. al.), participants were measured for $PM_{2.5}$ inside their car before, during, and after a 12 hour workshift, collecting average concentrations of 93.0 (44.1), 45.5 (26.7), and 77.3 (73.3) ug/m^3, respectively. The results supported that metallic $PM_{2.5}$ affects HRV in young and healthy individuals. In the other study (Deng, F. et. al.), participants also were monitored before, during, and after a 12 hour shift, collecting average $PM_{2.5}$ concentrations of 95.4 (58.6), 39.5 (25.2), and 64.0 (80.0) ug/m^3, respectively. This study found that traffic-related $PM_{2.5}$ exposure was associated with a change in cardiac autonomic function in young and healthy adults.

[21] (Shirai et al. 2011)

3.3c Truck Driver Endpoints and Associations

In the study conducted on truck drivers in the US (Hart, J. E. et. al.), drivers were monitored over the course of the year while working using a nearby ambient monitor and a mixed model that calculated average basal $PM_{2.5}$ levels based on location. After compiling all working years by participants, the average annual exposure to $PM_{2.5}$ was 4000ug/m^3. The results suggested an association between intake of $PM_{2.5}$ and cause-specific mortality due to either lung cancer, cardiovascular disease, or respiratory disease, suggesting that the cardiovascular system is altered and likely harmed by $PM_{2.5}$.

Table 3: *Mail/Truck/Taxi Drivers and $PM_{2.5}$ Exposure*

Study Design		Participants				Sampling Period	Monitor Placement	Avg. $PM_{2.5}$ Conc. ug/m^3 ± (sd)	Results and Cardiovascular Associations	Reference
Location	**Time**	**Number**	**Mean Age/ Gender**	**Exclusions**	**Existing Conditions**					
Mail Delivery										
Taipei, Taiwan	Feb.-Mar c h 2007	17	36.5, Male	Smokers, Alcohol, Cardiovascular Disease, Medications	None	5-6 consecutive working day hours	Personal	68.2	Found that an interquartile range increase in personal exposure to ozone and particulate matter of between 1.0 and 2.5 lm was associated with a 4.8% and 2.5% increase in CAVI, respectively.	(C. Wu et al. 2010)
Taipei, Taiwan	Feb-Mar c h 2007	17	32.4, Male	Cardiovascular Disease	Had asmoking history	5-6 days while delivering mail	CAVI- Quiet Room in office	12.7 (6.2)	Increase in exposure to PM from regional sources was significantly associated with a 3.28% increase in CAVI	(C. Wu et al. 2012)
Taxis										
Beijing, China	2008 Olympics	14	35.6 , Male and Female	Cardiac Risk Factors, smokers, obesity	None	Before 9am, 9am-9pm, and after 9pm	Monitor in Car	Before- 93.0 (44.1) During- 45.5 (26.7) After77.3 (73.3)	Supports the associations of several $PM_{2.5}$ metallic components with HRV in younger healthy individuals	(S. Wu et al. 2011)
Beijing, China	2008 Olympics	11	35.5 (Male and Female)	Data While Not Inside Taxi	None	Before, during, and after 12 hr work shift	Personal	Before- 95.4 (58.6) During- 39.5 (25.2) After- 64.0 (80.0)	Marked changes in traffic-related $PM_{2.5}$ exposure were associated with altered cardiac autonomic function in young, healthy adults	(S. Wu et al. 2010)
Trucks										
Continental US	Jan-Dec 1985	54,319	42.1, Male	Long-Haul Drivers	None	Annual Avg. Exposures were taken	Nearby Ambient Monitor	4000 ug/m^3 annually	Elevated associations with cause-specific mortality were observed for $PM_{2.5}$	(Hart et al. 2011)

Terms: CAVI- Cardio-Ankle Vascular Index; ECG- Electrocardiogram

3.4 Occupational $PM_{2.5}$ Associations in Commuters

Table 4 shows the data collected from commuters and the associations between $PM_{2.5}$ exposure and potential cardiovascular effects. The locations of the studies took place in China, Canada, Taiwan, and Georgia, US. Participants averaged 40 in number of both males and females, and the ages ranged from 18-50 years. Common exclusion criteria included diabetes, cardiovascular diseases, pregnancy, smokers, medications, hypertension, and systemic illnesses.

