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Understanding Air Pollution and Cardiovascular Diseases: Is It Preventable?

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Abstract

Fine particulate matter ($<2.5 \mu\text{m}$, $\text{PM}_{2.5}$) air pollution is a leading risk factor for morbidity and mortality worldwide. The largest portion of adverse health effects is from cardiovascular diseases. In North America, $\text{PM}_{2.5}$ concentrations have shown a steady decline over the past several decades; however, the opposite trend has occurred throughout much of the developing world whereby daily concentrations commonly reach extraordinarily high levels. While air quality regulations can reduce air pollution at a societal level, what individuals can do to reduce their personal exposures remains an active field of investigation. Here, we review the emerging evidence that several interventions (e.g., air filters) and/or behavioral changes can lower PM pollution exposure and as such, may be capable of mitigating the ensuing adverse cardiovascular health consequences. Air pollution remains a worldwide epidemic and a multi-tiered prevention strategy is required in order to optimally protect global public health.

Keywords

Air pollution; Interventions; Cardiovascular disease risks

Introduction

In the modern world, particulate matter $<2.5 \mu\text{m}$ in aerodynamic diameter ($\text{PM}_{2.5}$) air pollution is principally a consequence of the fossil fuel dependence (e.g., coal-fired power plants) of contemporary urban-industrial societies. $\text{PM}_{2.5}$ is an amalgam of solid and liquid combustion by-products including organic and elemental carbon, inorganic ions (sulfates, nitrates), and a host of metals/elements (e.g., iron) [1, 2, 3•]. As with cigarette smoking, the inhalation of these particles is clearly linked to adverse pulmonary health effects [1, 2, 3•]. However, the evidence accrued over the past few decades demonstrates that in fact, the

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largest portion of attributable deaths are due to cardiovascular causes [1, 2, 3••]. A wide array of epidemiological studies demonstrates that short-term exposures over a few days increases the risk for a variety of acute cardiovascular events (e.g., myocardial infarctions, heart failure exacerbations, and strokes) by about 1–2 % per 10 $\mu\text{g}/\text{m}^3$ (i.e., approximately 1 standard deviation $\text{PM}_{2.5}$ increase in the western world) [4–6]. Living in more polluted regions over several years increases cardiovascular morbidity and mortality by an even larger degree (~10 % increase per 10 $\mu\text{g}/\text{m}^3$) [7, 8]. Given the billions of people affected, the public health consequences are enormous. $\text{PM}_{2.5}$ ranks as the ninth leading risk factor for morbidity and mortality worldwide [9].

Several mechanistic pathways explaining the epidemiological associations have been demonstrated which strongly support that $\text{PM}_{2.5}$ is causally linked to cardiovascular diseases [1, 2, 3••]. $\text{PM}_{2.5}$ inhalation is capable of triggering systemic oxidative stress/inflammation and altering autonomic balance in favor of sympathetic activation. Certain nanoparticles or their constituents may even translocate into circulation and thereby directly harm the cardiovascular system. As a result, a variety of adverse physiological responses may ensue within hours- to-days including vasoconstriction, endothelial dysfunction, increased blood pressure (BP) and heart rate, myocardial ischemia, impaired heart rate variability (HRV), repolarization abnormalities, arrhythmias, and enhanced thrombotic and coagulation potential. Longer-term exposures have been linked to the chronic progression of atherosclerosis as well as the increased incidence of overt hypertension and diabetes mellitus [1, 2, 3••]. Both the American Heart Association and the European Society of Cardiology have reviewed the epidemiological and experimental studies and formally recognize $\text{PM}_{2.5}$ as an important cardiovascular risk factor [1,2, 3••].

Global Air Pollution Levels

Recent reports estimate that roughly 75 % of the world's population is exposed to $\text{PM}_{2.5}$ levels above the World Health Organization (WHO) annual air quality standards ($>10 \mu\text{g}/\text{m}^3$) [10]. While global concentrations continue to increase (mean level of approximately 20–25 $\mu\text{g}/\text{m}^3$), air pollution has steadily declined in North America since the 1970s to current values averaging around 10–13 $\mu\text{g}/\text{m}^3$ (with only 20 % of the population above WHO annual goals) [10]. On the other hand, many developing countries across Asia suffer from tremendously high levels averaging 45 $\mu\text{g}/\text{m}^3$ annually. In these countries, over 99 % of the populace is exposed to pollution above WHO standards. Daily concentrations commonly reach extraordinarily dangerous thresholds exceeding 150–500 $\mu\text{g}/\text{m}^3$ in certain highly polluted regions [10, 11]. It is important to note, however, that even the low pollution levels encountered throughout most of North America ($<15 \mu\text{g}/\text{m}^3$) are still linked to an increased risk for cardiovascular events [7].

