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Connections Between Air Pollution, Climate Change, and Cardiovascular Health

Barrak Alahmad, MD, PhD[a,b,c], **Haitham Khraishah, MD**[d], **Khalid Althalji, MD**[e], **William Borchert, MPhil, MHS**[a], **Fahd Al-Mulla, MD, PhD**[c], **Petros Koutrakis, PhD**[a]

[a]Department of Environmental Health, Harvard T.H. Chan School of Public Health, Boston, Massachusetts, USA

[b]Environmental and Occupational Health Department, College of Public Health, Kuwait University, Kuwait City, Kuwait

[c]Dasman Diabetes Institute (DDI), Kuwait City, Kuwait

[d]Division of Cardiovascular Medicine, University of Maryland School of Medicine, Baltimore, Maryland, USA

[e]Jaber Alahmad Hospital, Ministry of Health, Kuwait City, Kuwait

Abstract

Globally, more people die from cardiovascular disease than any other cause. Climate change, through amplified environmental exposures, will promote and contribute to many noncommunicable diseases, including cardiovascular disease. Air pollution, too, is responsible for millions of deaths from cardiovascular disease each year. Although they may appear to be independent, interchangeable relationships and bidirectional cause-and-effect arrows between climate change and air pollution can eventually lead to poor cardiovascular health. In this topical review, we show that climate change and air pollution worsen each other, leading to several ecosystem-mediated effects. We highlight how increases in hot climates as a result of climate change have increased the risk of major air pollution events such as severe wildfires and dust storms. In addition, we show how altered atmospheric chemistry and changing patterns of weather conditions can promote the formation and accumulation of air pollutants: a phenomenon known as the climate penalty. We demonstrate these amplified environmental exposures and their associations to adverse cardiovascular health outcomes. The community of health professionals—and cardiologists, in particular—cannot afford to overlook the risks that climate change and air pollution bring to the public's health.

Corresponding author: Dr Barrak Alahmad, Department of Environmental Health, Harvard T.H. Chan School of Public Health, 401 Park Drive, Landmark Center Room 412J, Boston, Massachusetts 02115, USA. balahmad@hsph.harvard.edu.

Ethics Statement

The research reported has adhered to the relevant ethical guidelines.

Patient Consent

The authors confirm that patient consent is not applicable to this review article.

Disclosures

The authors have no conflicts of interest to disclose.

RÉSUMÉ

Les maladies cardiovasculaires représentent la première cause de mortalité dans le monde. Or, les changements climatiques, qui amplifient l'exposition aux facteurs environnementaux, favoriseront la survenue de nombreuses maladies non transmissibles, y compris les maladies cardiovasculaires. Chaque année, la pollution atmosphérique est en outre à l'origine de millions de décès lies aux maladies cardiovasculaires. Bien qu'ils puissent sembler indépendants, les changements climatiques et la pollution atmosphérique sont étroitement imbriqués et présentment des liens de cause à effet bidirectionnels qui peuvent finir par altérer la santé cardiovasculaire. Dans cet article, nous montrons que les changements climatiques et la pollution atmosphérique s'aggravent l'un l'autre et entraînent divers effets par l'entremise des écosystèmes. Nous montrons de quelle façon les climats de plus en plus chauds découlant des changements climatiques ont augmenté le risque d'épisodes de pollution atmosphérique majeure comme les feux de forêt violents et les tempêtes de poussière. De plus, nous décrivons comment les modifications sur le plan de la chimie atmosphérique et l'évolution des conditions météorologiques peuvent peuvent favoriser la formation et l'accumulation de polluants atmosphériques: un phénomène connu sous le nom de « pénalité climatique ». Nous faisons état de l'amplification de ces expositions environnementales et de leurs conséquences indésirables sur la santé cardiovasculaire. Devant ce constat, les professionnels de la santé – et les cardiologues en particulier – ne peuvent se permettre de négliger les risques que les changements climatiques et la pollution atmosphérique font peser sur la santé publique.

