Climate change and the prevention of cardiovascular disease

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ABSTRACT

Climate change is a worsening global crisis that will continue negatively impacting population health and well-being unless adaptation and mitigation interventions are rapidly implemented. Climate change-related cardiovascular disease is mediated by air pollution, increased ambient temperatures, vector-borne disease and mental health disorders. Climate change-related cardiovascular disease can be modulated by climate change adaptation; however, this process could result in significant health inequity because persons and populations of lower socioeconomic status have fewer adaptation options. Clear scientific evidence for climate change and its impact on human health have not yet resulted in the national and international impetus and policies necessary to slow climate change. As respected members of society who regularly communicate scientific evidence to patients, clinicians are well-positioned to advocate on the importance of addressing climate change. This narrative review summarizes the links between climate change and cardiovascular health, proposes actionable items clinicians and other healthcare providers can execute both in their personal life and as an advocate of climate policies, and encourages communication of the health impacts of climate change when counseling patients. Our aim is to inspire the reader to invest more time in communicating the most crucial public health issue of the 21st century to their patients.

1. Introduction

Human activities, particularly the combustion of fossil fuels, have unequivocally warmed the earth’s atmosphere, ocean, and land by increasing the atmospheric concentrations of greenhouse gases (GHG) like carbon dioxide (CO₂) [1–5]. The big picture solution to climate change is that fewer climate forcing agents must be put into the atmosphere than are taken out, a process referred to as “mitigation” [6]. These mitigation strategies involve every energy-consuming human activity. Sustained reductions in total global CO₂ emissions have yet to occur. Several Western industrialized economies have managed to achieve small reductions in GHG emissions over the past two decades [7, 8]. Still, these reductions in GHG are inadequate to achieve emission goals to keep global warming below 1.5 °C this century. Furthermore, increased GHG production is expected from low- and middle-income countries, especially without appropriate assistance from high income countries [9].

1.1. The impact of climate change and planetary health on human health

Climate change affects human health through extreme weather events, heat stress, air pollution, infectious diseases, malnutrition, and other factors such as migration and displacement, outlined by the World Health organisation and The Lancet Countdown International Collaboration on Health and Climate Change [10–13]. The health effects of air pollution and increasing temperatures are particularly salient for those with or at increased risk of CVD [13–17]. In 2019, air pollution was responsible for 11.8% of deaths and 8.3% of disability-adjusted life years (DALYs), and high temperatures were responsible for 0.54% of death and 0.46% of DALYs globally [18,19]. Assuming no adaptation, the
global climate change-attributable, heat-related excess mortality is expected to almost triple between 2030 and 2050 [20].

“Adaptation” is defined by the Intergovernmental Panel on Climate Change (IPCC) as the process of adjustment to actual or expected climate [21] (Table 2). This can be considered analogous to the secondary prevention of cardiovascular disease (CVD). It does not encompass the activities that prevent the onset of climate change itself but, rather, actions that minimize the impact of climate change when it has already occurred. Adaptation capacity is strongly linked to socioeconomic status. Climate change disproportionately harms those with the fewest resources. Health care and other inequities will soar unless there are definite steps to avert this [22]. Already, racial minorities experience greater exposure to air pollution due to proximity to large roads and urban redlined areas (with the latter tending to be hotter than neighboring areas due to a lack of tree cover) [23-26].

Planetary health is a related but different concept to climate change. It refers to the health impacts of disruption to the earth’s natural systems caused by humans [27]. It expands the definition of environmental factors influencing health beyond climate change to include forest clearance, land degradation, biodiversity loss, freshwater depletion and damage to coastal reefs and ecosystems [28]. Climate change mitigation and adaptation strategies have widely differing planetary health implications. For example, afforestation and tree-planting programs, which create CO₂ sinks, also cause an imbalance in the water cycles of grasslands and their ecosystems [29,30]. Lithium mining in Chile, necessary for low carbon energy devices, has been criticized for depleting local groundwater resources across the Atacama Desert, destroying fragile ecosystems and converting meadows and lagoons into salt flats [31]. Considering the relationships among these global challenges may avoid the unintended consequences of addressing just one arm of planetary health.

1.2. The impact of climate change on cardiovascular disease

To date, review articles have centered primarily on the impact of pollution on CVD, with relatively few focusing on climate change as a broader topic [14,32–37]. This review broadens the focus of how climate change affects cardiac health beyond air pollution to include heat stress, infectious diseases, and other psychological and social factors (Fig. 1). Our specific aim is to evaluate, in the context of climate change, current CVD prevention lifestyle and pharmacological recommendations provided to patients by clinicians and other healthcare professionals and to conclude with practical suggestions for clinicians on how to use personal decisions, policy advocacy, and your role as a clinician communicator to help address climate change.

