Air pollution and cardiovascular disease: Can the Australian bushfires and global COVID-19 pandemic of 2020 convince us to change our ways?

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Abstract
Air pollution is a major global challenge for a multitude of reasons. As a specific concern, there is now compelling evidence demonstrating a causal relationship between exposure to airborne pollutants and the onset of cardiovascular disease (CVD). As such, reducing air pollution as a means to decrease cardiovascular morbidity and mortality should be a global health priority. This review provides an overview of the cardiovascular effects of air pollution and uses two major events of 2020—the Australian bushfires and COVID-19 pandemic lockdown—to illustrate the relationship between air pollution and CVD. The bushfires highlight the substantial human and economic costs associated with elevations in air pollution. Conversely, the COVID-19-related lockdowns demonstrated that stringent measures are effective at reducing airborne pollutants, which in turn resulted in a potential reduction in cardiovascular events. Perhaps one positive to come out of 2020 will be the recognition that tough measures are effective at reducing air pollution and that these measures have the potential to stop thousands of deaths from CVD.

KEYWORDS
air pollution, bushfires, cardiovascular disease, COVID-19, PM10, PM2.5

INTRODUCTION

Air pollutants have measurable health impacts, with particularly profound effects on cardiovascular health. Ambient air pollution is the number one environmental risk factor for all-cause mortality, and ranks 5th overall, higher than tobacco smoking, HIV/AIDS, and all forms of violence including war.1–11 An estimated 8.8 million excess deaths a year are attributable to poor air quality with an associated healthcare cost of US$1–3 trillion.12–23 We cannot choose the air that we breathe and while air quality has improved in many developed countries since the 1970s, 90% of the world’s population currently live in areas where air pollution exceeds World Health Organization’s (WHO) guidelines, with a disproportionate percentage of those in developing countries. Worryingly, recent epidemiological evidence suggests that even air pollution levels below WHO guidelines are still associated with damaging health effects.4,5

Numerous epidemiological studies have correlated increased air pollution to a range of cardiovascular diseases (CVDs) including arrhythmias,6,7 atherosclerosis,8 and acute myocardial infarction (AMI).9 Despite the lungs being the major entry point of airborne pollutants into the body, 60% of deaths attributed to pollution are due to CVD, outweighing mortality due to respiratory disease.11 These findings demonstrate that airborne pollutants play a key role in mediating CVD progression. With 30% of global CVD deaths currently attributable to air pollution, combatting poor air quality to reduce CVD morbidity should now be a global priority.

Here, we discuss the ways in which air pollution elicits its detrimental effects on the cardiovascular system and how two major events in
2020 drastically altered air pollution levels and their knock-on effects on CVD mortality. Using the Australian bushfires of 2019–2020 and the COVID-19 pandemic lockdown as examples, we consider the importance of reducing air pollution as a mechanism to combat CVD.

**COMPOSITION AND SOURCES OF AIRBORNE POLLUTANTS**

Air pollution is a heterogeneous mix of gases, semi-volatile liquids, and particles, the exact composition of which is dependent on pollutant sources and environmental factors. In general, these pollutants are classified into primary and secondary pollutants, formed via physicochemical transformation of primary pollutants. Primary pollutants encompass particulate matter (PM), hydrocarbons, and inorganic gasses (e.g., ozone, nitrogen dioxide [NO$_2$], sulfur dioxide [SO$_2$]). Although negative correlations have been reported between gaseous pollutants, studies performed using isolation chambers found that ambient levels of these gases alone do not induce acute cardiovascular dysfunction. It is proposed that instead of acting directly to induce CVD, gaseous pollutants can act as co-pollutants, amplifying the detrimental effects of PM. Consequently, PM is currently considered to be the primary mediator of air pollutant-induced cardiovascular events and will be focused upon throughout this review.