Climate change has led to unprecedented challenges to the balance of ecosystems and sustainable living. Through continuous environmental degradation and hyper-consumption of food, water, and land, anthropogenic emissions of greenhouse gases continue to trap heat in the atmosphere and induce significant and dangerous changes to Earth's climate. A failed ecosystem, as described by Schaeffer et al.,[1] happens when the homeostatic repair mechanisms of that system can no longer support the negative degrading effects that become irreversible and lead to a collapse of its structure and function. Unmitigated climate change could push the limits of our ecosystem and will undoubtedly result in detrimental outcomes on individual, community, and global levels. The latest report from the Intergovernmental Panel on Climate Change (IPCC) established that climate change is already harming the health of populations.[2] The pathways in which climate and health interact, however, are rather complex and interdependent.[3–7]

Climate change is promoting the development of non-communicable diseases such as cardiovascular disease (CVD), which is the leading cause of death globally. There are direct and indirect ways in which climate change can worsen cardiovascular health or lead to premature deaths from cardiovascular disease.[6] Direct pathways include, for example, exposure to extreme heat and poor air quality. Indirect pathways include exposure to the by-products of climate change, such as the production of secondary air pollutants and damaged infrastructures from extreme weather events.

Air pollution is another important yet often overlooked environmental risk factor for cardiovascular health outcomes.[8] The evidence shows that exposure to short-term

concentrations of fine particulate matter is associated with increased risks (across hours to days) of myocardial infarction, stroke, and death from other CVDs.[9]

Interchangeable relationships and bidirectional cause-and-effect arrows between climate change and air pollution can eventually lead to poor cardiovascular health (Fig. 1). In one direction, excessive fossil fuel burning and industrial air pollution emissions result in more heat-trapping gases that worsen climate change. In another direction, climate change leads to a number of ecosystem-mediated effects that worsen air pollution. In this review, we show how climate change and air pollution worsen each other, then we show 3 examples in which climate-mediated air pollution can be linked to cardiovascular disease: wildfires, dust storms, and weather-related penalty.

Air Pollution Makes Climate Change Worse

When fossil fuels are burned for energy use and production, large quantities of gases and particles are released into the air, especially greenhouse gases. When solar radiation reaches the Earth's surface, it heats up the planet, and, as a result, the Earth radiates this heat back into space in the form of long-wave radiation. However, the greenhouse gases in the atmosphere have the property of absorbing and re-emitting some of this heat back toward the surface. This trapping of heat by greenhouse gases inevitably increases global atmospheric temperatures. Although greenhouse gases are a natural part of the Earth's atmosphere, excessive amounts have been emitted since the industrial revolution from smokestacks at factories, power plants, vehicle exhausts, agricultural operations, and other sources. Since 1880, Earth's temperature increased by 0.08 °C (0.14 °F) each decade, and since 1981, the rate of warming has doubled to be 0.18 °C (0.32 °F) per decade and continues to rise.[10] In fact, even if all anthropogenic emissions are to stop abruptly today, the global temperatures can continue to rise by approximately 0.5° C (0.9 °F) in the following decade.[11] The balance of nature and the Earth's ecosystem continues to be disrupted as a result of anthropogenic fossil fuel emissions, which have increased the temperature of the atmosphere and oceans and subsequently have led to increased rates of extreme climate conditions. The IPCC and the international community warned that maintaining the status quo of fossil fuel emissions will inevitably increase Earth's temperature by 1.5 °C above preindustrial levels, which is risking further sea level rise, severe loss in biodiversity and species extinction, water and food scarcity, poverty and migration for millions of people worldwide, and serious deterioration of public health.[2]

Carbon dioxide is a long-lived gas that is very well mixed in the atmosphere. It is therefore assumed that its impact on heating the Earth is rather uniform and independent of emission location. However, new evidence now suggests that the coemissions from human activity produce compounds that are shorter-lived than carbon dioxide—such as black carbon and organic carbon aerosols, carbon monoxide, volatile organic compounds, sulfur dioxide and others are not well mixed—and will exert geographically heterogeneous effects on atmospheric composition and climatic systems.[12] Not only are these coemitted aerosols associated with poor health outcomes when inhaled by humans, they are also disturbing the balance of climate by influencing precipitation patterns and temperatures.[13]