2. Air pollution

2.1. Epidemiology and pathophysiology of air pollution

Air pollution is defined as an unwanted, dangerous material introduced into the earth’s environment due to human activity [38]. It is composed of a mixture of gasses and particles, and the dominant source is fossil fuel combustion [32]. Particulate matter (PM) is categorized into coarse particles (PM10), which have an aerodynamic diameter of <10 μm, fine particles (PM2.5, <2.5 μm in diameter), and ultrafine particles (PM0.1, <0.1 μm). PM2.5 is the principal air pollutant contributing to CVD. The gaseous components of pollution are composed of primary gasses such as nitrogen oxides, sulfur dioxide, and carbon monoxide, and secondary gaseous pollutants such as tropospheric ozone (found at

![Fig. 1. Climate change and cardiovascular disease.](image-url)
ground level and not a component of the stratospheric ozone layer) generated by nitrogen oxides and volatile organic compounds reacting in the presence of sunlight.

Air pollution and climate change are intrinsically linked [39]. Fossil fuel combustion including “natural” gas [40], is the major cause of both problems. Increased temperatures and dry conditions have extended the fire season and increased the risk of wildfires [41]. Temperature tightly correlates with tropospheric ozone concentrations [42,43]. Increased temperatures are associated with stagnant atmospheric conditions [44–46], whereby light winds and a stable lower atmosphere prevent horizontal and vertical dispersion of airborne pollutants, while an absence of precipitation prevents the washing away of pollutants [47]. Air conditioning can help prevent heat-related deaths, but the current technology leads to further fossil fuel combustion [48].

Furthermore, associations between particulate matter and mortality, including cardiovascular mortality, are more robust with higher mean annual temperatures [49].

Mechanistic frameworks linking the pathophysiological effects of air pollution to CVD health begin with the deposition of PM2.5 in the lungs, where it interacts with cells and endogenous structures, both locally in the lungs and systemically across various vascular beds to initiate oxidative stress, low-grade inflammation, and create harmful biological intermediates (e.g., modified phospholipids) [33]. These primary initiating pathways activate subsequent effector pathways such as (1) endothelial barrier dysfunction; (2) systemic inflammation; (3) prothrombotic pathways; (4) autonomic imbalance; (5) hypothalamic-pituitary-adrenal axis activation; and (6) neural reflex arcs, all of which progress to the development of CVD risk factors such as hypertension [50,51], diabetes mellitus [52], dyslipidemia[53], and subclinical atherosclerosis [54]. Ultimately, clinical outcomes, such as

Fig. 2. Exposure-response curve for the relative risk of cardiovascular disease associated with long-term exposure to PM2.5.
cardiovascular death [55–57], myocardial infarction [58,59], stroke [60,61], arrhythmia [62] and heart failure [63] have all been linked to air pollution [32,33,38,64–68].

Three characteristics of air pollution are worth highlighting to aid in developing strategies to counteract its adverse cardiovascular effects:

- First, air pollution can be classified as ambient (outdoor) or household (indoor), which is vital in highlighting the differing sources, populations affected, and strategies required to improve health in these settings.
- Second, the exposure-response curve displaying CVD risk as a function of pollutant concentration rises steeply at low levels before flattening out at extreme concentrations, without a threshold below which exposure can be considered safe (Fig. 2) [33,69]. As a result, 99% of the global population breathes air pollution at levels that exceed the World Health Organization’s air quality guidelines, with low- and middle-income countries experiencing the highest exposures [70].
- Third, while chronic exposure to air pollution is similar to traditional risk factors, like hypertension, instigating chronic pathophysiologic processes that ultimately result in cardiovascular events, short-term (up to 7 days) exposure to even minimally increased concentrations of PM2.5 are also associated with increases in acute myocardial infarction (2.5% per 10 mg/m$^3$) and heart failure hospitalization or death [59,63].

The WHO interim targets (Its) are also highlighted. The Its are successive targets of 35, 25, and 15 μg/m$^3$, which were set up to reduce mortality. Exceeding IT-1 levels is associated with increased mortality by 15%. IT-2 and IT-3 reduce mortality by 6% at or below each level. For typical days in U.S. cities, the levels range within 24-h standards (5 to 15%). IT-2 and IT-3 reduce mortality by 6% at or below each level. For example, when the AQI is 101–150, people with cardiopulmonary disease, children, and older adults are advised to reduce prolonged or heavy exertion (such activities include playing basketball or soccer, chopping wood, heavy manual labor, and vigorous running, cycling, or hiking).