Airborne PM is a mixture of solid and liquid particles of a variety of sizes suspended in the air. Particles are composed of various materials including, but not limited to, elemental or organic carbon, mineral dust, organic compounds (e.g., polycyclic aromatic hydrocarbons [PAHs]), biological material (e.g., cell fragments), metals (e.g., lead), and sea salt. Primary particles can react with gases in the atmosphere resulting in secondary PM formed of a core surrounded by a variety of chemical compounds. The availability of chemicals on the surface of particles changes their ability to induce pathological responses, and the cardiovascular effects of PM are highly dependent on their source. Urban PM has been linked to multiple cardiac effects while rural PM increased blood pressure. However, maritime wind PM was observed to have no adverse cardiac effects. Consequently, PM is currently considered to be the primary mediator of air pollutant-induced cardiovascular events and will be focused upon throughout this review.

Particles with a diameter of 0.1–2.5 µm are defined as fine particles or PM$_{2.5}$. These particles are typically carbon-based and are the major product of combustion from sources including vehicle engines, coal power plants, and bushfires. Primary PM$_{2.5}$ readily form secondary particles with a coat of constituents including ions (e.g., nitrates, sulfates), redox-active metals (e.g., copper, iron) and PAH (Figure 1B). These fine particles can travel ~1000 km away from their source with a half-life of up to a week, allowing them to exert their effects over vast areas. Their small size provides a relatively large reactive surface area and allows for penetration deep into the lungs to reach the alveoli, thereby increasing their toxicity. WHO guidelines recommend PM$_{2.5}$ exposure should average below 25 µg/m$^3$ over 24 h. Numerous epidemiological studies have correlated elevations in PM$_{2.5}$ with an increased risk of cardiovascular events, as previously reviewed.

Short-term elevations in daily PM$_{2.5}$ exposure by 10 µg/m$^3$ have been found to increase daily CVD mortality by up to 2%. Currently, only PM$_{2.5}$ and PM$_{10}$ levels are detected by most monitoring stations; however, there is a third class known as ultrafine PM with a diameter of less than 0.1 µm. Ultrafine particles are similar in composition to PM$_{2.5}$ and are primarily composed of organic carbon, hydrocarbons, and metals. These particles are of particular interest as they can deposit deep within the alveoli. They also have the potential to pass directly into the circulatory system resulting in systemic
dissemination and enhanced biological toxicity. However, as they tend to coalesce into larger particles, they have a relatively short half-life. Due to the abundance of ultrafine particles from vehicle exhaust and their potential to induce higher degree adverse effects, there are concerns that current methods to measure air quality rely only on PM10 and PM2.5 are oversimplistic, resulting in researchers overlooking the importance of ultrafine particles in CVD progression.

THE CARDIOMETABOLIC EFFECTS OF AIR POLLUTANTS

Extensive epidemiological studies have correlated air pollution to various CVDs, including atherosclerosis, venous thrombosis, stroke, arrhythmia, cardiac arrest, and heart failure. Both acute and long-term exposure to pollutants, particularly PM, are correlated with CVD mortality. Epidemiological studies, along with work performed in small animals and humans, illustrate the ability of airborne pollutants to impinge on almost all aspects of cardiovascular function. While the effects of air pollution are extensive and have been reviewed in detail elsewhere, below is a brief overview of the effects upon the vasculature and heart.

Vascular disease

Exposure to air pollution is associated with increased blood pressure predominantly due to altered vascular function. Human studies revealed that PM in diesel exhaust promotes vasoconstriction and decreases vasorelaxation in response to agonists. In apolipoprotein E knockout mice (a model of atherosclerosis), long-term exposure to PM2.5 resulted in altered vascular tone and increased the accumulation of oxidised lipids, which together potentiated atherosclerosis. It is proposed that PM2.5 impairs high-density lipoprotein function leading to increased plaque burden. Additionally, PM may facilitate the transition of plaques from a stable to unstable phenotype with an increased risk of plaque rupture. These findings emphasize the ability of airborne pollutants to induce vascular dysfunction and accelerate the progression of vascular disease.

