The Effects of Climate Change on Cardiac Health

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Key Words

Climate change · Cardiac health · Heat exposure

Abstract

The earth's climate is changing and increasing ambient heat levels are emerging in large areas of the world. An important cause of this change is the anthropogenic emission of greenhouse gases. Climate changes have a variety of negative effects on health, including cardiac health. People with pre-existing medical conditions such as cardiovascular disease (including heart failure), people carrying out physically demanding work and the elderly are particularly vulnerable. This review evaluates the evidence base for the cardiac health consequences of climate conditions, with particular reference to increasing heat exposure, and it also explores the potential further implications.

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ergy-trapping gases (greenhouse gases) emitted by burning fossil fuels like coal, oil and natural gas, and the methane emitted by ruminant agricultural animals like cows and sheep. The earth's average temperature has increased by approximately 0.85°C in the 20th century and most of this warming has occurred since 1975. The rate of global warming has accelerated to over 0.18°C per decade over the last 30 years. Changing temperature is the most valid model output of climate change, so any important effects on health that are related to ambient temperature can be analyzed with some certainty. Other expected climate change features, such as more violent and extreme weather events, the rise in sea level and the lack of water and food in large areas are also of importance to population health, but are not as much related to cardiac health as the increasing heat. This paper will focus on the effects of increasing ambient heat on cardiac health.

Introduction

Human activity is contributing to the altering of climate on our planet [1]. In part, the climate changes are due to the increasing atmospheric concentrations of en-

Climate and Health

A number of different health-hazard exposures and associated effects on health are due to climate conditions. If a health condition is influenced by climate, it could, of course, also be affected by climate change in a particular

Table 1. Climate change-related health impacts according to the IPCC, WHO and other sources

Exposure hazard	Health impact	Confidence ^a	Link to specific effects at the organ level	Source
Intense heat	death from heat stroke	very high	heart strain, CNS malfunction, dehydration	IPCC (2014) [61] ^a WHO (2014) [62] ^b
	heat stroke morbidity	very high	heart strain, CNS malfunction, dehydration	IPCC (2014) [61]
	heat exhaustion loss of work capacity	high	heart strain, mental fatigue	IPCC (2014) [61]
Failed local agriculture	undernutrition	high	metabolic energy loss, heart strain, loss of work capacity	IPCC (2014) [61] WHO (2014) [62]
Lack of safe water and food	water and food-borne diseases	very high	infectious intestinal diseases, diarrhoea	IPCC (2014) [61] WHO (2014) [62]
Changed ecology	vector-borne diseases	medium	malaria, dengue fever, other insect-borne diseases	IPCC (2014) [61] WHO (2014) [62]
Reduced temperatures	cold-related mortality/morbidity	low	heart strain, but respiratory disease is more prominent	IPCC (2014) [61]
Storms/floods/droughts/fires	injuries, drowning, burns	very high	injuries (any organ can be affected)	IPCC (2014) [61]
Forced migration	undernutrition, infectious diseases, mental stress	high	disease, violence and mental health problems	IPCC (2014) [61]
<i>Health concerns not mentioned in detail in the IPCC or WHO reports</i>				
Intense heat	chronic kidney disease linked to dehydration			Wesseling et al., 2012 [63]
	teratogenic effects of high body temperature in pregnant women, damage to brain development			Edwards et al., 1995 [64]
	interactions with prescription drugs			Vanakoski and Seppälä, 1998 [56]
	deteriorated clinical status of people with chronic non-communicable diseases			Kjellstrom et al., 2010 [7]; Parsons, 2014 [2]
Changed pollen amounts	allergies due to pollen			USEPA, 2008 [65]
Mental stress from local impacts	mental health problems, suicides, increased violence			Berry et al., 2010 [66]
Specific hazards for working people	non-heat-related health risks: vector-borne diseases, undernutrition, poisoning in the workplace			Bennett and McMichael, 2010 [67]
Societal collapse	violence, mental stress, daily needs not supplied			Butler and Harley, 2010 [68]
CNC = Central nervous system.				
^a The IPCC 2014 assessment judgement of the confidence for the impact of climate change [61].				
^b The WHO 2014 report on climate change-related mortality [62].				

area. Table 1 highlights the effects on health presented in the assessment by the Inter-Governmental Panel on Climate Change (IPCC) in 2014 [1] and a World Health Organization (WHO) report published in the same year. Effects on heart health are primarily linked to intense heat exposure and undernutrition. The expected reduced exposure to intense cold may have a positive impact on heart diseases, but the link between cold and health is stronger for respiratory diseases and the areas of the world where the effects of cold on health are occurring have a much smaller population than the tropical areas where the intense heat is already a real health problem. It is worth noting that the main causes of mortality involve the heart (fig. 1).