Compounding the adverse effects of outdoor “ambient” $\text{PM}_{2.5}$ is the potentially even larger global public health threat of household air pollution (HAP) [12–14]. More than 3 billion people (particularly in regions across India, China, and Africa) rely on solid fuels (e.g., biomass) for household heating and cooking [12–14]. Indoor particle concentrations due to HAP commonly exceed those of secondhand smoke [13], ranging from 100–500 $\mu\text{g}/\text{m}^3$. While fewer studies have directly evaluated the cardiovascular health effects of wood

smoke, biomass, or other household sources of pollution (e.g., cooking stoves), most recent estimates support that HAP is the third leading cause of disability-adjusted life years lost worldwide due to an excess of both pulmonary and cardiovascular diseases [9]. As such, in order to optimally protect the global public health, efforts to reduce exposures to both ambient PM_{2.5} as well as HAP (at least in many developing regions) are both important.

Health Effects of Lowering Ambient PM_{2.5} at a Societal Level

Several “natural experiments” provided the first evidence that reductions in air pollutants can yield observable health benefits within a short period of time. A ban on coal sales in Dublin Ireland in 1990 led to a 70 % (36 µg/m³) reduction in black smoke within 72 months [15], and deaths from cardiovascular causes significantly decreased by 10.3 % (243 fewer deaths/year) in this period. An earlier study in Utah valley showed that a 13-month strike at a steel mill led to a 15 µg/m³ reduction in PM₁₀, while all-cause mortality decreased by 3.2 % [16]. Restrictions on the sulfur content of fuel in Hong Kong in 1990 [17] (45 % reduction in SO₂) and a nation-wide US copper smelter strike in 1967 [18] (2.5 µg/m³ reduction in regional sulfate) also led to significant mortality benefits within a short period of time.

The health impact of country-wide changes in air pollution levels over longer periods of time have also been explored. This has been possible because PM_{2.5} concentrations have fallen during the past three decades in the USA. This is largely a result of the institution of the Clean Air Act from the US Environmental Protection Agency (EPA) in the early 1970s [19]. Since that time, National Ambient Air Quality Standards (NAAQS) have been further strengthened on several occasions. Aggregate emissions of all air pollutants (particles and gases) have fallen by 68 %. This trend continues as even from 2000 to 2013 annual average PM_{2.5} levels have been further reduced by 34 % [20]. Follow-up analyses of the Harvard Six Cities Study have demonstrated that this national reduction in pollution has translated into substantial health benefits [21, 22]. From the early 1970s to the late 1990s, a 10 µg/m³ reduction in estimated PM_{2.5} levels among the six cities led to a 27 and 31 % reduction in adjusted all-cause and cardiovascular mortality rates, respectively [21]. Even larger analyses of life expectancy increases attributable to nation-wide reductions in PM_{2.5} across the USA from 1980 to 2000 [23] and from 2000 to 2007 [24] also support that the long-term trend for improvements in air quality translates into observable health benefits. During both periods, reductions in PM_{2.5} levels by 10 µg/m³ were independently associated with increases in life expectancy by between 4 and 7 months.

The recent 2008 Beijing Olympics offered a unique opportunity to evaluate the potential health effects of air pollution reductions [25••]. During the games (July–September) several pollution control measures led to significant decreases in both particulate and gaseous pollutants. Mean levels of PM_{2.5} were 100.9 and 84.2 µg/m³ before and afterward, while they averaged 69.4 µg/m³ during the Olympic months. Several bio-markers of inflammation and thrombosis, as well as BP, were not only associated with various gaseous and particle pollutant levels during the previous few days, but they were improved during the Olympic period. This study shows that even among healthy individuals, wide-scale interventions to

improve air quality can positively influence cardiovascular health within a short period of time.

Interventions to Reduce Air Pollution Exposures at a Personal Level

It is an encouraging fact that most air pollutants have fallen considerably in the USA over the past few decades. Unfortunately, even low levels of PM_{2.5} (5–15 µg/m³) are still associated with significant cardiovascular health risks. Epidemiological studies demonstrate that no lower “safe” threshold of exposure seems to exist at the population level [7, 22]. Moreover, pollution levels remain high and are increasing for more than half of the world’s population [10]. Even if national regulations help to reduce outdoor ambient concentrations, billions of people still face the health risks posed by HAP and/or high exposures due to “hot spots” (e.g., near roadways, industrial settings) of emissions even when overall regional background average levels remain controlled within air quality standards.

An international multidisciplinary workshop reported that interventions which separate people from pollution, thereby reducing exposure and mitigating health impacts, have been largely overlooked as components of formal strategies [26]. This raises important questions, including: what can any single person do to help reduce their exposure? Are any of these measures feasible and/or effective in mitigating the ensuing negative health impacts? In the following section, we review the available evidence showing that interventions can reduce PM_{2.5} exposure and effect improvements in cardiovascular health. Findings in this section are summarized in Table 1.