Climate Change Makes Air Pollution Worse

Climate is one of the determinants of air quality.[14] Meteorologic factors—such as ambient temperature, humidity, precipitation, wind speed and direction, and vertical mixing—can influence air pollutants after emission.[15] Pollutants after discharge from a mobile or stationary source go through processes of transport, dispersion, chemical transformation, and deposition. Climate change can disrupt many of these atmospheric conditions and hence impair the dilution and removal processes of pollutants from the ecosystem. Excessive warming of the planet can lead to changes in precipitation patterns and other climate anomalies, resulting in drier conditions that can increase the frequency and intensity of wildfires and dust storms. These events are known to produce toxic air pollutants. Higher temperatures, decreased rainfall, and air stagnation are favourable conditions to promote the formation and accumulation of secondary pollutants. Airborne allergen pollutants are also exacerbated by climate change. The pollen and flowering season is now, on average, happening 20 days earlier and 8 days longer than it used to be.[16] Overall, densely populated areas may particularly be disproportionately affected by climate-mediated worsening of air quality.

Many of these climatic and air pollution conditions can affect cardiovascular health through multiple cascading and compounded environmental exposures.[6] There is over-whelming evidence in support of air pollution's links with cardiovascular and cardiometabolic morbidity and mortality.[8,17] Although air pollution and climate change effects on cardiovascular health may appear to be independent, they are closely interlinked ecologically. We highlight 3 extensively studied examples in environmental health literature of climate-mediated air pollution events and review the available evidence on their effects on cardiovascular health (Table 1).

Example 1: wildfires and CVD

In recent years, the frequency and intensity of wildfires have been on the rise.[18] A changing climate of heat mixed with drought and lightning strikes has made forests into tinderboxes for a wildfire. Smoke from wildfires is a heterogeneous mixture of coarse, fine, and ultrafine particles; volatile organic compounds; metals; hazardous air pollutants; gaseous pollutants; and water vapor.[19–21] In addition to the notable respiratory effects from wildfires, their smoke has been associated with numerous cardiovascular outcomes and risk factors.[19] Moreover, wildfires contribute to pollution and atmospheric warming, which are 2 leading drivers of climate change. The increases in the prevalence of wildfires have dramatically increased carbon emissions, erasing the effects of 16 years of carbon emission-reduction strategies.[22,23]

Smoke from wildfires contributes to all-cause mortality[24] as well as CVD mortality.[25] Notably, a meta-analysis of data from 10 European cities reported that smoke from forest fires was associated with increased CVD mortality, and this mortality was largely driven by large particulate matter with aerodynamic diameter < 10 microns (PM_{10}).[26] This effect, however, was not seen in a study of fine particulate matter with aerodynamic diameter < 2.5 microns ($PM_{2.5}$) in the United States.[27] Of the 10 studies that were identified that

examined an association between wildfires and CVD mortality in Western and Eastern Europe, Southeast Asia and South America, 8 of them reported an association with increased risk of death.[26,28–33] These results are consistent with studies evaluating the effect of occupational exposure to particulate matter such as in persons fighting wildfires[34] and exposure to ambient particulate matter in the absence of wildfires.[35] Two studies reported null associations with monitored $PM_{2.5}$[36] and PM_{10}[37] and cardiorespiratory and CVD mortality, respectively.