Table 1 also includes some health concerns that have been given very little attention so far in health impact analysis reports on climate change. This includes the epidemic of chronic kidney disease among agricultural workers in Central America, which is most likely linked to daily dehydration in workers who do not get sufficient drinking water relative to how much they sweat. Another major issue for women in low-income, tropical countries is the teratogenic effect of body temperature $>39^{\circ}\text{C}$, which has been known in veterinary medicine for several decades.

The effects on health of extremely high or low temperatures are well known [2], and cardiac effects are a major component. Both excessively low and high tem-

Table 2. Calculated mortality for the whole world according to the WHO 2014 report [62]

Year	Undernutrition of children	Malaria	Diarrhoeal disease in children	Heat mortality among the elderly (>65 years)
2030	+95,176 (–119,807 to +310,156)	+60,091 (37,608 to 117,001)	+48,114 (21,097 to 67,702)	+37,588 (26,912 to 48,390)
2050	+84,697 (–29,203 to 163,989)	+32,695 (22,786 to 40,817)	+32,955 (14,914 to 49,151)	+94,621 (70,775 to 126,684)

peratures affect cardiac diseases [2], and climate change can therefore affect local patterns of heart disease in several ways. The effects of climate change on health are expected to be negative [1]. The basic requirements for maintaining health – peaceful societies, clean air, sufficient clean drinking water, sufficient food and adequate shelter – may all be affected by the changing climate [3, 4]. For instance, people will be exposed to higher temperatures in the local climate where they live and work [2]. The impact of climate conditions, in particular ambient temperature, both now and in the future, will vary according to local vulnerabilities, geographical and socio-political situations and the promotion of protective measures [5]. Elderly people [4], workers carrying out heavy physical labour in non-cooled environments [6] and people with pre-existing medical conditions – such as cardiovascular (CV) disease and heart failure (HF) – are expected to be particularly vulnerable to increasing temperatures [7].

Many of the effects of climate change on general health are mediated via CV effects, with patients with pre-existing heart conditions being among the most susceptible [4]. An example of the type of assessments available is the WHO report on mortality in relation to climate change (table 2). This analysis took into account the changing population size in different parts of the world and used socio-economic models to assess the likely change in access to prevention for health problems such as undernutrition and diarrhoeal diseases among children, malaria in the whole population and heat mortality among the elderly. It is noteworthy that in spite of continuing climate change until 2050, the global mortality due to climate change is calculated to be less in 2050 than in 2030, except for heat mortality amongst the elderly. As this mortality is very much associated with acute HF as an effect of intense heat stress, it is an important factor to consider for cardiac health in the future.

Climatologists forecast further warming ('heating' is a better description in tropical areas) in the years to

Fig. 1. The IPCC is the world body for assessing the science related to climate change. Confidence levels of different health effects becoming future threats are depicted [61].

come, increased climatic variability and changes in precipitation and humidity [1]. We should point out that impacts on cardiac health are associated with the local climate, and special local assessments may be necessary to develop suitable prevention programs (local climate change and health impacts data are available on this website: www.ClimateCHIP.org). According to the phenomenon of the urban heat island [1], additional heat exposure will need to be considered, as urban development trends and urban design influence the local heat exposures. The population size in a geographic area is of course of importance for the overall impact on the health of the population, but the overall impact on local populations is also influenced by the internal heat stress caused during physical labour.

U-Shaped Relation between Temperature and All-Cause Mortality and CV Events

There is a clear U-shaped (or V-shaped) relationship between temperature and all-cause mortality [8]. Mortality increases from the 'optimum temperature', with different slopes at the cold and hot ends according to latitude [8]. In the moderate climate of the Netherlands [9], the lowest mortality has been found to occur at a daily average temperature of 16.5°C, with CV deaths accounting for 57% of cold-related mortalities. According to the Eurowinter study [10], the 3°C band with the lowest mortality varies between regions. The mortality is lowest between 14.3 and 17.3°C in north Finland, 19.3 and 22.3°C in London and 22.7 and 25.7°C in Athens [11]. In Taiwan, with a relatively hot summer season, a right-shifted, U-shaped relation between outdoor temperature and mortality from coronary artery disease has been described, with a rightward shift of the lowest mortality temperature range (26–29°C) compared to that of countries with colder climates [12]. In Beijing, extreme cold and hot temperatures were also found to be significantly associated with an increased risk of mortality from coronary heart disease, with women and anybody ≥65 years being more sensitive [13]. In a part of France with relatively cool summers (the city of Lille), a 10°C decrease in mean temperature was associated with a 13% increase in coronary events, and this association was more pronounced in older patients [14]. In 15 conurbations in England and Wales, a 1°C reduction in temperature would cumulatively increase the risk of myocardial infarction by 2.0%, with the strongest effect at time lags of 2–7 and 8–14 days. Adults aged 75–84 years and patients with previous coronary artery disease seem to be more vulnerable [15]. Few studies are available from countries with very hot seasons, but the impact on mortality of hot days then exceeds the impact on cooler days [16].