Air Filtration

There is strong evidence that portable air filtration systems can reduce indoor PM_{2.5} concentrations [27, 28]. For example, Barn et al. reported that high-efficiency particulate air (HEPA) filtration effectiveness for forest fires and other wood smoke during summer was 65 ± 35 % [28]. Henderson et al. also reported a PM_{2.5} decrease of 63–88 % in homes during wildfires and prescribed burns [27]. More importantly, several recent studies have shown significant cardiovascular health benefits can be realized through the reduction of exposure to PM_{2.5} while indoors by HEPA filtration [29,30,31]. Weichenthal et al. reported that electrostatic filtration use was associated with small decreases in systolic BP and diastolic BP [32]. On the other hand, Karottki et al. reported mixed results due to low PM_{2.5} levels in the absence of filtration, along with possible confounding due to subjects’ use of medication [33]. A limitation of this general strategy is that while most individuals indeed spend the majority of their time at home and indoors (which makes this approach even feasible), air filters will not reduce exposures encountered outside of this setting. Given the great potential for air filtration to improve health and protect susceptible populations and those spending most of their time indoors (e.g., retirement communities), the utility of air filtration remains an important area for future research.

Closing Windows and Air Conditioning

In a study of young adults in Taipei, Lin et al. reported that closing windows reduced indoor PM concentrations and modified the effect of PM₁₀ on BP and heart rate [34]. In a

subsequent study, Lin et al. also reported that closing windows and turning on air conditioners at home can reduce indoor air pollution and improve cardiovascular endpoints (e.g., HRV and C-reactive protein in plasma) [35•]. Furthermore, another recent study reported that operation of a car's air conditioning system during commutes modified the associations between in-vehicle PM_{2.5} and decreases in HRV [36]. Bell et al. examined PM_{2.5} and risk of urgent cardiovascular hospitalizations for persons 65 years and older in 168 US counties from 1999 to 2005, and reported that communities with higher air conditioning prevalence exhibited a reduced association between outdoor PM_{2.5} and cardiovascular-related hospital admissions [37]. However, it is not currently clear whether increased air conditioning usage would provide an *overall* health benefit to the population in regards to air pollution exposures given the associated need for heightened electrical power generation [38].

Wearing Face Masks

In Beijing, China, using a particulate respirator face mask appeared to effectively reduce symptoms and improve cardiovascular health measures in patients with coronary heart disease [39•], and to reduce adverse effects of air pollution on BP and HRV in young health subjects [40]. However, the authors noted that these study were not double blinded, and so the findings may be biased by subjects' awareness of whether they were wearing a respirator or not. Another study reported that markers of oxidative stress (nitrite and nitrite + nitrate) in exhaled breath condensate were increased immediately after 1.5-h car rides during which subjects breathed unfiltered vehicle cabin air, but not after rides during which subjects breathed HEPA-filtered air via a powered air purifying respirator [41]. Unfortunately, under most daily life scenarios, wearing face masks is not a long-term practical strategy; however, it may be useful for short periods during extreme exposures (e.g., high-risk individuals during travel to polluted regions, occupational exposures, activities near hot spots such as roadways).

Car Filters and Particle Traps

Recently, Pui et al. demonstrated that air recirculation using relatively inexpensive filters can substantially and rapidly reduce exposure to airborne nanoparticles within enclosed spaces [42]. This is a key finding as relatively inexpensive low-efficiency filters could provide an economical and easily implemented method to effectively protect people from traffic PM exposure. Another study documented that the use of exhaust particulate traps on diesel-powered vehicles is a highly efficient method of reducing particle exposure and providing beneficial effects on biomarkers of cardiovascular health [43••]. However, Muala et al. reported that while exhaust-induced symptoms reported by subjects decreased, the use of cabin air inlet filters including activated charcoal did not show any significant changes in any inflammatory markers in the peripheral blood samples 5 h post exposure [44]. Overall, results suggest that cabin filters and/or diesel particle traps can reduce exposure to traffic-related air pollutants and can be effective in reducing associated adverse cardiovascular health changes.

Modified Cook Stoves

Clark et al. reported that the use of an Eco-stove—a wood-burning cook stove with a more efficient combustion chamber and a chimney—resulted in a 5.9-mmHg reduction in systolic BP among women over 40 years of age, and a 4.6-mmHg reduction among obese women in Granada, Nicaragua [45]. Another intervention study in Guatemala by McCracken et al. reported on associations between exposure to wood smoke from household stoves and electrocardiographic [46] as well as BP changes [47]. The authors found that compared to open fire, a chimney stove intervention was associated with reduced occurrence of nonspecific ST-segment depression [46] and lower BP [47]. Given that “solid-fuel-fired cooking and heating stoves are used in more than half the world’s households and have been shown in many locations to produce high indoor concentrations of particulates and other combustion-related pollutants,” individual-level interventions to reduce these exposures are important. Major international initiatives such as the Global Alliance for Clean Cookstoves (<http://cleancookstoves.org/>) are underway to help reduce the worldwide public health burden due to indoor and HAP.

Other Behavioral Changes

Lissåker et al. found that certain groups including the elderly and those with either respiratory or cardiovascular disease are more likely to change behaviors when they are aware of poor air quality [48]. This suggests that some of the most susceptible groups do, in fact, take measures to protect themselves against air pollution; unfortunately, the authors also noted that most people did not alter their behavior. As such, there is still an urgent need to better educate the public about the health risks of air pollution.