Literature assessing the effect of wildfire exposure on the incidence of myocardial infarction was mixed, with some reporting no association to wildfire smoke.[38–41] Several studies using different metrics to measure wildfire smoke such as directly measured $PM_{2.5}$, modelled $PM_{2.5}$ concentrations, and smoke density, did report an association between wildfire smoke and incident myocardial infarction[38,40,42] or death from myocardial infarction.[31] Weichenthal et al. found that this effect was more pronounced in the elderly in whom a 10-$\mu g/m^3$ increase in the 3-day mean $PM_{2.5}$ was significantly associated with acute myocardial infarction in those 65 years of age and older. A similar association was not detected in younger participants.[38]

Although there was a heterogeneous association between wildfire smoke and arrhythmia in the literature,[40,42–47] there was a consistent association between wildfire smoke and sudden cardiac arrest.[41,48–50] Studies on admissions because of congestive heart failure also were mixed.[43,44,51,52] A study by Rappold et al.,[52] however, reported an association between modelled $PM_{2.5}$ from wildfire smoke and emergency department visits for heart failure. This association does not appear to be uniform across all communities and age groups.[53] Notably, when stratifying by age, seniors (those above 65 years of age) seem to be at higher risk of hospital visits for congestive heart failure caused by wildfire smoke exposure.[54,55] In addition, stroke[42,49,56] and ischemic heart disease[41,44,57] have also been suggested to be associated with wildfire smoke, although the results have not been consistent.

The current literature on risk factors for CVD, such as hypertension from exposure to wildfire smoke is limited, but some studies have reported an association. In adults, a 10-mg/m^3 increase in $PM_{2.5}$ from peat fire was associated with an increase of emergency department visits for hypertension.[58] An additional study reported a similar association between smoke-plume density and emergency department visits for hypertension.[42] In contrast, this association was not evident in a New Mexico study of acute exposure to $PM_{2.5}$ from wildfires and hypertension emergency visits.[56] These studies do not tell us whether these additional visits were incident hypertension events or an exacerbation in already known hypertensive patients.

Studies used different exposure definitions of wildfire smoke and applied different study designs and statistical models. Nevertheless, despite some variability in the results of the studies on wildfires and cardiovascular disease, the majority of studies showed a positive association between increased wildfire smoke and increased cardiovascular morbidity and mortality, particularly among those with advanced age. Although the association between the underlying cardiovascular conditions and wildfire smoke is modest, there was a clear relationship between cardiovascular mortality and exposure to wildfire smoke. As climate

change continues to initiate and perpetuate wildfires, the resulting unhealthy polluted environments will continue to contribute to additional cases and deaths from CVD.

Example 2: dust storms and CVD

Climate anomalies, including global warming, shifts in the El Niño phenomenon, and deforestation, can all contribute to droughts.[59] Soil moisture is reduced significantly with persistent water loss and hot weather, which, in turn, will lead to inevitable desertification and soil erosion.[60] With strong blowing winds, more loose soil is now elevated and transported from degraded land, leading to an increase in frequency and magnitude of dust storms. For example, in the arid regions of Kuwait and Iraq, significant loss in vegetation and variations in long-term climatic factors were associated with increased dust levels.[61] In another example, in the desert of Mongolia, significant increases in summer temperatures and anomalous cyclonic circulation resulted in more dust particles being lifted high into the air to travel hundreds of miles downwind toward China and South Korea.[59]

Overall, more than 150 countries worldwide are affected by sand and dust storms[62] in which many vulnerable people could be at risk of adverse health effects. Dust storms consist of large (PM_{10}), coarse ($PM_{2.5–10}$), and fine ($PM_{2.5}$) particulate matter. The evidence shows that an exposure to increased dust particles is associated with acute mortality during dust-storm days. One systematic review and meta-analysis showed a 0.27% increase in all-cause mortality when comparing dust days with nondust days.[63] With regard to cardiovascular mortality and morbidity, various studies have demonstrated an adverse association with desert-dust events. A meta-analysis of studies of dust exposure in Asia showed a 2.33% relative increase in combined respiratory and circulatory deaths when comparing dust days with nondust days.[64] In Cyprus, cardiovascular hospitalizations were reported to have increased by 10.4% during dust-storm days.[65] In Taipei, Taiwan, researchers found a relative increase of 26% in overall emergency admissions for CVDs during dust-event days.[66] Specifically, there was a 35% and 20% increase in the relative risk of ischemic heart disease and stroke emergency visits, respectively, during dust periods compared with predust periods. A number of other studies have shown an association between exposure to dust and the risk of stroke (both ischemic and hemorrhagic).[67–70] Hospitalizations for risk of heart failure increased by 3.7% only 1 day after a dust storm in Taiwan.[71]