In one study (Sarnat. J. A. et. al.) of residents in Atlanta, GA, 50% of participants had asthma and were compared to those without asthma. Monitored for two periods of 2 hours during commutes and for three hours after the commutes, average concentrations of $PM_{2.5}$ were found to be 19.2 (13.6) ug/m^3. Results showed that elevated levels of nitric oxide exhalation (eNO) were associated with systemic inflammation, decreasing HRV indices and indicating cardiac dysfunction in both asthma and non-asthma groups. In another study by Jia, X, et. al, Beijing commuters were monitored from 9:00am to 1:00pm on weekends, collecting median and range $PM_{2.5}$ concentrations of 64.1 ug/m^3 and 6-325.1 ug/m^3, respectively. This study suggested that for all averages taken at 5 minutes, 1 hour, and 2 hours, increased $PM_{2.5}$ levels were associated with decreases in HRV. For another study conducted in Beijing (Yannan, Z. et. al.), participants were monitored for 4 hours during travel with or without a respirator, finding average $PM_{2.5}$ concentrations of 86.77 ug/m^3. The results indicated that short-term exposure to $PM_{2.5}$ increased the likelihood of cardiovascular disease, but also suggested that a respirator could effectively reduce these effects. Another study (Dales, R. et. al.) monitored Canadians in Ottawa, collecting average $PM_{2.5}$ concentrations of 40 (20) ug/m^3 after 2 hour periods at bus stops in the downtown area. Researchers found that a 30 ug/m^3 increase of $PM_{2.5}$ was associated with a 5% decrease in

the elasticity of arteries, indicating a weakened ability to vasodilate. The last study (Liu, W. et. al.) monitored commuters in Taipei, Taiwan in four different travel modes. Participants were monitored for 1 hour in each mode, collecting average $PM_{2.5}$ concentrations of 29.2 (11.3), 22.3 (6.9), 42.1 (18.2), and 32.2 (12.4) ug/m^3 for car, subway, walk, and bus modes, respectively. The results indicated an inverse relationship between $PM_{2.5}$ exposure and HRV indices. In all, 5 studies were conducted on commuters, associating $PM_{2.5}$ exposure with decreased HRV (n=3), increased risk of CVD (n=1), and decreased ability to dilate arteries (n=1

Table 4: *Commuters and $PM_{2.5}$ Exposure*

Study Design		Participants				Sampling Period	Monitor Placement	Avg. $PM_{2.5}$ Conc. ug/m^3 ± (sd)	Results and Cardiovascular Associations	Reference
Location	**Time**	**Number**	**Mean Age/ Gender**	**Exclusions**	**Existing Conditions**					
Metro in Atlanta, GA	Dec. 2009-April 2011	42	32.4, Male and Female	Pregnancy, Diabetes, CVD/CPD	50% (21) had asthma	2 periods of 7am-9am with posttests every hour until 3 hours after	Personal (in car <1m from breathing zone)	19.2 (13.6)	An elevated eNO response was indicative of pulmonary and systemic inflammation, decreased HRV, and indicative of autonomic dysfunction.	(Sarnat et al. 2014)
Beijing, China	March and May, 2007	39	21.2, Male and Female	Smokers, any history of cardiovascular, pulmonary, neuronal, or endocrine disease	None	9:00am to 1:00pm on weekends	Personal	Median 64.1 Range 6–325.1	For 5-min, 1-h, and 2-h moving averages, study showed decreases in HRV indices in all three variables.	(Jia et al. 2018)
Beijing, China	March-May 2017	39	21.4, Male and Female	Smokers, medications, cardiovascular disease	None	4 hours during travel, once with a respirator and once without	Personal	86.77	Results Indicated that short-term exposure to PM in a subway environment may increase the risk of cardiovascular disease. Wearing a facemask could reduce adverse effects.	(Zhang et al. 2019)
Ottawa, Canada	Unspecified	39	Male and Female Range: 18-50	Artery diseases, hypertension, diabetes, smokers, medications, respiratory illnesses	None	2 hr monitor periods at bus stops in a downtown setting	Personal	40 (20)	A 30ug/m^3 increase in $PM_{2.5}$ was associated with an equivalent of 5% relative change in maximum ability to vasodilate.	(Dales et al. 2007)
Taipei, Taiwan	Jan-March 2012-2014	120	21.3, Male and female	Smokers, cardiovascular disease, hypertension, diabetes, artery disease	None	1 hr monitoring on 4 different commutes	Personal	Car: 29.2 (11.3) Subway: 22.3 (6.9) Walk: 42.1 (18.2) Bus: 32.2 (12.4)	The results showed an inverse relationship between $PM_{2.5}$ exposure and HRV indices	(Liu et al. 2015)