The Detroit “RAPIDS” Study

In order to effectively implement interventions that separate people from pollution, a greater understanding of exposure at the community and personal level is needed. Moreover, limited research is currently available documenting the effects of specific personal-level interventions on clinically relevant cardiovascular outcomes among susceptible populations in urban air sheds where both stationary (e.g., iron/steel manufacturers, incinerators) and mobile sources are present. With that in mind, we are currently conducting a randomized blinded crossover intervention study of the effect of air filtration (sham filter [placebo] versus low-efficiency (LE) versus high-efficiency (HE) air filter systems) on repeated cardiovascular health and exposure measurements among senior citizens in the Reducing Air Pollution in Detroit Intervention Study (RAPIDS). Data is being collected in a low-income senior citizen facility located in downtown urban Detroit, Michigan, in an area where we have previously linked outdoor PM_{2.5} to adverse cardiovascular health effects including increased BP and arterial vasoconstriction. This study is designed to address three specific gaps in the existing data by (1) assessing the effectiveness of economical LE filters as well as HE filters for improving indoor air quality and cardiovascular health endpoints, (2) understanding cardiovascular health effects of outdoor/indoor/personal PM air pollution on senior citizens living in urban environments, and (3) identifying which sources and their associated cardiovascular health effects are most responsive to filtration. Preliminary data validate the relevance and importance of this intervention study as (1) the average personal

PM_{2.5} level without any filtration (i.e., control) was 23.9±19.5 µg/m³, which is about twice the annual NAAQS for PM_{2.5}, and (2) both LE and HE air filtration reduced personal PM_{2.5} levels by 28±6 and 60±27 %, respectively. In conjunction with the compelling evidence from our previous studies in the Detroit area which showed that ambient and personal PM_{2.5} exposures are significantly associated with elevated BP levels within 1–3 days after exposure (e.g., for personal-level exposures systolic BP increased 1.4 mmHg per 10 µg/m³ of PM_{2.5}) [49], we expect our study to confirm that air filtration can significantly reduce PM_{2.5}-induced BP elevations and other cardiovascular health endpoints.

Conclusions

PM_{2.5} is an important global risk factor for cardiovascular morbidity and mortality. Both large-scale national as well as personal-level interventions have the potential to reduce air pollution exposures and the ensuing adverse cardiovascular health effects. Personal measures (e.g., air filters) may be of particular importance in developing nations where ambient pollutant levels remain high and resources to reduce emissions are limited. Numerous study factors including the participant characteristics (e.g., young versus elderly), residential locations (e.g., urban versus rural), pollution sources and levels (e.g., wild fires versus traffic), and underlying health issues or susceptibility (e.g., lung or heart diseases) can influence the effectiveness of the studied interventions. In this regard, future studies are required to improve our understanding of how personal-level interventions perform under a variety of conditions and among different populations in order to make optimal use of the various strategies in the global effort to reduce the adverse public health effects of air pollution.

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References

Papers of particular interest, published recently, have been highlighted as:

• Of importance

•• Of major importance

1. Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, et al. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*. 2004; 109:2655–2671. [PubMed: 15173049]
2. Brook RD, Rajagopalan S, Pope CA III, Brook JR, Bhatnagar A, Diez-Roux A, et al. Particulate matter air pollution and cardiovascular disease. An update to the scientific statement from the American Heart Association. *Circulation*. 2010; 121:2331–2378. [PubMed: 20458016]
3. Newby DE, Mannucci PM, Tell GS, Baccarelli AA, Brook RD, Donaldson K, et al. Expert position paper on air pollution and cardiovascular disease. *Eur Heart J*. 2015; 36:83–93. [PubMed: 25492627] This is the latest updated review of the cardiovascular health effects of air pollution.
4. Mustafic H, Jabre P, Caussin C, Murad MH, Escolano S, Tafflet M, et al. Main air pollutants and myocardial infarction: a systematic review and meta-analysis. *JAMA*. 2012; 307:713–721. [PubMed: 22337682]