The core intervention against air pollution requires societal and governmental changes in shifting to renewable energy sources. Until that can be achieved, alternative policy interventions such as the promotion of low/zero-emission transportation and urban landscape reform, in combination with personal interventions such as minimizing commuting during rush hour, and the use of face masks and air purifiers should be employed. Some studies, primarily from Asian megacities, have found the use of facemasks such as the N95 respirator and air filters/cleaners to be associated with improvements in cardiopulmonary surrogate markers such as blood pressure [73,74]. There may be a particular benefit for those living near heavily trafficked highways and powerplants or during periods of poor air quality due to meteorological stagnation events or wildfires [75,76]. This initial data is promising and summarized elsewhere [77], but further trials of personal strategies to reduce air pollution exposure and improve health outcomes are warranted.

### 3. Heat

#### 3.1. Epidemiology of increasing temperatures

The impact of increasing temperatures on human society is dependent on both vulnerability and the magnitude of exposure to heat. Vulnerability refers to the propensity to be adversely affected by climate change and encompasses a variety of elements, including susceptibility to harm and lack of capacity to cope and adapt [78]. Vulnerability differs within communities, across societies, regions and countries, and may also change over time. For example, vulnerability is modulated by population age, access to air conditioning, overall health expenditure, and rates of CVD, obesity and mental health disorders [35,79–81].

Regarding heat exposure, the overall temperature-attributable excess mortality related to climate change is determined by the net effect of increased heat-related mortality and decreased cold-related mortality [82,83]. The frequency of heat waves, which are responsible for more significant mortality than any other extreme weather event in the U.S. and other parts of the world [84], is increasing in concert with mean temperatures [85,86]. Extreme cold weather conditions that accompany climate change also contribute to an increase in temperature variability that is expected to increase clinical cardiovascular events [87–91]. Although high-income countries will be better placed to prevent harm from some health consequences of climate change – like undernutrition, diarrheal disease, malaria or dengue – heat mortality is expected to increase, even in wealthy countries [89]. The European heat wave of 2003 was a devastating example of this, with a death toll exceeding 30,000 [94].

#### 3.2. Heat stress and wet bulb temperature

Heat stress refers to the environmental conditions, for example, temperature or humidity, that an individual is exposed to, whereas heat strain refers to the physiological response to these conditions [95]. The wet bulb temperature represents the temperature a thermometer (or the human body) may be cooled to by ventilation (convection of ambient air) and evaporation [96]. At 100% relative humidity, the wet bulb temperature is equal to the air temperature, so the human body can only be cooled by ventilation. The wet bulb temperature is lower than the air temperature at lower humidity, when the human body can be cooled by both ventilation and evaporation. Building on the wet bulb concept, the Universal Thermal Climate Index (UTCI) also incorporates wind and radiation [97].

According to the UTCI, mild heat strain starts at 23 °C with high humidity or 27 °C with low humidity. Exposures as short as 6 h to temperatures above 35 °C – just below body temperature – with high humidity can be lethal as neither ventilation nor evaporation is effective [98]. The National Weather Service issues Extreme Heat Warnings or Advisories based on the “Heat Index,” which is the temperature at a reference humidity level that produces the same discomfort as the temperature and humidity of the actual environment [99]. However, there is mixed evidence that such alerts prevent excess death, which may relate to heterogeneity in heat response plans that facilitate individual behavior change in response to these alerts [100,101]. Public health officials must work with the weather service and behavioral scientists to develop an effective system for recognizing risks and implementing an effective plan that engages the public.

#### 3.3. Physiology of heat stress

Humans cool themselves by dissipating heat with a combination of ventilation; conduction by direct contact with a surface; respiration by inhaling cool air and exhaling warm air; evaporation by sweating; and radiation. There is wide variation in an individual’s capacity to tolerate heat stress. The primary physiologic response to increased temperature involves endothelial vasorelaxation with increased cutaneous blood flow [93].
flow [102,103]. This mechanism is blunted in the elderly and those with hypercholesterolemia; the latter is thought to occur due to atherosclerosis-mediated decreases in arterial smooth muscle responsiveness to nitrovasodilators [104,105]. The shift in blood flow from the core to the peripheral circulation results in a reduction of intracardiac filling pressures and systemic vascular resistance, necessitating an increased cardiac output [106-108].