Terms:
eNO- exhaled Nitric Oxide

3.5 Occupational $PM_{2.5}$ Associations in Other Occupations

Table 5 displays the association between cardiovascular effects and $PM_{2.5}$ exposure from studies that were from other occupational groups. These consisted of a study each from car-patrol officers, sugar and ethanol mill workers, highway patrol cars, aluminum industry workers, hairdresser assistants, traffic police men, and convenience store workers. The average age of participants was 26, with one study (Neophytou, A. M. et. al.) having an average of 44.3 years of age and one study (Zhao, J. et. al.) having a range of 25-55 years in place of an average. All but two studies (Ma, C. M. et. al; Chuang, K. J. et. al.) consisted of male-only participants. Common exclusion criteria included smokers, CVD, hypertension, medications, and caffeine usage. Only one study contained preexisting conditions (Neophytou, A. M. et. al.), where some participants were either current (<29.7%) or former (<34.1%) smokers.

In the study conducted on car-patrol officers (Riediker, M. et. al.), a pair of troopers in North Carolina were monitored each day in their car for four 9-hour workdays, collecting average $PM_{2.5}$ concentrations of 23.0 (10.8) ug/m^3. The results suggested that emissions from other vehicles are important sources of $PM_{2.5}$ that require further investigation for its impact on cardiovascular health in urban centers. In the study on sugar and ethanol mill workers (Barbosa, C. M. et. al.), participants in the Brazilian countryside were monitored for a complete 24 hours, collecting a median $PM_{2.5}$ concentration of 87.0 ug/m^3 with a range of 70-100 ug/m^3. These results displayed an increase in diastolic blood pressure by 11.12 mmHg and 5.13 mmHg during harvest and non-harvest periods, respectively. A healthy diastolic blood pressure is present at

values less than 80 mmHg, with hypertension being diagnosed when it reaches above 90 mmHg, resulting in an increased risk of stroke and heart disease[22]. In a study conducted in Wake County, NC (Riediker, M.), highway patrol officers were monitored from Monday-Thursday from 3pm-12am, collecting average $PM_{2.5}$ concentrations of 24.1 (13.5) ug/m^3. Research suggested that pollutant particles such as copper, sulfur, and calcium may be directly associated with the development of inflammation, coagulation, and other cardiac problems in healthy young men. For the study of aluminum workers (Neophytou, A. M. et. al.), 6058 smelters and 5623 fabricators across 11 US locations were monitored for various lengths of time during a workday. After $PM_{2.5}$ collection, smelters averaged 198 (162) ug/m^3 annually and fabricators 34 (5.0) ug/m^3 annually. The results suggested an observed association between risk of Ischemic Heart Disease (IHD) and increased $PM_{2.5}$ exposure. In the study on hairdresser assistants in Taiwan (Ma. C. M. et. al.), workers were monitored for 12 hours from 9am-9pm during work shifts, collecting average $PM_{2.5}$ concentrations of 31.7 (10.4) ug/m^3. The results showed associations between volatile organic compounds (VOCs) and increases in C-reactive protein levels (CRP). VOCs are gaseous byproducts such as ethylene-glycol and formaldehyde that are dangerous and have been linked to diseases like cardiovascular cancers[23]. CRP is a biological marker of inflammation due to its release when arteries and tissues become inflamed, serving as an efficient indicator of potential heart and arterial diseases[28]. For the article studying traffic policemen in Shanghai (Zhao, J. et. al.), workers were monitored for a 24hr cycle from 8am-8pm, collecting mean concentrations of 101.7 (46.8) ug/m^3. After analysis, $PM_{2.5}$ exposure was associated with an increase in CRP of 1.1%. Lastly, in a study on convenience workers in Taipei, Taiwan (Chuang,