5. Yu XB, Su JW, Li XY, Chen G. Short-term effects of particulate matter on stroke attack: meta-regression and meta-analyses. *PLoS One*. 2014; 9:e95682. [PubMed: 24802512]
6. Shah AS, Langrish JP, Nair H, McAllister DA, Hunter AL, Donaldson K, et al. Global association of air pollution and heart failure: a systematic review and meta-analysis. *Lancet*. 2013; 382:1039–1048. [PubMed: 23849322]
7. Crouse DL, Peters PA, van Donkelaar A, Goldberg MS, Villeneuve PJ, Brion O, et al. Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. *Environ Health Perspect*. 2012; 120:708–714. [PubMed: 22313724]
8. Hoek G, Krishnan RM, Beelen R, Peters A, Ostro B, Brunekreef B, et al. Long-term air pollution exposure and cardio-respiratory mortality: a review. *Environ Health*. 2013; 12:43. [PubMed: 23714370]
9. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet*. 2012; 380:2224–2260. [PubMed: 23245609]
10. Van Donkelaar A, Martin RV, Brauer M, Boys BL. Use of satellite observations for long-term exposure assessment of global concentrations of fine particulate matter. *Environ Health Perspect*. 2015; 123:135–143. [PubMed: 25343779]
11. Zhao X, Sun Z, Ruan Y, Yan J, Mukherjee B, Yang F, et al. Personal black carbon exposure influences ambulatory blood pressure. Air pollution and cardiometabolic disease (AIRCMD- China) study. *Hypertension*. 2014; 63:871–877. [PubMed: 24420543]
12. Rajagopalan S, Brook RD. The indoor-outdoor air-pollution continuum and the burden of cardiovascular disease: An opportunity for improving global health. *Glob Heart*. 2012; 7:207–213. [PubMed: 23181202]
13. Smith KR, Peel JL. Mind the gap. *Environ Health Perspect*. 2010; 118:1643–1645. [PubMed: 20729177]
14. Balakrishnan K, Ramaswamy P, Sambandam S, Thangavel G, Ghosh S, Johnson P, et al. Air pollution from household solid fuel combustion in India: an overview of exposure and health related information to inform health research priorities. *Glob Health Action*. 2011; 4
15. Clancy L, Goodman P, Sinclair H, Dockery DW. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. *Lancet*. 2002; 360:1210–1214. [PubMed: 12401247]
16. Pope CA III, Schwartz J, Ransom MR. Daily mortality and PM₁₀ pollution in Utah Valley. *Arch Environ Health*. 1992; 47:211–217. [PubMed: 1596104]
17. Hedley AJ, Wong CM, Thach TQ, Ma S, Lam TH, Andderson HR. Cardiorespiratory and all-cause mortality after restrictions on sulphur content of fuel in Hong Kong: an intervention study. *Lancet*. 2002; 360:1646–1652. [PubMed: 12457788]
18. Pope CA III, Rodemund DL, Gee MM. Mortality effects of a copper smelter strike and reduced ambient sulfate particulate matter air pollution. *Environ Health Perspect*. 2007; 115:679–683. [PubMed: 17520052]
19. [Accessed 21 Feb 2015] <http://www.epa.gov/air/criteria.html>
20. [Accessed 21 Feb 2015] <http://epa.gov/airtrends/aqtrends.html>
21. Laden F, Schwartz J, Speizer FE, Dockery DW. Reduction in fine particulate air pollution and mortality. Extended follow-up of the Harvard Six Cities Study. *Am J Respir Crit Care Med*. 2006; 173:667–672. [PubMed: 16424447]
22. Lepeule J, Laden F, Dockery D, Schwartz J. Chronic exposure to fine particles and mortality: an extended follow-up of the Harvard Six Cities Study from 1974 to 2009. *Environ Health Perspect*. 2012; 120:965–970. [PubMed: 22456598]
23. Pope CA III, Ezzati M, Dockery DW. Fine-particulate air pollution and life expectancy in the United States. *N Engl J Med*. 2009; 360:376–386. [PubMed: 19164188]
24. Correia AW, Pope CA III, Dockery DW, Wang Y, Ezzati M, Dominici F. Effect of air pollution control on life expectancy in the United States. An analysis of 545 U.S. counties for the period from 2000 to 2007. *Epidemiology*.