Many inflammatory cascading events may be involved in the cardiovascular effects following dust inhalation. Inflammation from dust exposure may lead to increased heart rate and blood pressure along with a decrease in myocyte contractility.[72] The differences in effect size, however, among dust-health studies may be attributed to a number of factors. First, the composition and source of dust storms may be different from one place to another. Second, there are different underlying vulnerabilities of populations (eg, aging, prevalence of cardiometabolic disease). But, mostly, a large aspect of the variability could arise from the differences in exposure assessment strategies of dust storms and epidemiologic designs used to evaluate health outcomes.[73]

Example 3: climate penalty and CVD

The concept of “climate penalty” provides another example of the potential for climate change to augment the adverse health effects caused by air pollution. Climate penalty refers to the enhanced formation of secondary pollutants such as ground-level ozone (tropospheric) with rising temperatures and altered meteorologic conditions caused by climate change. Decreased precipitation and humidity, changes in wind characteristics, and increased frequency of hot temperatures are meteorologic conditions that favour the promotion and accumulation of ozone. Recent studies have also predicted a similar effect on the concentration of fine particulate matter ($PM_{2.5}$).[74] Rising temperatures increase the oxidizing potential of the atmospheric components to produce more sulfate particles, which are among the major components of fine particulate matter.

There has been an increasing body of evidence linking short-term exposure to ground-level ozone with higher all-cause mortality. A large global epidemiologic investigation that analyzed approximately 50 million deaths in 20 countries found an excess mortality of 0.18% associated with each 10-mg/m^3 increase in ozone.[75] A similar association was observed between exposure to $PM_{2.5}$ and all-cause mortality non-accidental in another global study in which the magnitude this effect was larger with an estimated increase of 0.68% per 10-mg/m^3 increase in $PM_{2.5}$.[76]

CVDs consistently have been shown to be a leading cause of death caused by exposure to both tropospheric ozone and ambient particulate matter. In an analysis that collected data from 272 Chinese cities, the authors found a 0.27% increase in death caused by CVDs per 10-mg/m^3 increment in ozone.[77] In addition, the authors noted a higher ozone concentration during warmer seasons, which translated to higher CVD mortality in some regions compared with cooler periods. Until recently, most of the evidence showing a positive association between increased ozone exposure and increased CVD mortality focused on short-term exposure. However, in 2 studies performed in the United States, Turner et al.[78] and Lim et al.[79] managed to demonstrate a significant association between long-term ozone exposure and mortality caused by ischemic heart disease, arrhythmias, and heart failure. A similar association was observed in a nationwide cohort study that was done in China, a populous middle-income country, showing a linear increase in cardiovascular mortality with long-term exposure to higher ozone concentration in warm seasons.[80]

With regard to fine particulate matter, numerous studies indicated higher risk of cardiovascular diseases associated with high $PM_{2.5}$ exposure. A systematic review that evaluated 33 studies from 4 continents concluded that short-term exposure to higher concentrations of $PM_{2.5}$ results in an excess CVD morbidity risk of 2.65%. Analysis of cause-specific CVD was particularly suggestive of increased risk of myocardial infarction morbidity and out-of-hospital cardiac arrest.[81] Several studies have also found an increased risk of hospital admission because of heart failure with both acute and chronic exposure to fine particulate matter.[82–85] Long-term $PM_{2.5}$ exposure has been shown in multiple studies from the United States, Europe, and China to be associated with increased risk of CVD morbidity and mortality.[86–89]