Cerebrovascular disease

Several meta-analyses and other studies have demonstrated a robust association between both gaseous and PM pollution with stroke hospital admissions and mortality. A recent study into long-term exposure to differing levels of PM2.5 in cities across China found that with each 10 µg/m3 increase in PM2.5, there was a 13% increase in stroke incidence, with the most notable increase observed in ischemic stroke, which had a 20% increase in presentations per 10 µg/m3 increase in PM2.5. Additionally, short-term elevations in multiple gaseous pollutants correlate with a significant increase in ischemic stroke mortality. Elevations in ozone were linked to an increase in stroke mortality 3-days post-exposure whereas elevated CO and NO2 levels increase stroke mortality in the following 24 h. These studies highlight the acute and varying effects of different air pollutants on the cerebrovascular system. Air pollutant-induced stroke events are exacerbated in patients with a history of stroke, demonstrating the detrimental effects of airborne pollutants in at-risk populations.

Cardiac disease

Long-term exposure to air pollution is ranked in the top 10 risk factors for ischemic heart disease, placing it above the impact of a sedentary lifestyle (lack of exercise). PM2.5, NO2, and ozone have all been associated with AMI, with PM2.5 having a near-linear concentration-response relationship to the disease. Air pollution can rapidly induce cardiac events with acute exposure to traffic-derived pollutants increasing the risk of AMI in the following 2 h. Elevated risk of AMI in response to traffic-derived pollutants was shown to be independent of the mode of transport or level of physical exercise. The effects of pollutants are exacerbated in vulnerable individuals predisposed to cardiac problems, with reduced heart rate variability (HRV) being one example of this. While exposure to air pollution is associated with reduced HRV, these effects were potentiated and occurred upon exposure to a lower concentration of pollutants in elderly individuals. Preclinical studies found that pollutant-induced reduced HRV is associated with increased risk of arrhythmias and delayed cardiac conductance with long-term exposure resulting in cardiac hypertrophy and loss of cardiac function.

MECHANISMS OF POLLUTANT-INDUCED CVD

The precise mechanisms by which airborne pollutants elicit their effects on the cardiovascular system are still largely unknown. It is proposed that pollutants elicit direct effects on the cardiovascular system as well as indirect effects via epigenetic changes, inflammatory responses, and other mechanisms (Figure 2). The combined effects of air pollution stimulate pathological processes including endothelial damage, vascular dysfunction, autonomic and neuroendocrine dysfunction, thrombosis, and atherosclerosis.

One mechanism by which inhaled pollutants are proposed to alter cardiovascular function is via direct activation of alveolar receptors that alter neuroendocrine signaling and autonomic balance. Reduced HRV in response to PM exposure results from changes in the autonomic nervous system with increased sympathetic activity and reduced parasympathetic activity. PAHs present on the surface of PM appears to be a key driver in activating sensory neurons.

The soluble fraction of PM and ultrafine PM (<30 nm) may pass through into the bloodstream resulting in direct systemic effects on the vasculature. Iron-based particles have been identified in the hearts and brains of cadavers from Mexico City, which is known for high air pollution. The presence of these particles was associated with cellular damage in the surrounding tissues. Recent human studies
accumulate in atherosclerosis-prone arteries. Build-up of PM more, once particles enter the circulatory system, they preferentially persist within the circulatory system for up to 3 months. Further- utilizing inhaled 5 nm gold particles demonstrated that particles could oxidize leading to cardiac dysfunction. They also activate inflammatory cells in the lungs, including macrophages, leading to systemic inflammation and potentiation of oxidative stress leading to cardiac dysfunction.

Air pollutants induce additional indirect effects on the cardiovascular system via inflammatory agents, oxidative stress, and epigenetic changes. Macrophages and other inflammatory cells present within the lining of the alveoli can phagocytose inhaled pollutants, as they would other invading pathogens. The physico-chemical properties of PM derived from combustion sources promote activation of inflammatory cells leading to local and systemic inflammation. The activation of inflammatory cells has the potential to amplify the detrimental effects of PM via the release of inflammatory mediators that pass into the circulation and induce marked changes in other cells, including endothelial cells, thereby altering cardiovascular function. As well as inflammatory markers, oxidative stress markers are also consistently elevated in the blood after exposure to PM. Pollutants induce oxidative stress via mechanisms including PM-mediated free radical generation, oxidant production from pollutant-activated immune cells, and indirect activation of intracellular oxidant-producing enzymes such as nicotinamide ade- nine dinucleotide phosphate (NADPH) oxidase. Current research has identified a clear role for oxidative stress in governing CVD progression.