25. Rich DQ, Kipen HM, Huang W, Wang G, Wang Y, Zhu P, et al. Association between changes in air pollution levels during the Beijing Olympics and biomarkers of inflammation and thrombosis in healthy young adults. *JAMA*. 2012; 307:2068–2078. [PubMed: 22665106] This study demonstrates that policy efforts to reduce ambient air pollutants at a city-wide scale can rapidly improve markers of cardiovascular health.
26. Giles LV, Barn P, Kunzli N, Romieu I, Mittleman MA, van Eeden S, et al. From good intentions to proven interventions: effectiveness of actions to reduce the health impacts of air pollution. *Environ Health Perspect*. 2011; 119:29–36. [PubMed: 20729178]
27. Henderson DE, Milford JB, Miller SL. Prescribed burns and wildfires in Colorado: impacts of mitigation measures on indoor air particulate matter. *J Air Waste Manag Assoc*. 2005; 55:1516–1526. [PubMed: 16295277]
28. Barn P, Larson T, Noullett M, Kennedy S, Copes R, Brauer M. Infiltration of forest fire and residential wood smoke: an evaluation of air cleaner effectiveness. *J Expo Sci Environ Epidemiol*. 2008; 18:503–511. [PubMed: 18059421]
29. Bräuner EV, Forchhammer L, Møller P, Barregard L, Gunnarsen L, Afshari A, et al. Indoor particles affect vascular function in the aged: an air filtration-based intervention study. *Am J Respir Crit Care Med*. 2008; 177:419–425. [PubMed: 17932377]
30. Allen RW, Carlsten C, Karlen B, Leckie S, van Eeden S, Vedal S, et al. An air filter intervention study of endothelial function among healthy adults in a woodsmoke-impacted community. *Am J Respir Crit Care Med*. 2011; 183:1222–1230. [PubMed: 21257787] This study shows that an indoor air filter can improve endothelial function within a few days.
31. Lin LY, Chen HW, Su TL, Hong GB, Huang LC, Chuang KJ. The effects of indoor particle exposure on blood pressure and heart rate among young adults: an air filtration-based intervention study. *Atmos Environ*. 2011; 45:5540–5544.
32. Weichenthal S, Mallach G, Kulka R, Black A, Wheeler A, You H, et al. A randomized double-blind crossover study of indoor air filtration and acute changes in cardiorespiratory health in a First Nations community. *Indoor Air*. 2013; 3:175–184. [PubMed: 23210563]
33. Karotki DG, Spilak M, Frederiksen M, Gunnarsen L, Brauner EV, Kolarik B, et al. An indoor air filtration study in homes of elderly: cardiovascular and respiratory effects of exposure to particulate matter. *Environ Health*. 2013; 12:116. [PubMed: 24373585]
34. Lin LY, Lin CY, Lin YC, Chuang KJ. The effects of indoor particles on blood pressure and heart rate among young adults in Taipei, Taiwan. *Indoor Air*. 2009; 19:482–488. [PubMed: 19682103]
35. Lin LY, Chuang HC, Liu IJ, Chen HW, Chuang KJ. Reducing indoor air pollution by air conditioning is associated with improvements in cardiovascular health among the general population. *Sci Total Environ*. 2013; 463:176–181. [PubMed: 23803502] This study shows that closing windows and using air conditioning can lower blood pressure among people living in a highly-polluted city.
36. Chuang HC, Lin LY, Hsu YW, Ma CM, Chuang KJ. In-car particles and cardiovascular health: an air conditioning-based intervention study. *Sci Total Environ*. 2013; 452:309–313. [PubMed: 23523729]
37. Bell ML, Ebisu K, Peng RD, Dominici F. Adverse health effects of particulate air pollution: modification by air conditioning. *Epidemiology*. 2009; 20:682–686. [PubMed: 19535984]
38. Vedal S. Does air conditioning modify the health effects of exposure to outdoor air pollution? *Epidemiology*. 2009; 20:687–688. [PubMed: 19680037]
39. Langrish JP, Li X, Wang S, Lee MM, Barnes GD, Miller MR, et al. Reducing personal exposure to particulate air pollution improves cardiovascular health in patients with coronary heart disease. *Environ Health Perspect*. 2012; 120:367–372. [PubMed: 22389220] This study shows that wearing a facemask can prevent myocardial ischemia and lead to lower blood pressure levels during walking in a highly-polluted city.
40. Langrish JP, Mills NL, Chan JK, Leseman DL, Aitken RJ, Fokkens PH, et al. Beneficial cardiovascular effects of reducing exposure to particulate air pollution with a simple facemask. *Part Fibre Toxicol*. 2009; 6:8. [PubMed: 19284642]

41. Laumbach RJ, Kipen HM, Ko S, Kelly-McNeil K, Cepeda C, Pettit A, et al. A controlled trial of acute effects of human exposure to traffic particles on pulmonary oxidative stress and heart rate variability. *Part Fibre Toxicol.* 2014; 11:45. [PubMed: 25361615]
42. Pui DY, Qi C, Stanley N, Oberdörster G, Maynard A. Recirculating air filtration significantly reduces exposure to airborne nanoparticles. *Environ Health Perspect.* 2008; 116:863–866. [PubMed: 18629306]
43. Lucking AJ, Lundbäck M, Barath SL, Mills NL, Sidhu MK, Langrish JP, et al. Particle traps prevent adverse vascular and prothrombotic effects of diesel engine exhaust inhalation in men. *Circulation.* 2011; 123:1721–1728. [PubMed: 21482966] This study provides strong evidence that it is the particulate matter portion of air pollution that plays a role in causing endothelial dysfunction and adverse cardiovascular responses. Diesel particle traps are able to reduce particulate exposures and mitigate the harmful effects of exposures.
44. Muala A, Sehlstedt M, Bion A, Osterlund C, Bosson JA, Behndig AF, et al. Assessment of the capacity of vehicle cabin air inlet filters to reduce diesel exhaust-induced symptoms in human volunteers. *Environ Health.* 2014; 13:16. [PubMed: 24621126]
45. Clark ML, Bachand AM, Heiderscheidt JM, Yoder SA, Luna B, Volckens J, et al. Impact of a cleaner-burning cookstove intervention on blood pressure in Nicaraguan women. *Indoor Air.* 2013; 23:105–114. [PubMed: 22913364]
46. McCracken J, Smith KR, Stone P, Díaz A, Arana B, Schwartz J. Intervention to lower household wood smoke exposure in Guatemala reduces ST-segment depression on electrocardiograms. *Environ Health Perspect.* 2011; 119:1562–1568. [PubMed: 21669557]
47. McCracken JP, Smith KR, Díaz A, Mittleman MA, Schwartz J. Chimney stove intervention to reduce long-term wood smoke exposure lowers blood pressure among Guatemalan women. *Environ Health Perspect.* 2007; 115:996–1001. [PubMed: 17637912]
48. Lissåker C, Talbott EO, Kan H, Xu X. Status and determinants of individual actions to reduce health impacts of air pollution in U.S. adults. *Arch Environ Occup Health.* 2014
49. Brook RD, Bard RL, Burnett RT, Shin HH, Vette A, Croghan C, et al. Differences in blood pressure and vascular responses associated with ambient fine particulate matter exposures measured at the personal versus community level. *Occup Environ Med.* 2011; 68:224–230. [PubMed: 20935292]