[22] (CDC 2021)
[23] ("Volatile Organic Compounds (VOCs) in Your Home - EH: Minnesota Department of Health" n.d.)
[28] ("C-Reactive Protein (CRP) Testing for Heart Disease" n.d.)

K. J. et. al.), participants were monitored for 8 hours in either a daytime or nighttime workshift, collecting average $PM_{2.5}$ concentrations of 26.5 (9.5) ug/m^3 and 22.1 (4.3) ug/m^3, respectively. Researchers observed an inverse relationship between $PM_{2.5}$ exposure and HRV with decreasing HRV indices as exposure increased. In all, 7 studies were conducted that suggested $PM_{2.5}$ was associated with increased diastolic blood pressure (n=1), increased risk of IHD (n=1) , increased CRP levels (n=2), decreased HRV indices (n=1), and potential cardiovascular risks to individuals in areas of high-density traffic (n=2).

Table 5: *Other Occupations and $PM_{2.5}$ Exposure*

Study Design		Participants					Sampling Period	Monitor Placement	Avg. $PM_{2.5}$ Conc. ug/m^3 ± (sd)	Results and Cardiovascular Associations	Reference
Location	**Time**	**Occupation**	**Number**	**Mean Age/ Gender**	**Exclusions**	**Existing Conditions**					
North Carolina Highways, US	Sep.-Oct., 2001	Car-patrol officers	10	NS	None	NS	2 troopers monitored each day for four workdays (3pm-11: pm)	Personal (in patrol car)	23.0 (10.8)	The positive association with traffic volume around this relatively small urban center raises many questions about the future health effects from carbon fuel-burning vehicles for the people living near centers that grow in population and traffic density.	(Riediker et al. 2003)
Brazil Countryside	Oct.-Nov. 2007 and March-April 2008	Sugar and ethanol mill workers	28	31, Male	Clinical history or use of medication for CPD.	None	24hr continuous data collection	Personal	No mean given 87.0 (Median*) 70-100 (Range)	Results showed rest-to-peak diastolic blood pressure increased by 11.12 mmHg and 5.13 mmHg in the harvest and non-harvest period, respectively.	(Barbosa et al. 2012)
Wake County, NC	Fall, 2001	Highway patrol	9	27.3, Male	Smokers, Those who had taken alcohol, caffeine, or medication within 24hr of testing.	None	Monday Thursday 3pm-12am	Personal (in patrol car)	24.1 (13.5)	Copper, sulfur, aldehydes, calcium, and chromium or compounds containing these elements seem to be directly involved in the development of the inflammatory, coagulatory, and cardiac response to traffic particles in healthy young men	(Riediker 2007)
Locations in the US	Jan. 1996-2012	Aluminum industry workers	6058 Smelters 5623 Fabricators	44.3, Male	None	Current (<29.7%) or past (<34.1%) smokers	During Workday	Personal	Smelters: 198 (162) annually Fabricators: 34 (5.0) annually	There was an observed increased risk of incident IHD in relation to occupational exposure to PM in a prospective cohort study in the aluminum industry.	(Neophytou et al. 2016)
Hair Salons in Taipei, Taiwan	August-June 2009	Hairdresser assistants	62	25.3, Male and Female	Medication that affected cardiac rhythm, CVD or history of CVD.	None	12hr continuous monitoring from 9am-9pm	Personal	31.7 (10.4)	Occupational exposure to VOCs in hair salons can lead to increases in serum CRP levels and decreases in HRV indices.	(Ma et al. 2010)