There is mounting evidence that airborne pollutants can indirectly influence cardiac homeostasis through epigenetic changes. A recent animal study demonstrated that exposure to PM2.5 altered chromatin accessibility causing dysregulation of multiple cardiometabolic pathways. Sustained exposure of mice to PM2.5 resulted in impaired glucose and insulin tolerance associated with differential expression of gene clusters involved with metabolism, circadian rhythm, and inflammation. It is postulated that exposure to PM alters DNA methylation in genes governing inflammation, cytokine production, and endothelial dysfunction, all of which contribute to pollutant-induced CVD. Interestingly, changes in response to PM2.5 were transient with the cessation of exposure resulting in a reversal of insulin resistance.

The ability of air pollutants to induce complex direct and indirect effects is exemplified by studies on diesel exhaust. Acute exposure to diesel exhaust impairs vasorelaxation in response to vasodilators in human studies, suggesting direct impairment of NO-mediated vasorelaxation. The effects on the vasculature were found to be rapidly induced (1–2 h), and these effects could persist for up to 24 h. Diesel exhaust has been shown to directly activate platelets that promoted blood clotting in an ex vivo thrombosis model. In addition to these direct effects, PM from diesel fumes induces indirect oxidative stresses and alters redox signaling in multiple cardiovascular systems. Diesel PM has been shown to generate superoxide and hydroxyl free radicals and to induce oxidative stress via alternative pathways including the uncoupling of NO synthase and mitochondrial dysfunction. Interestingly, diesel exhaust that was filtered to remove particulate matter significantly reduced the vascular impairments observed from whole exhaust fumes, highlighting that simple interventions are available to limit the effects of air pollutants on the surrounding population.

THE IMPACT OF MAJOR EVENTS IN 2020 ON AIR POLLUTION AND CVD

Naturally occurring events can lead to direct and indirect changes in air quality. Two such events in 2020 were the vast bushfires in Australia that burnt from September 2019 into January 2020, which directly increased pollution, and the COVID-19 pandemic that indirectly decreased global air pollution due to restrictions on travel and industrial processes. While most prior studies have evaluated the effects of air pollutants on CVD and mortality between different populations in the same timeframe, these events allow us to evaluate the short- and long-term effects of changes in air pollution within the same population during and after an event. This approach has the advantage of controlling for multiple variables including climate, population ethnicity, population demographics, and socio-economic factors. As such, these events allow us to directly assess the impact of perturbations in air pollution on CVD.
FIGURE 3  Natural events in 2020 drastically changed global levels of air pollutants. (A,B) The aerosol optical depth (AOD), detecting aerosols including smoke-derived particulate matter, across Australia was increased in January 2020 compared to January 2019 due to extensive bushfires. Image credit: NASA Earth Observations team based on data provided by the MODIS Atmosphere Science Team, NASA Goddard Space Flight Center. (C,D) The COVID-19 lockdown led to a significant decrease in NO2 emissions in major cities across the Indian subcontinent compared to the average emissions over the same period in the previous 3 years. Image credit: NASA’s Scientific Visualization Studio.

Australian bushfires

The scale of the 2019–2020 Australian bushfires was unprecedented in recent history. Over 17 million hectares of land were burned across the country, according to the Australasian Fire and Emergency Service Authorities Council. At the start of January 2020, real-time air quality measurements by IQAir ranked the Australian city of Canberra as having the worst air quality in the world, outranking both Delhi and Beijing. Visualizing the magnitude of the smoke released from the fires using NASA satellite imaging of the aerosol optical depth, which indicates PM levels in the atmosphere, showed a significant increase in aerosols in January 2020 compared to the same time the previous year (Figure 3A,B). The increase in observed aerosol optical depth resulting from the 2019–2020 Australian bushfire smoke broke the previous record from 2017 Canadian forest fires and the strongest volcanic eruptions of the last 29 years (Calbuco volcano in 2015 and Raikoke volcano in 2019).[69]