There has been an increasing interest recently in the potential adverse health effects of the interaction between air pollution and air temperature. A time-series study in 2017 examined the 2-way effect modifications of higher air temperature and pollution in 8 European cities.[90] Consistent with previous studies from several other countries, the authors have found a greater effect of PM on both total nonaccidental mortality and cardiovascular mortality on days with higher air temperature. Similarly, ozone-related mortality was found to be higher on warmer days. The authors noted an increase in heat-related cardiovascular mortality with higher concentrations of PM and ozone. This effect, although not fully understood, is thought to be related to a synergistic effect of air pollutants and higher air temperature on the cardiovascular system, resulting in increased blood viscosity, systemic oxidative stress, inflammatory response, endothelial dysfunction, and impaired fibrinolysis. In a recent systematic review, Anenberg et al.[91] demonstrated a strong interactive effect between exposure to heat and air pollutants, with the strongest evidence for exposure to ozone and $PM_{2.5}$. The authors found a moderate quality of evidence of a dose-response relationship between heat and air pollution, with sufficient evidence pointing to a combined effect on all-cause and cardiovascular mortality. Another very recent study in California estimated the effect of coexposure to extreme heat and fine particulate matter on mortality and found a larger increase in cardiovascular mortality on days with both extreme heat and $PM_{2.5}$ concentrations.[92] This effect was higher than the sum of the individual effects of each of these risk factors. This growing evidence suggests that the synergistic adverse health impact of climate change through pollutants might be larger than our previous predictions.

Mechanistic Insights

Air pollutants can cause cardiovascular toxic effects through complex and varied mechanisms. Mechanisms can be classified into 3 categories: initiating mechanisms, effector pathways, and risk-factor development.[8] Initiating mechanisms involve processes such as inflammation, activation of neural reflex arcs, and ligation of pattern-recognition receptors.[8] Depletion of endogenous antioxidants and oxidative stress play a critical role air-pollution toxicology. Effector mechanisms involve the activation of quick neural pathways and the release of biologically active substances such as inflammatory cytokines, oxidized lipids, immune cells, microparticles, and microRNA.[9,17] Endocrine disruption can occur with both air pollution and many synthetic chemicals.[8] Finally, risk-factor development, such as hypertension and type 2 diabetes, results from chronic pollution-induced oxidative stress and inflammation.[9] Increases in blood pressure after exposure to air pollution may involve alterations in vascular and autonomic tone, whereas prolonged exposure to inhalational $PM_{2.5}$ can result in transcriptional and epigenetic reprogramming.[93]

Environmental Justice Considerations

Air pollution and wildfires can disproportionately affect vulnerable communities such as low-income neighborhoods, indigenous communities, and communities of color.[94] These communities may already face higher levels of pollution owing to factors such as proximity to industrial facilities or highways. Exposure to air pollution and wildfires can further increase the risk of adverse cardiovascular health and premature death. Dust storms can be particularly harmful to migrant workers[95] who are often exposed to high levels of

dust and particulate matter in agricultural fields or construction sites. This exposure can exacerbate existing cardiovascular conditions or increase the risk of developing new ones. Systematically disadvantaged and disenfranchised communities may have limited access to health care, making them even more vulnerable to the health effects of environmental exposures. Therefore, efforts to reduce air pollution and mitigate the risk of wildfires and dust storms must consider rooted community engagement and prioritize equity efforts to ensure that all communities have access to clean air and a healthy environment.

Conclusions

There is a complex 2-way interplay between air pollution and climate change. This process starts with anthropogenic greenhouse gas emissions that—through numerous mechanisms—damage the ecosystem, resulting in environmental stressors that adversely affect cardiovascular health. Although, globally, the rate of cardiovascular mortality has been declining for decades, it remains the leading cause of death globally. The expected increase in the frequency of adverse environmental events (eg, wildfires, dust storms, climate penalties, and extreme weather events) carries a significant threat to cardiovascular health and public health in general. Although the health effects are assessed and public health policies are created to deal with the global issues of climate change and air pollution, we are not only sounding the alarm for health professionals—and cardiologists, specifically—to be aware of environmental consequences but are also viewing this as an opportunity for the community of health professionals to present a set of policies and strategies that integrate individual, community, and governmental interventions to promote health in a changing climate.