Study Design		Participants					Sampling Period	Monitor Placement	Avg. $PM_{2.5}$ Conc. ug/m^3 ± (sd)	Results and Cardiovascular Associations	Reference
Location	**Time**	**Occupation**	**Number**	**Mean Age/ Gender**	**Exclusions**	**Existing Conditions**					
Shanghai, China	2009-2010	Traffic Policemen	110 (68 control)	Male 25-55 (range)	CPD, smokers, medications	None	24hr cycle from 8am-8am	Personal (or in car)	101.7 (46.8)	$PM_{2.5}$ exposure is associated with the increases in hs-CRP of 1.1%,	(Zhao et al. 2013)
Taipei Metropolitan Areas	2009-2012	Convenience Store Workers	60 (30 day shift and 30 night shift)	22.1, Male and Female	Smokers, CVD, Hypertension, any age above 30 or below 18.	None	8hrs during daytime or 8hrs during nighttime for 1-week intervals	Personal	Daytime: 26.5 (9.5) Night Time: 22.1 (4.3)	Observed the inverse association between indoor $PM_{2.5}$ exposures and HRV indices, with a decrease in all HRV indices with increased indoor $PM_{2.5}$ exposure	(Chuang, Chuang, and Lin 2013)

Terms: CPD- Cardiopulmonary Disease; CVD- Cardiovascular Disease; VOCs- Volatile Organic Compounds; CRP- C-Reactive Protein; IHD- Ischemic Heart Disease

4.0 Discussion

We reviewed articles that studied the association between occupational $PM_{2.5}$ exposure and their consequential impact on the human cardiovascular system. The goal of this study was to determine the degree of impact that $PM_{2.5}$ exposure has on the cardiovascular health of workers using average and median concentration data from 31 different case studies. Our results suggest that increasing $PM_{2.5}$ exposure is directly correlated with increasing risk of cardiovascular disease. The results also point to various side effects leading to cardiovascular disease including raised blood pressure, altered heart rate variability, and other autonomic disturbances in the body. These findings show potential for further research into $PM_{2.5}$ as a better understanding could help lower cardiovascular disease for workers.

4.1 Health Associations and Differences Among Studies

I hypothesized that increased occupational exposure to $PM_{2.5}$ would be positively correlated with increased risk of cardiovascular disease in workers. After analysis of all articles, this was found to be true in 93.5% of studies, with the other 6.5% of articles showing no correlation. Of the studies showing significant results, 38.0% were associated with decreased HRV, 17.2% with increased heart rate, 17.2% with increased risk of cardiovascular disease, 13.7% with alternating levels of HRV, 10.3% with increased arterial stiffness, 3.4% with increased blood pressure, and 3.4% with decreased cardiac output.

The associations between $PM_{2.5}$ exposure and HRV indices differed between case studies, making it difficult to make conclusive correlations. Out of all articles that found associations

with $PM_{2.5}$ and HRV, 73% showed a negative correlation, with 27% finding no definitive increase or decrease. This is likely due to the multivariable assessment of HRV which analyzes over seven different aspects of heart function, one of which being heart rate, and then combining all variables together to calculate an average total increase or decrease in HRV[24]. With 17.2% of studies finding increased heart rates in their experiments, it is possible and even likely that an increase in heart rate overshadowed decreased variables in other tested aspects of HRV, counterbalancing the total trend and resulting in an HRV that neither increased nor decreased. Another possible reason for these findings is the lack of total understanding of how HRV works and why it tends to fluctuate, as it has been shown to do so even under conditions without exposure to $PM_{2.5}$. From current research on HRV, the fluctuations are non-linear and seemingly rise and fall for reasons not yet known to science experts, suggesting that other factors contribute to the change in HRV and $PM_{2.5}$ may be another contributor[25]. Therefore, a greater demand for research and knowledge regarding the methodology of HRV indices need to be collected in order to fully understand its mechanisms.