As a result of these fires, up to 11 million Australians were exposed to bushfire smoke with population-weighted PM2.5 concentrations exceeding the 95th percentile of mean daily values for 125
near-consecutive days.\textsuperscript{70} In Sydney, average PM\textsubscript{2.5} concentrations over 24 h exceeded 100 μg/m\textsuperscript{3}—this increase is estimated to have increased cardiovascular mortality by 4.5%.\textsuperscript{71,72} Bushfire smoke was deemed to be responsible for an estimated 1124 excess hospitalizations for CVD and 417 excess all-cause deaths.\textsuperscript{70} Studies on Australian bushfires in previous years have correlated smoke exposure to an elevated risk of cardiac arrest within 48 h of exposure.\textsuperscript{73} Fire-induced elevations in PM\textsubscript{10} were also associated with increased hospital admissions due to ischemic heart disease in Indigenous Australians 3 days post-exposure.\textsuperscript{74} Data from American bushfires found that extended periods of bushfire smoke exposure of up to 1 month increased cardiovascular mortality by 4.5%.

Bushfire smoke particles tend to be small, with PM in the range of 0.4–0.7 μm, allowing for deep penetration into the lungs and impacting the cardiovascular system via several mechanisms. Smoke extract from Australian native plants was found to increase the production of pro-inflammatory mediators in cultured macrophages and impair their phagocytic function.\textsuperscript{76} Exposure of lung fibroblasts to smoke extract resulted in the release of cytokines that promote systemic inflammation.\textsuperscript{77} This ability of smoke PM to induce systemic inflammation was observed in healthy volunteers who were exposed to wood smoke particles for just 2 h, which was shown to increase the number of inflammatory cells the following day.\textsuperscript{78} Additionally, wood smoke with PM\textsubscript{2.5} concentrations in the range observed during the 2020 Australian bushfires has been shown to have acute effects on the cardiovascular system by significantly increasing systolic blood pressure 24 h post-exposure.\textsuperscript{79}

Although the smoke-related health impacts from the 2020 Australian bushfires were substantial, the full extent of this will only be known after comprehensive epidemiological analyses have been performed. In terms of financial impact, it is estimated that the long-term smoke-related health burden of the 2020 bushfires will cost the Australian government approximately US$1.41 billion.\textsuperscript{80} Importantly, it is likely that the increased frequency of major bushfires is linked to climate change and global warming.\textsuperscript{81} Only by tackling climate change, using measures including curbing the use of fossil fuels, will we be able to address this critical global issue.

**COVID-19 pandemic**

In late 2019, a novel coronavirus (COVID-19) was detected in Wuhan, China, which turned into a global pandemic bringing worldwide industrial sectors and travel to a standstill. By the end of March 2020, half of the world’s population was under some form of lockdown, causing drastic social and economic knock-on effects. An indirect positive effect of the COVID-19 lockdown was a drastic reduction in air pollution globally. Stringent lockdown restrictions resulted in a 90% reduction in road and air travel bringing it to a 75-year low, as assessed by the Google mobility index. Consequently, global CO\textsubscript{2} emissions were reduced by 8.8%, a larger decrease than any other drop-in recorded history.\textsuperscript{82} The combined effects of the lockdown led to a reduction of 20%–30% in global NO\textsubscript{2} emission, a major product of fossil fuel combustion.\textsuperscript{83} Lockdown-induced improvement of air quality was strikingly evident in India (Figure 3C,D), which saw a reduction in PM\textsubscript{2.5} and NO\textsubscript{2} of 64.7% and 65.8%, respectively.\textsuperscript{84} Similarly, PM\textsubscript{2.5} was reduced in China and Europe by 30% and 17%, respectively.\textsuperscript{85}

Because at the time of writing (February 2021) we are still in the midst of the COVID-19 pandemic, it is challenging to draw strong conclusions on how reductions in air pollution have affected CVD mortality. However, modeling studies have estimated that the short-term effect of reduced PM\textsubscript{2.5} levels averted 24,200 deaths in China and an additional 2,190 deaths across Europe.\textsuperscript{86} If stringent lockdowns continue throughout 2021, this model predicts that over 300,000 deaths due to air pollution could be avoided across both regions. While this modeling only considered data from the first half of 2020, it shows the sizeable effects that reducing airborne pollutants can have on mortality.