An increase in ejection fraction is frequently seen with heat stress; however, diastolic function is often impaired such that stroke volume decreases, and cardiac output is augmented primarily by heart rate [109]. These hemodynamic changes may be less well tolerated in those with poor cardiovascular reserve, particularly those with underlying cardiomyopathy and valvular heart disease, and those who use cardiac medications that slow heart rate [110]. Acute rises in red cell and platelet counts, blood viscosity and plasma cholesterol may help explain the increased mortality from arterial thrombosis during heat stress [111].

Heatstroke is a specific pathology defined as hyperthermia associated with a systemic inflammatory response leading to a syndrome of multi-organ dysfunction in which encephalopathy predominate [112,113]. Classic heatstroke typically occurs in elderly, chronically ill patients whose physiologic reserves are overwhelmed by heat stress. Patients with underlying CVD or taking medications such as diuretics and beta-blockers are at increased risk of heatstroke. Exertional heatstroke occurs in otherwise healthy individuals performing strenuous outdoor activities, such as laborers, athletes, and soldiers.

Initial management includes rapid on-site cooling with cold-water immersion for exertional heatstroke (before transporting to the emergency department). Conductive or evaporative cooling is used for classic heatstroke as cold-water immersion is thought less well tolerated by patients whose physiologic reserves are overwhelmed by heat stress. Heatstroke occurs in otherwise healthy individuals performing strenuous outdoor activities, such as laborers, athletes, and soldiers.

3.4. Strategies to minimise the impact of rising temperatures on CVD

As patients with CVD are at the highest risk for dying in a heatwave, it is prudent for clinicians to provide such patients with practical advice for coping with heat exposure [115,116]. Advice that is supported by scientific evidence includes to: (1) increase fluid intake and not wait for thirst to trigger drinking, particularly amongst the elderly; (2) remain in a cool or air-conditioned environment and wear loose-fitting clothes; (3) reduce normal activity levels; and (4) provide patients with "heatwave rules" specific to their cardiovascular medications. Examples could include careful home monitoring of their weight, blood pressure and symptoms of presyncope and halving their diuretic or antihypertensive dose on particularly hot days [81].

Finally, there should be a greater emphasis on educating the public on the risks of heat waves. In particular, the first heatwave of the season is usually the most dangerous for human health as individuals have not had the opportunity to develop thermal tolerance or acclimate [35,117,118]. Thermal tolerance refers to cellular adaptation with the accumulation of heat-shock proteins caused by a single, severe, nonlethal heat exposure. In contrast, heat acclimation refers to physiologic adaption as improved sweating, cutaneous blood flow, fluid balance and altered metabolism. Consequently, there is greater resilience against heat stress [119-121]. Employing preventive measures such as those described above during this first heatwave can prevent substantial heat-related morbidity and mortality when humans are particularly vulnerable.

4. Vector-borne disease

Climate-driven vector-borne illness affects other specialties to a greater extent however the cardiovascular system may be involved [122]. The incidence of Lyme disease, a climate change indicator used by the Environmental Protection Agency, has doubled since 1990 [123]. The *Ixodes scapularis* deer tick that predominantly transmits the bacteria in North America is most active when temperatures and humidity are above 7°C and 85% respectively [124,125]. Lyme carditis is an uncommon manifestation, occurring when *Borrelia burgdorferi* disseminates from the tick bite site in the skin, infecting heart tissue and causing inflammation [126]. The predominant cardiac manifestation is partial heart block, which usually is mild and resolves, though rarely progresses to complete heart block, which may be permanent without antibiotic therapy. Lyme myocarditis or pericarditis may also occur and can be fatal [127]. The same *Ixodes* tick is also responsible for transmitting the *Babesia microti* parasite, which infects erythrocytes [128]. Up to 10% of individuals presenting with babesiosis have evidence of decompensated heart failure, presumably due to anemia [129]. Babesiosis-associated myocarditis has also been described [130].

Between 8 and 12 million people worldwide are estimated to have Chagas disease [131], which results from infection with the protozoan *Trypanosoma cruzi* endemic to Central and South America. The infection is becoming more common in the U.S. primarily due to migration [132,133]. However, autochthonous infection i.e., spread between individuals in the same place (as opposed to importation or migration), has been acquired Arizona, Arkansas, California, Louisiana, Mississippi, Missouri, Tennessee, and Texas [134]—suggesting expanded climate-driven range. The triatomine insects involved in transmitting *T. cruzi* are becoming more common in these southern U.S. states, tend to develop faster and feed more often to avoid dehydration when temperatures are >30°C, resulting in this indigenous spread [135-137]. Manifestations of Chagas heart disease include dilated cardiomyopathy, ventricular aneurysm, ventricular arrhythmias and conduction disturbances (classically right bundle branch block) [109].