Table 1
Studies examining PM exposure interventions and improvements in cardiovascular health markers

Authors	Population	Intervention	Exposure metrics	Measured health outcomes	Major findings
Brauner [29]	Nonsmoking elderly (60–75 years) in urban Copenhagen, <i>N</i> = 41 (21 couples, 1 person excluded)	Double blind random crossover with 48 h of either HEPA or sham filtration (filtration units placed in bedroom and living room)	Indoor PM _{2.5} and PM _{10-2.5} mass, particle number concentration (PNC) (particles between 10–700 nm diameter), elemental composition	Microvascular function (MVF), biomarkers in blood and urine, BP	HEPA filter use significantly improved MVF by 8.1 % (95 % CI=0.4–16.3 %); indoor PM _{2.5} mass significantly associated with MVF; significant associations between increases in iron, copper, potassium, zinc, lead, and arsenic concentrations in PM _{2.5} and reduced MVF
Allen [30]	Healthy adult nonsmokers (<i>N</i> = 45) in a Canadian community impacted by residential wood combustion	Randomized crossover intervention study with 7 days of either HEPA or sham filtration (filtration units running in living room and bedroom)	Ambient and indoor PM _{2.5} mass, concentration of wood smoke tracer levoglucosan	Reactive hyperemia index (RHI), inflammatory markers (C-reactive protein (CRP), band cells, interleukin-6), markers of oxidative stress (malondialdehyde, 8-iso-prostaglandin F _{2α})	Air filtration resulted in 60 % reduction of indoor PM _{2.5} mass, 75 % reduction of levoglucosan, 9.4 % (95 % CI=0.9–18 %) increase in RHI and 32.6 % (95 % CI=4.4–60.9 %) decrease in CRP; air filtration was associated with improved endothelial function and decreased concentrations of inflammatory biomarkers but not markers of oxidative stress
Lin [31]	Healthy young students (<i>N</i> = 60) in Taipei, Taiwan	Air conditioner with non-HEPA filter for 2 visits, air conditioner without filter for 2 visits	Indoor PM _{2.5} mass, volatile organic compounds (VOCs)	Systolic BP (SBP), diastolic BP (DBP), Heart rate (HR)	Adverse effects of indoor PM _{2.5} on raising BP and HR were greatest during visits without filtration and mitigated by filters
Weichen- thal [32]	Adult residents (<i>N</i> =37) in a First Nations Community in Manitoba, Canada	Randomized crossover study using electrostatic filtration in main living area with 1 week each of active filtration and placebo filtration	Integrated weekly avg of indoor PM ₁₀ , PM _{2.5} , PM ₁ mass; and PAH, VOCs, and NO ₂	RHI, SBP, DBP, lung function by spirometry	Air filter use was associated with small decreases in BP, and with significant improvements in forced expiratory volume in 1 s (FEV1) and peak expiratory flow rate (PEFR)
Karotki [33]	Nonsmoking elderly (<i>N</i> = 48, 51–81 year) in urban Copenhagen	Double blind random crossover study with 2 weeks of either HEPA or sham filtration (filtration units placed in bedroom and living room)	Indoor PM _{2.5} mass, PNC (particles between 10 and 300 nm diameter), PAH, black carbon	BP, MVF, lung function by spirometry, biomarkers of systemic inflammation in blood	No significant effect on MVF or lung function; MVF of subjects not taking vasoactive medication significantly improved with reduction in bedroom PM _{2.5}
Lin [34]	Healthy young adults (<i>N</i> = 40) in Taipei, Taiwan	Windows open for 2 48-h periods followed by windows closed for 2 48-h periods	Indoor PM _{2.5} and PM ₁₀ mass; temp, humidity, NO ₂ during waking hours	BP and HR continuously monitored during waking hours	Open windows associated with increased PM ₁₀ , PM _{2.5} BP, and HR
Lin [35]	Healthy nonsmoking adults (<i>N</i> = 300, 20–65 years old) in Taipei, Taiwan	3 conditions: (1) windows open; (2) windows closed, air conditioner off; (3) windows closed, air conditioner on Each condition repeated 2 × for 24 h	Indoor PM ₁₀ and PM _{2.5} mass, CO, CO ₂ , VOCs, noise	ECG, blood samples tested for high sensitivity (hs)-CRP, 8-hydroxy-2'-deoxyguanosine (8-OHdG), fibrinogen	Indoor PM _{2.5} mass and VOCs were associated with increased hs-CRP, 8-OHdG and fibrinogen, and decreased HRV indices; closed windows and air