Between January and April 2020, hospital admission from AMI decreased by 48% in Northern California, with similar decreases observed in both ST-segment elevation MI and non-ST-segment elevation MI.\textsuperscript{86} A similar decrease was reported in Milan, however, this was predominantly in non-ST-segment elevation MI.\textsuperscript{87} One theory is that patients with chest pain and symptoms of AMI are less likely to present at hospitals due to fear of contracting COVID-19. However, given the causal relationship between air pollution and AMI\textsuperscript{88} combined with the dramatic reduction in pollution over lockdown, it is conceivable that the observed decrease in AMI is directly linked to improvements in air quality.

As well as improving air quality, the “safer at home” policies employed by governments have increased time spent indoors, and when outdoors people have been encouraged to wear face masks to prevent viral transmission (Figure 4). The use of face masks has been shown to reduce inhalation of PM\textsubscript{2.5} and correlates with a reduced risk of cardiovascular events.\textsuperscript{89} These measures have unintentionally mitigated exposure to ambient air pollutants and in turn, may have contributed toward the drop in pollutant-induced CVD mortality resulting from lockdown.

However, the relationship between air pollution, COVID-19 and CVD is complex with airborne pollutants proposed to exacerbate COVID-19 and CVD comorbidity (Figure 4). Patients with underlying CVD have an increased risk of mortality from COVID-19, in part due to their frailty but also because of interaction between COVID-19 and the cardiovascular system.\textsuperscript{89,90} Despite the reduced AMI presentations during this pandemic, total CVD events have risen with data from the United Kingdom suggesting that CVD mortality has increased by 8%.\textsuperscript{91} Potentially, this may be partly attributable to COVID-19-induced cardiovascular events, as well as people not seeking appropriate treatment when they are unwell with CVD due to fear of hospital-acquired COVID-19 infection. Additionally, a positive correlation has been found between PM\textsubscript{2.5} levels and COVID-19 incidence (r = 0.67), mortality rate (r = 0.65) and the case fatality rate (r = 0.7).\textsuperscript{92} PM was determined to be a contributing factor in 15% of global mortality from COVID-19, which appears partially attributable to air pollutants exacerbating underlying cardiovascular conditions.\textsuperscript{93}
Newly emerging research has proposed that air pollution potentiates COVID-19-induced CVD by several mechanisms. Chronic exposure to PM2.5 increases the expression of angiotensin-converting enzyme 2 (ACE2), which is present as a transmembrane protein in the cell wall of differing cardiovascular cell types (including cardiomyocytes, endothelial cells, and pericytes) as well as in the lungs, kidney, and intestines. ACE2 normally lowers blood pressure by catalyzing the hydrolysis of angiotensin II (a vasoconstrictor peptide) into angiotensin 1–7 (a vasodilator). The SARS-CoV-2 virus, which causes COVID-19, enters cells using ACE2 as a "gateway" to cell infection. Massive viral binding to ACE2 results in a reduction in the availability of ACE2 to bind its usual substrate angiotensin II. This is speculated to lead to an increase of angiotensin-II and decreased production of the vasodilator angiotensin 1–7. Furthermore, it has been postulated that pollution-induced overexpression of ACE2 facilitates higher levels of virus binding, which increases viral load within cells and simultaneously depletes ACE2, leading to an exaggerated disease response. Recent reports also suggest that SARS-CoV-2 can absorb onto the surface of PM creating PM-SARS-CoV-2 particles. The creation of particle-virus hybrids may conceivably facilitate entry of the virus deep into the alveoli or facilitate translocation directly into the circulatory system, thus increasing viral load. As both PM and SARS-CoV-2 are known to induce systemic inflammatory responses, their combined effects may elicit the so-called "cytokine storm" characteristic of COVID-19. In support of this theory, recent reports have found that high viral load can induce fulminant myocarditis with infiltration of inflammatory cells. PM exposure is therefore proposed to mediate the adverse cardiovascular outcomes of COVID-19 by directly exerting deleterious effects and indirectly by facilitating virus entry and increasing viral load.