Temperature and humidity influence mosquito survival, frequency of blood feeding, and the development of parasites within mosquitoes [138]. Malaria, caused by *Plasmodium* protozoa and transmitted by *Anopheles* mosquitoes [139], is endemic in sub-Saharan Africa, Asia, Oceania, South and Central America. Fluctuations in autochthonous transmission of malaria have also been seen in areas like Greece and the Anhui Province in China [140-142]. Cardiovascular involvement is not commonly reported in patients with malaria, but is associated with considerable morbidity when present modulated by a dysregulated inflammatory cytokine response, endothelial dysfunction and red blood cell sequestration [143,144].

The *Aedes* mosquito native to Southeast Asia is involved in the transmission of both chikungunya and dengue virus. Over the last two decades, climate conditions have developed such that much of Europe is increasingly suitable for breeding the *Aedes* mosquito [145]. Chikungunya disease typically presents with fever and disabling arthralgias but can present with systemic involvement and more severe disease [146]. When this is the case, cardiovascular involvement typically presents acutely with myocarditis and arrhythmia, which may progress to chronic dilated cardiomyopathy [147]. Similarly, most dengue infections are relatively benign; however, severe presentations include a dengue-associated vasculopathy with hemorrhage, endothelial dysfunction with capillary leak and hypovolemic shock, and organ dysfunction [148]. Direct effects of the dengue virus on the heart include myocardial impairment through circulating myocardial depressant factors, myocarditis, and arrhythmias, of which relative bradycardia is a notable feature [149,150].

Similar increases in the number of West Nile virus cases, are expected with current climate projections [135]. Atypical or particularly severe presentations of West Nile virus with cardiovascular involvement have been described [151], and cardiologists will need greater awareness of the clinical presentation and increasing prevalence of these infectious diseases.
5. Mental health disorders

“Solastalgia” refers to the distress that is produced by environmental change [152], while climate anxiety refers to typical anxiety symptoms such as obsessive thinking, insomnia and panic attacks related to the global climate crisis and the threat of environmental disaster [153–155]. Some climate-related exposures, including heat and humidity and disasters such as droughts, wildfires and floods, are associated with substance use disorders, schizophrenia, mood disorders, anxiety and vascular dementia [156,157]. This is pertinent because several psychiatric illnesses, including schizophrenia [158] and anxiety [159] have clear associations with CVD. For example, major psychiatric illness and atypical antipsychotic medications are components of cardiovascular risk prediction algorithms such as the QRISK3 risk calculator [160]. Beyond the more significant burden of CVD mediated by chronic mental health conditions, a greater incidence of Takotsubo cardiomyopathy has been documented in response to extreme weather events [161,162].

6. Ocean health and CVD

Climate change results in increasing ocean temperature, higher sea levels, more acidic seawater, and greater levels of salinity with knock-on effects on the cardiovascular system. First, increasing sea levels has resulted in increased soil and potable water salinity with multiple adverse effects including increased rates of hypertension [163]; second, loss of fisheries impedes the public’s ability to follow the American Heart Association’s (AHA) recommendation to consume fish at least twice a week; third, microplastic accumulation such as bisphenol-A in our oceans is consumed by humans and is associated with increased CVD [164]. Addressing these impacts of climate change on our oceans may ultimately yield positive effects on cardiovascular health [165].

7. How climate change impacts a cardiologist’s practice and recommendations

7.1. Lifestyle

Reviews have not gone beyond synthesizing evidence of harm from climate change. Here we go further to provide actionable recommendations regarding lifestyle, review what limited evidence exists regarding pharmacologic therapies, and discuss the bidirectional relationship between the cardiovascular healthcare system and climate change.

Certain “health co-benefits” may be experienced with climate change mitigation and adaptation strategies, particularly exercise and diet (Fig. 3) [34,166,167]. Adopting a low greenhouse gas emission diet through decreased meat consumption from methane-producing animals such as sheep and cows is associated with reduced mortality, ischemic heart disease, and stroke [168–170]. Mediterranean-type diets, which are high in plant foods and emphasize plant protein sources, simultaneously decrease the risk of CVD induced by air pollution and minimize greenhouse gas production [171–176].