A number of studies contained preexisting conditions (n=10) during the monitoring period, which could alter the associations and conclusions. 7 studies had participants who either were current smokers or had a history of smoking, although 1 study (Magari, S. R. et. al) compared smokers to nonsmokers and found different associations for both groups. 6 studies had instances of hypertension, with 2 of those studies alleviating the hypertension before monitoring. 3 studies had a history of cardiovascular disease, and 2 studies had respiratory disease or diabetes. In one study, (Cavallari, et. al.) showed that 33% (n=12) of all participants were smokers with 17.5% (n=6) having hypertension, potentially accounting for the decrease in HRV

[24] (Hoyer et al. 2019)
[25] (Shaffer and Ginsberg 2017)

and suggesting data that may be less consistent with other studies that had no preexisting conditions. Another study (Magari, et. al.) consisted of 51% (n=20) smokers, with another 15 participants having either hypertension or chronic bronchitis. This could potentially account for the increase in mean SDNN of HRV as the association was found due to increased levels of vanadium and lead concentrations in the $PM_{2.5}$ collection, and both metals are a byproduct of cigarette smoking[26]. Another study (Neophytou, A. M. et. al.) consisted of <29.7% of participants as current smokers and <34.1% as former smokers, potentially allowing for the association between increased $PM_{2.5}$ exposure and increased risk of IHD. One study (Chen, J. C. et. al.) noted an unspecified number of smokers, which may have affected the heart rate associations made by the study. One study (Eninger, R. S. et. al.) listed an unspecified number of participants that may have had diabetes. Previous research has shown that increased levels of long-term $PM_{2.5}$ exposure have been linked to increased risk of type 2 diabetes[27], and other research suggests $PM_{2.5}$ exposure is associated with diabetes due to factors such as increased heart rate and respiratory illness[28], but further research should be conducted to determine if diabetes has an effect on metallic $PM_{2.5}$ exposure intake. The remainder of the studies with preexisting conditions either removed those participants after monitoring (n=3) or were small enough in number to consider the study still viable (n=2). It should be noted that 4 studies did not specify if any preexisting conditions were present, and thus could have affected their associations.

[26] (Bernhard, Rossmann, and Wick 2005)
[27] (He et al. 2017)
[28] (Lao et al. 2019)

4.2 Variability in Monitoring Methods

A large majority of the studies reviewed used personal aerosol monitors to collect $PM_{2.5}$ exposure (n=28). It was considered personal monitoring if the participant's breathing zone was within 1 meter (1m) to the monitor at all times, and monitors placed inside vehicles within 1 meter of the driver were considered personal attachments as well. One study (Eninger, R. M. et. al.) placed a stationary monitor on top of the participants' toolboxes, allowing for periods that the worker was not always within 1m of the monitor. This could result in less accurate $PM_{2.5}$ collection and associated health endpoints due to the possibility of $PM_{2.5}$ concentration being lesser or greater at the site of the toolbox than where the worker was situated throughout the sampling period. Two studies (Byun, H. M. et. al; Hart, J. E. et. al.) placed monitors in a generalized area of the workspace, and thus the same potential errors as described could have resulted in altered HRV measures in the study. In order to connect exposure to cardiovascular impact, most studies used ambulatory electrocardiogram (ECG) monitors to assess heart rate and heart rate variability throughout the monitoring period. If an ECG was not used, a 5-Lead Holter monitor was used for precision monitoring of HRV. One study measuring CAVI (Wu, C. F. et. al.) placed a portable CAVI monitor (Vasera VS-1000) in a quiet room of the participants' office, and thus read arterial stiffness at numerous points but was not able to take continual measurements, or moving averages. Having numerous, continual averages help make sure that readings are consistent while also showing potential outliers in monitoring that would otherwise have been missed. With moving averages, this allows any drastic changes in measurement to be reevaluated to make sure collection is accurate. Out of all articles reviewed, it should be noted that only one study (Yannan, Z. et. al.) used a respirator as a potential intervention, and therefore

more research should be conducted before confirming the positive benefits that the study observed with respirators.