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		each			conditioners were determined to be effect modifiers; open windows had the greatest negative impact on CV endpoints
Chuang [36]	Healthy adults ($N = 60$, 20–50 years old) in Taipei, Taiwan	2-h car trip in morning traffic under each of three conditions: (1) Air conditioner off and windows open, (2) OA mode with ambient air being brought into the car, (3) IA mode with air recirculating throughout the car	In-car $PM_{2.5}$ mass, noise	HRV indices	Increased levels of $PM_{2.5}$ were associated with decreased HRV
Langrish [39]	Nonsmoking coronary heart disease patients ($N = 98$) in Beijing, China	Open randomized crossover study in which: 1 Subjects wore mask for 48 h (starting 24 h before study period) and walked a 2-h outdoor course 2 Subjects w/o mask walked same course	Ambient $PM_{2.5}$ mass, personal $PM_{2.5}$ mass (recorded with pDR and estimated using 97 % mask efficiency), personal PNC, CO, NO ₂ , SO ₂	electrocardiography (ECG), BP, HRV, symptom survey	Mask use associated with reduced maximal ST-segment depression, reduced self-reported symptoms (over the 24-h period), reduced mean arterial BP, and increased HRV (during the 2-h walk)
Langrish [40]	Healthy nonsmoking adults ($N = 15$, median age 28 years) in Beijing, China	Open randomized crossover study in which: 1 Subjects wore mask for 48 h (starting 24 h before study period) and walked a 2-h outdoor course 2 Subjects w/o mask walked same course	Ambient $PM_{2.5}$ mass, personal $PM_{2.5}$ mass (recorded with pDR and estimated using 97 % mask efficiency), personal PNC, CO, NO ₂ , SO ₂	ECG, BP, HRV, symptom survey	Mask use associated with statistically significant decrease in SBP (during the 2-h walk) and increase in HRV (over the 24-h period)
Laumbach [41]	Young adults ($N = 21$, mean age 22 years) near Rutgers University	Randomized crossover study with 1.5-h car rides during traffic under two conditions: 1 wearing respirator with HEPA filter 2 wearing respirator without a filter	PNC and $PM_{2.5}$ mass measured inside the vehicle and respirator	HRV, concentrations of nitrate, nitrite + nitrate, malondialdehyde, 8-isoprostane in exhaled breath condensate (markers of oxidative stress)	Significant increase in PNC associated with increased nitrite and nitrite + nitrate; mean PNC inside respirator was 99.99 % lower with HEPA filtration than with no filter; mean $PM_{2.5}$ mass inside respirator was also lower with HEPA filtration (1.4 $\mu\text{g}/\text{m}^3$) than with no filter (9.1 $\mu\text{g}/\text{m}^3$)
Lucking [43]	Healthy nonsmoking men ($N = 19$, mean age 25 years)	Randomized double blind 3-way crossover trial with 1 h of moderate exercise under each of the following conditions: 1 Filtered air	Total PM mass, PNC (particles <1 μm), NOX, total gaseous hydrocarbons	Endothelial vasomotor and fibrinolytic function, ex vivo thrombus formation, platelet activation, markers of inflammation, arterial stiffness and airway inflammation	Compared to unfiltered diesel exhaust, particle trap decreased PNC by >99.8 % and PM mass by 98 %; particle trap use associated with improved arterial vasodilatory function, reduced low-shear chamber

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		2	Unfiltered, diluted diesel engine exhaust		thrombus formation, and increase in tissue type plasminogen activator release; no statistically significant difference in health endpoints between filtered air and particle trap filtered diesel exhaust conditions suggesting that diesel particles (not gases/vapors) responsible for adverse cardiovascular health effects
		3	Dilute diesel engine exhaust passed through particle trap		
Muala [44]	Healthy adult nonsmokers (N = 30)	Crossover study with: (1) filtered air, (2) Unfiltered Diesel exhaust, (3) Diesel exhaust passed through filter and activated charcoal	PM ₁₀ mass, PNC, NO, NO ₂ hydrocarbons	Lung function: before and after exposure, general symptoms before and during exposure, inflammation markers 5 h after exposure	Compared to unfiltered exhaust, PM ₁₀ concentration decreased by 46 % with standard filter and 74 % with standard filter + activated charcoal; no changes in inflammatory markers; filtration reduced exhaust-induced symptoms
Clark [45]	Nonsmoking primary cooks (N = 79) in El Fortin, Nicaragua	Longitudinal study with all subjects receiving intervention of Eco-Stove (efficient wood-burning cookstove with chimney)	Indoor CO and PM _{2.5} mass, personal CO continuously for 48 h	SBP, DBP at end of 48-h metric period Baseline: May-July 08 Stoves Implemented: June-Sept 08 Follow-up metrics from May-June 09 (273–383 days later)	Intervention reduced average indoor PM _{2.5} concentrations by 77 %; greater SBP improvements among certain subgroups of the population (e.g., older and obese participants) were observed
McCracken [46, 47]	Women in rural Guatemala >38 years old who cook daily (N = 119)	Traditional open-fire cookstove vs. intervention with a chimney. Two different comparisons: 1 between groups of women with either open fire (N = 70) or chimney (N = 49) 2 before-and-after comparison of subset of women who used open fire and then switched to chimney intervention (N = 55)	Personal PM _{2.5} mass	Electrocardiogram, ST segment values, HRV, SBP, and DBP	Between-group comparison, OR of 0.26 (95 % CI=0.08, 0.90) for ST-segment depression; before-and-after comparison, OR=0.28 (95 % CI=0.12, 0.63); chimney intervention associated with lower systolic and diastolic BP in both comparisons