Physical activity, including work and commuting-related, leisure, and fitness purposes, increases longevity [179] and decreases the risk of CVD [180], hypertension [181], obesity[182] and diabetes [183]. Active transportation methods such as cycling and walking simultaneously address physical inactivity and (by reducing CO₂ emissions) climate change [184–187]. Urban planning more conducive to active transportation with decreased emissions may result in better cardiovascular health and reduce heat exposure due to increased greenspace with less asphalt. There are some concerns regarding outdoor exercise when air quality is particularly poor [188]. However, the cardiometabolic health benefits of active commuting outweigh the increased health risks of traffic-related pollutants in all but the most extreme pollution cases [189,190]. High temperatures can also reduce the frequency and duration of physical exercise that can be tolerated [191,192]. As a result, sports medicine authorities in many countries, including the US, suggest postponing outdoor competitive events if the wet-bulb temperature is above 28 °C [193,194]. Cardiologists and primary care physicians should advocate for community designs that emphasize areas where individuals can exercise under natural foliage and away from polluting foci such as busy roads and industrial areas. Cigarette smoking and nicotine ingestion increase physiologic strain during heat exposure and may increase the risk of heat stress [195].

Finally, the AHA defines inadequate sleep duration as <7 h per night and is recognized as an important cardiometabolic risk factor [196,197]. In large prospective studies, healthy, nonobese adults, who reported <6 h of sleep a day were at increased risk of myocardial infarction, obesity, dyslipidemia, hypertension and elevated fasting glucose [198,199]. Sleep disruption has also been reported as a result of climate-related temperature changes and climate-related stress responses [200,201].

Sound pollution from industry and motor vehicles (particularly those...
with combustion engines) further contributes to poor sleep. Encouraging adequate sleep is yet another health co-benefit in that our patients’ metabolic health will benefit from more sleep. A sleeping human tends to consume less energy resulting in less GHG emissions.

7.2. Medications

Commonly prescribed cardiovascular medications impact our patients’ susceptibility to heat-related illness [202–204]. Diuretics, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, and sodium-glucose cotransporter-2 inhibitors increase the risk of hypovolemia and heatstroke [205–207]. Beta-blockers decrease blood flow to the skin and reduce cardiac output, also placing individuals at higher risk of heatstroke [208].

Aspirin and clopidogrel use results in attenuated reflex cutaneous vasodilation, which may elevate core temperatures in the setting of both passive and exertional heat stress [209]. Conversely, there is some evidence that aspirin may inhibit heat-induced platelet hyperaggregability involved in DIC secondary to heat stroke [210], and aspirin pretreatment has been found to prolong survival in rats who sustain heat stroke [211]. Aspirin does not appear to modify the effect of PM exposure on ischemic stroke [212].

Statin medications may be thermo-protective primarily via cutaneous vasodilation [213,214], achieved by stabilizing the essential cofactor tetrahydrobiopterin, necessary for nitric oxide production [215,216]. An analysis of a large U.S. Medicaid program found a protective association of statin use against all-cause mortality which strengthened as daily average and daily maximum temperature increased [217]. Preclinical studies have suggested that the inflammatory response induced by exposure to ambient air pollution may be nullified by statin therapy; however, this has not been borne out by observational data [218,219]. By understanding how medications impact heat and pollution sensitivity, clinicians can give individualized recommendations regarding their patient’s risk during extreme exposures.

Certain medications are particularly heat-sensitive. For example, the efficacy of PCSK9-inhibitors significantly decreases when stored at temperatures higher than 25°C for several hours [220], and insulin has long been recognized as a heat-sensitive medication that undergoes degradation at temperatures above 30°C. With increasing temperature and temperature variability, those most disadvantaged will not have the resources to store their therapeutics correctly. Medicines stable at higher room temperatures are needed to avoid refrigeration, benefitting those without access to such appliances.

7.3. Preparing the healthcare system for climate change

Cardiovascular specialists need to work with hospital administrators in the healthcare system in which they practice to prepare for the increasing impact of climate change. Disruption of healthcare infrastructure, due to events such as wildfires, floods and hurricanes is already affecting a substantial amount of healthcare delivery [221]. Desertification, ocean warming and acidification threaten the availability of healthy diets. Health systems should invest in disaster planning and early warning systems to prepare for waves of patients in extreme weather events or climate refugees [34]. Plans to access and provide nutritious food should also be developed.