4.3 Limitations of the Study

Although many articles were able to contribute to the study, there were multiple limitations that made confirmation of our hypothesis challenging. The first limitation was the total number of available studies to be analyzed. With only 64 articles that dealt with associations between occupational exposure and cardiovascular effects, this field of study is more difficult to draw conclusions from. This had even greater limitations due to the fact that only 31 of the original 64 were viable for our study, as a large portion of the papers reviewed lacked actual $PM_{2.5}$ measurements (n=20). From the small pool of available literature, studies containing pre existing conditions may have skewed general associations, as those who already show increased risk of cardiovascular disease are often more affected by increased exposures to $PM_{2.5}$. Former research has suggested that preexisting conditions such as hypertension, diabetes, and high cholesterol contribute to higher risk of cardiovascular disease[29], a factor also seen in those with preexisting cardiovascular disease history such as heart failure or coronary artery disease[35]. Nevertheless, additional studies should therefore be conducted in order to determine the degree to which pre existing conditions affected this review.

Another limitation to the review was the participant number of some papers, as 3 studies had 10 or less participants being monitored. One study in particular (Eninger, R. M. et. al.) followed only 5 male workers, and only 1 of those subjects showed significant associations with

[29] ("Heart Disease Explained: Signs, Symptoms, and How to Reduce Your Risk" n.d.)
[35] ("UtahAir - Particulate Matter" n.d.)

$PM_{2.5}$ exposure, making it difficult to draw conclusions as a whole from the study. Another study (Riediker, M. et. al.) followed only 10 patrol car officers, but were each monitored four times during the study, increasing its verifiability in its associations. This is similar to the study with only 9 participants (Riediker, M.), as each worker was monitored from Monday-Thursday during the exposure assessment. In addition to participant number, the total $PM_{2.5}$ concentrations varied greatly between the studies. First, not all studies had average concentrations, instead listing a median and range as measurements (n=3), which limits the viability of those conclusions with this study. Second, 1 study in particular (Hart, J. E. et. al.) recorded annual average accumulations of $PM_{2.5}$ (4000ug/m^3) from 54,319 participants, making it difficult to determine the individual cardiac effects of the exposure assessment. The last limitation to our study is a lack of generalizability for both genders, as only 32% of studies monitored females (n=10). This leads us to suggest that any associations and conclusions made in the study should be further researched in order to assure that women are affected by $PM_{2.5}$ in the same way as participants are shown in this review.

The range of average collected $PM_{2.5}$ concentrations varied greatly over the 31 studies, from 1.92 (1.22) ug/m^3 to 4000 ug/m^3. Because many of the studies had $PM_{2.5}$ levels with large disparages between one another, it is also difficult to formulate associations and correlations for particular concentration levels. More research is needed in order to accurately be able to determine what averages of $PM_{2.5}$ concentration are most dangerous in the workplace.

Our results suggest that increased occupational exposure of various levels of $PM_{2.5}$ increases the risk of cardiovascular disease in workers. Having a better understanding of the effects of $PM_{2.5}$ can positively impact the safety of workers, especially those consistently

surrounded by smoke and byproducts in fumes, by establishing the potential need for additional regulations in order to prolong life in the workplace. Although intervention usage was nearly absent in this review, understanding the cardiovascular effects of $PM_{2.5}$ is advantageous for those who wish to incorporate interventions or learn more about its risks. Currently, no established limit for occupational $PM_{2.5}$ exposure has been established by the EPA. Thus, additional continuous and extensive research on the effects of $PM_{2.5}$ are needed in order to be able to make safe regulations for workers today.

5. Conclusion

Our study found nearly all reviewed papers to show significant, positive associations between increased occupational $PM_{2.5}$ exposure and cardiovascular health effects. This review highlights the hazardous potential that $PM_{2.5}$ exposure can have on the circulatory system, as well as the increased risk of cardiovascular disease in the workplace. Additional research on how $PM_{2.5}$ specifically affects factors such as HRV, blood pressure, and arterial disease could provide beneficial information that helps keep humans safe at work, and could lead to new discoveries in disease prevention in the near future.

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