Taking all the above data as a whole, a complex balance of competing factors appears to be at play, juxtaposing beneficial cardiovascular effects of reduced pollution and airborne PM through the current pandemic, versus adverse cardiovascular effects of COVID-19, and additional in vivo and ex vivo interactions between COVID-19 and airborne pollutants. Epidemiological studies to determine the precise relationship between air pollution, COVID-19, and CVD are currently ongoing, but it may be some time before we fully understand the effects of these factors. However, PM-mediated COVID-19 and CVD comorbidity could mask any positive effects. The true benefits of the lockdown-induced reduction in air pollution on CVD will not become clear until detailed epidemiological studies have been undertaken. Worryingly, it is possible that the reduction in air pollution observed during the pandemic lockdown was not severe or long-lived enough to have any substantial effects on CVD mortality. Indeed even with the stringent lockdown and 30% reduction in PM2.5, the concentration of PM2.5 in 95 locked-down cities across China was still four times higher than deemed safe by WHO.

CAN WE LEARN FROM 2020 AND CHANGE OUR WAYS?

The Australian bushfires of 2020 highlighted that increases in air pollution come with a substantial human and financial cost. Taken together with the wealth of prior data (as outlined above), it is clear that from a cardiovascular and many other perspectives, reducing ambient air pollution should be a priority. The 2020 European Environmental Plan put the reduction of emissions at the top of its agenda; however, the COVID-19 pandemic has put a substantial and prolonged strain on global industry, threatening to push the world into an economic recession. The threat of recession may lead societies to do "whatever it takes" to stimulate economic growth, thereby pushing air pollution out of the limelight. However, delays in tackling air pollution will result in major long-term health consequences, from which governments will face considerable financial repercussions.

While the stringent measures taken during the COVID-19 pandemic are not feasible or practical to continue permanently, the lockdown demonstrated that changes in human behavior can rapidly reduce air pollution. There are simpler long-term alternatives that can be employed to reduce industrial and traffic pollution than total lockdown. Measures put in place during the 2008 Beijing Olympics regulating power plant operation times and reducing traffic resulted in a 30% drop in PM10 concentrations across targeted cities, which
was associated with a decrease in cardiovascular mortality, particularly in women and the elderly.[101] As another measure, the enforced use of higher quality gasoline in motor vehicles in China resulted in a 13% reduction in pollution across all pollutants.[102] However, moving away from our reliance on fossil fuels should be the goal. Globally fossil fuel combustion accounts for 50%–60% of human-made airborne pollutants, with up to 80% of this generated by West Asia, North America, and Europe.[93] It is estimated that by ceasing the burning of fossil fuels we could increase the global mean life expectancy by 1.1 years.[22]

In the past, government legislation has created changes that have directly reduced pollution and in turn, reduced CVD mortality. In Scotland, the 2006 ban of smoking in public places resulted in a 17% reduction in rates of hospital admission from acute coronary syndrome.[103] Interestingly non-smokers accounted for 67% of the reduction, demonstrating that second-hand smoke is a major player in the onset of acute coronary syndrome. While cigarette smoke is not directly comparable to environmental pollution, the positive effects of the smoking ban on CVD may draw parallels to the effects of reducing air pollutants.

In addition to utilizing legislation to reduce air pollution, personal measures can be taken to reduce exposure. The COVID-19 pandemic has normalized everyday wearing of face masks to prevent virus transmission. As discussed, a secondary benefit from this is a marked reduction in the inhalation of PM104 and it has been shown that wearing a face mask attenuates the reduction in HRV in response to PM, with N-95 masks providing the greatest level of protection.[105] These studies demonstrate the need for health care providers to integrate air pollution management into treatment plans for CVD, which could include steps such as monitoring PM2.5 levels and wearing face masks when appropriate to reduce the risk of acute cardiovascular events.

CONCLUDING REMARKS

Reducing air pollution is perhaps one of the single most important actions we can take to actively prevent adverse health events and reduce global mortality from CVD. If we act now, we may be able to reverse some of the negative effects air pollution has had on the current generation, as recent research has demonstrated that the detrimental epigenetic changes associated with PM2.5 exposure are reversible.[59] While we will only know the full impact of the events of 2020 on CVD with hindsight, perhaps one positive outcome from this annus horribilis will be the realization that we can control air pollution levels and that stringent measures could save hundreds of thousands of lives.

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CONFLICT OF INTEREST

There are no conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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