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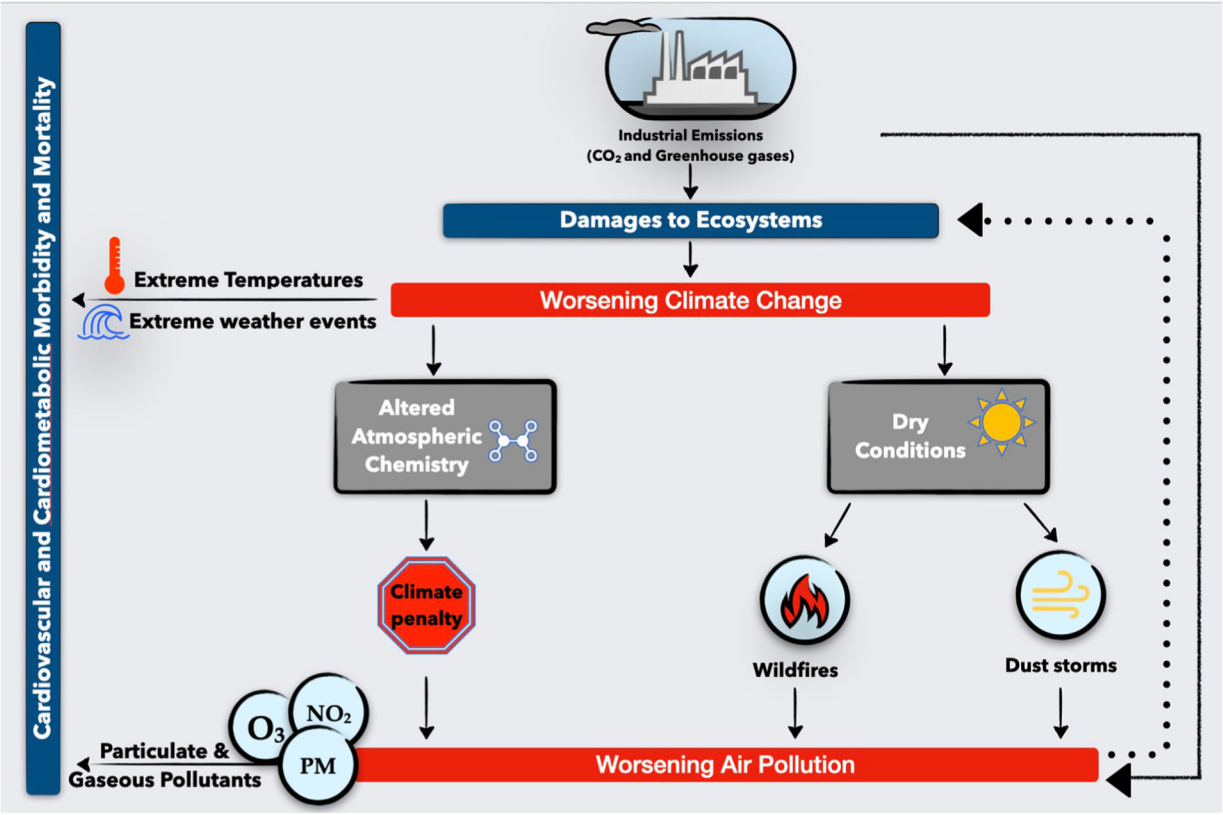

Figure 1.

Interchangeability between worsening climate change and worsening air pollution, leading to cardiovascular and cardiometabolic diseases. NO_2, nitrogen dioxide; O_3, ozone; PM, particulate matter.

Table 1.

Summary of climate-mediated events that increases the concentrations of gaseous and particulate air pollutants and their association with potential adverse cardiovascular outcomes

Climate-mediated events	Air pollution exposure pathways	Potential cardiovascular outcomes
• Wildfires • Dust storms • Climate penalty	• Fine particulate matter (PM_{25}) • Large particulate matter (PM_{10}) • Ozone (O_3) • Nitrogen dioxide (NO_2)	• Overall CVD mortality • Overall CVD hospitalizations • Ischemic heart disease • Stroke (hemorrhagic and ischemic) • Arrhythmia • Sudden cardiac arrest • Congestive heart failure • Hypertension

CVD, cardiovascular disease.