While climate change has significant impacts on health care delivery [222], the inverse is also true [223]. The U.S. healthcare system accounts for 8.5% of all U.S. GHG emissions and, if it were a country, would rank 13th in the world in terms of emissions [224]. Steps to eliminating GHG production in the healthcare sector include reducing demand through preventive care, use of clean energy, choosing medical supplies and equipment with lower carbon footprints, and employing telemedicine where possible [225,226]. Cardiac catheterization labs and intensive care units are particularly carbon intense environments that require significant sustainability assessment and, where we should be especially sensitive to the consequences of providing futile care [227].

7.4. Personal and policy action items

Climate health literature is dominated by impact research, however mitigation and adaptation responses remain niche topics [228]. Here we propose a list of actionable items that physicians may implement in their personal and professional capacity to reduce their impact on climate change (Table 1). Incorporating personal action items such as flying less and eating a plant based diet are important short term strategies to engage with other individuals and convince them of the important changes that need to occur. However, these actions alone are grossly inadequate, without wider communication or policy action [229]. Instigation of personal action items in the short term may also counter the “governance trap” wherein the public and governments each seek to attribute responsibility for instigating change to the other [230].

7.5. Climate change communication

We have an opportunity to adapt our practice to protect our patients in this changing climate and use our voice to influence the mitigation and adaptation strategies required to combat climate change. The public’s prioritization of climate change fluctuates. At the beginning of the last decade, it has fallen below that of the 1980s [230,240]. The discrepancy between the increasing scientific certainty of anthropogenic climate change and a decreasing public concern for the issue is known as the “psychological climate paradox” [241]. An information deficit does not explain this, but rather cognitive biases and social influences that prevent the facts about climate change from being internalized and influencing behavior [242]. One such critical influence has been the

| Table 1 |
| --- |
| **Actionable items physicians can implement in their personal and professional capacity.** |
| **High Impact Personal Actions for Individuals in High-Income Countries** [231]: |
| ○ Fly less. |
| ○ Drive less, in a more efficient car, or do not drive. |
| ○ Eat a plant-based diet, be less wasteful with food. |
| ○ Make your home more energy efficient. |
| ○ Dress and shop sustainably. |
| ○ Consider having fewer children. |
| ○ Consider choosing carbon friendly pets. |
| **Policy, Advocacy and Media Action** [232]: |
| ○ Legislative advocacy: E-mail, call or meet with your local, state or federal representatives about the health effects of climate change. Support candidates committed to addressing climate change. Testify at hearings. Join or follow advocacy groups to keep informed in terms of legislation, such as “Physicians for Social Responsibility” [233] or the “Medical Society Consortium on Climate and Health” [234]. |
| ○ Determine if your hospital system has a climate solution plan, and if not, advocate or work to ensure one is developed [235]. |
| ○ Engage in non-violent social protests to address the climate emergency [236]. |
| ○ Pen an Op-Ed or write letters to the Editor about the connection between climate change and health after extreme weather events. |
| **Climate Change Communication** [237]: |
| ○ Open up the conversation with patients or colleagues whenever there is a significant weather abnormality. Food, tropical storms, heatwave, wildfire. |
| ○ Highlight the health co-benefits of a low greenhouse gas lifestyle and diet. |
| ○ Discuss the health effects of climate change as a matter of routine when discussing other health maintenance. |
| ○ Encourage high-risk patients to make personal disaster action plans during hot-weather or disaster seasons (e.g., wildfire season, hurricane season). |
| ○ When a patient presents with a complication of climate change, alert them that this is the case. |
| Syncope due to heat. |
| Acute kidney injury due to hypovolemia. |
| Asthma or COPD flares due to worsening air pollution. |
| Myocardial infarction or heart failure exacerbation due to particulate matter or wildfire smoke exposure. |
Table 2 Glossary.

| Term                        | Explanation                                                                 |
|-----------------------------|-----------------------------------------------------------------------------|
| Air pollution               | Unwanted, dangerous material that is introduced into the earth’s environment as a result of human activity |
| Air Quality Index           | A tool for reporting air quality and is calculated based on the ambient air concentrations of five major pollutants |
| Anthropocene                | The most recent period in the earth’s history when human activity significantly impacted the planet’s climate and ecosystems. |
| Autochthonous transmission  | Spread of a microorganism between individuals in the same place (as opposed to importation or migration) |
| Climate forcing             | Any influence on climate that originates from outside the climate system itself, for example, GHG or surface reflectivity (as opposed to radiative forcing as below) |
| Climate change adaptation   | The process of adjustment to actual or expected climate                        |
| Climate change mitigation   | Actions that reduce the rate of climate change by decreasing the rate of GHG emissions and increasing the rate of GHG removal [6] |
| Health co-benefits          | Climate change mitigation activities that also provide health gains          |
| Heat acclimation            | Physiologic adaptations including improved sweating, cutaneous blood flow, fluid balance and altered metabolism, and consequently, greater resilience against heat stress |
| Heat index                  | The temperature at a reference humidity level produces the same level of discomfort as the temperature and humidity of the actual environment. |
| Heat strain                 | The physiological response to environmental conditions that an individual is exposed to |
| Heat stress                 | The environmental conditions, for example, temperature or humidity, that an individual is exposed to |
| Heatstroke                  | A form of hyperthermia associated with a systemic inflammatory response leads to a multi-organ dysfunction syndrome in which encephalopathy predominates. An entirely separate entity to a cerebrovascular accident |
| Heatwave                    | A series of unusually hot days. Variably defined, but the EPA defines as two or more consecutive days when the daily minimum apparent temperature (the actual temperature adjusted for humidity) in a particular city exceeds the 85th percentile of historical July and August temperatures (1981–2010) for that city |
| Intergovernmental Panel on Climate Change (IPCC) | The international body set up by the United Nations provides reports on the scientific basis of climate change, its impacts and future risks, and options for adaptation and mitigation |
| Particulate matter (PM)     | The particle component of air pollution is categorized based on the diameter of the particle |
| Planetary health            | Refers to the health impacts of disruption to the earth’s natural systems caused by humans |
| Psychological climate paradox | The discrepancy between the increasing scientific certainty of anthropogenic-driven climate change and a decreasing public concern for the issue |
| Radiative forcing           | The difference between incoming and outgoing radiation is known as a planet’s radiative forcing (as opposed to climate forcing as above) |
| Representative Concentration Pathway (RCP) | RCPs are pathways that provide time-dependent projections of atmospheric greenhouse gas (GHG) concentrations [238] |
| Shared Socioeconomic Pathway (SSP) | SSPs are reference pathways describing plausible alternative trends in the evolution of society and ecosystems over a century timescale, in the absence of climate change or climate policies [239] |
| The Universal Thermal Climate Index (UTCI) | Provides an assessment of thermal strain in human incorporating temperature, humidity, wind and radiation |
| Thermal tolerance           | Cellular adaptation with the accumulation of heat-shock protein caused by a single, severe but nonlethal heat exposure |
| Tropospheric ozone         | One of the gaseous components of air pollution found at ground level (as opposed to stratospheric ozone, which protects life on Earth from harmful ultraviolet radiation) |
| Vulnerability               | Vulnerability refers to the propensity or predisposition to be adversely affected by climate change and encompasses a variety of concepts and elements, including sensitivity or susceptibility to harm and lack of capacity to cope and adapt [78] |
| Wet bulb temperature        | The temperature to which a thermometer (or the human body) may be cooled by ventilation and evaporation |

concerted efforts of high emitting industries to influence the discourse on climate change, similar to big tobacco and the risks of smoking [243].

Humans tend to struggle with future, uncertain (or perceived to be debatable), and costly to correct (both financially and non-financially) problems [244]. Unfortunately, climate change is all three, which partially explains our inability to confront the issue. Climate communication must address these barriers by making the climate issue more personal, nudging the public towards action to minimize cognitive dissonance and denial, providing a narrative of opportunity and giving meaningful indicators on progress, for example, the proportion of reduction in CO₂ emissions, or share of fossil fuels in total energy use [241,245].

Patients and the public respond to simple, clear messages repeated often and from trusted sources. Physicians have the experience and skills to communicate messages to influence their patients’ behaviors. For example, convincing a patient to take a statin deals with an uncertain and potentially costly future cardiovascular event. Yet, patients will regularly leave their cardiologist’s office assured that this is necessary.

In 2020 many clinicians embraced a new role by rapidly educating themselves on the health effects of COVID-19 to allow them to provide reliable public health information [246]. Up to 75% of physicians report that it is their responsibility to inform patients about the health impacts of climate change [232,247]. Changing current behaviors should be the immediate priority, while larger-scale policy and regulatory solutions should be the focus long-term [248]. This is our opportunity to impress upon our patients the health impacts of climate change and provide guidance on what needs to be done to address the greatest public health challenge of the 21st century [249,250].

8. Conclusion

Cardiovascular disease related to climate change can be prevented. This review has summarized the pathophysiology of cardiovascular disease associated with air pollution, heat and other medical conditions. Evidence-based recommendations relating to lifestyle, medication, cardiovascular care, and communication have been devised so that clinicians and other healthcare professionals may take action to prevent climate-change-associated heart disease among their patients.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.
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