Heat and CV Morbidity and Mortality in Epidemiological Studies

One study found that, in 12 US cities, hospital admissions for CV disease and myocardial infarction increased in synchrony with the average temperature on the same or the preceding day [17]. During the London heat wave of 1976, daily deaths from coronary thrombosis nearly doubled [18]. Daily mortality of patients with congestive HF in Montreal was found to be strongly associated with

the maximum daily temperature, with a strong exponential increase starting at around 25°C [19]. Confinement to bed, a pre-existing heart condition and/or a pulmonary condition were identified as risk factors for heat-related deaths and deaths due to CV causes during the 4-day heat wave in Chicago in July 1995, and the strongest protective factor was having access to air-conditioned environments [20]. In a meta-analysis, CV illness emerged from amongst pre-existing medical conditions as one of the factors associated with higher risk, and home air-conditioning was the factor associated with the lowest risk of death [20]. More recently, Christenson et al. [21] conducted a study on a case series of 27 heat-related fatalities that occurred during the summer of 2012. All of the cases had ≥1 known risk factors, 70% were >65 years old, 75% had a CV disease and 52% had a mental health condition. Of note, none of them had access to functioning residential air-conditioning.

Basu and Malig [22] examined mean daily apparent temperature and mortality in 13 counties in California during the warm season. They found an excess risk of 4.3% per 5.6°C increase in apparent temperature for non-accidental mortality, and a similar risk for individuals with CV and respiratory diseases, with no evidence of mortality displacement. Ownership and usage of air-conditioning significantly reduced the effects of temperature [23]. Hajat et al. [24] demonstrated that, in Delhi, where the mean temperature is 25°C, an increase of 2–4% in all-cause and CV mortality was observed with a lag time from the same day to 1 week before death.

Heat Stroke and Heat Stress

It is expected that heat waves will occur more frequently and will be of longer duration as a consequence of the ongoing climate change [25]. Heat waves are associated with increased mortality, and in hot areas of the world, the risk of heat stroke is present even on 'normal' heat exposure days. Some of the increase in mortality is directly due to the increase in heat stroke, but the number of deaths of patients with pre-existing medical conditions is also higher than usual. Heat stroke can be defined, as proposed by Bouchama and Knochel [26], as follows: 'a form of hyperthermia associated with a systematic inflammatory response leading to a syndrome of multi-organ dysfunction in which encephalopathy predominates'. Heat stress is normally associated with an acute CV response in which there is a shift of the heated blood from the core circulation to the peripheral circu-

lation [27]. When cardiac output cannot increase to meet the need for sufficient heat loss, heat tolerance is impaired. Medications that interfere with the salt and water balance, like diuretics, predispose to heat stroke [26].

People carrying out heavy labour are at particular risk, due to the surplus internal heat created by muscle work [6]. Some of the earliest and most detailed studies in field situations, including physiological variables, were carried out in the middle of the 20th century in the UK, Germany and South Africa [2]. Both fatal and non-fatal heat stroke incidence data were presented as well as data on the loss of labour productivity due to heat exposure [6]. The physiological limits of the cardiac system to maintain an efficient oxygenation of muscles is a likely cause of loss of productivity, but the central nervous system is also involved in 'fatigue' [2].

Experimental and Clinical Studies on Heat Stress in Humans

The physiological reactions to heat in current temperature conditions have been studied and analyzed in many experimental settings. Many studies have focused on young and healthy volunteers, particularly sports people and military staff, while studies on the most vulnerable groups are few, for obvious ethical reasons. It has been shown that cutaneous [28] and systemic [29] vascular conductance is significantly increased during heat exposure, thus ameliorating the increase in core temperature. This, on the other hand, imposes an acute demand on the CV system by recruitment of cardiac reserve. Indeed, whole-body heating increases the heart rate and the cardiac output by up to 7–10 l/min [29–31] and decreases central blood volume, right atrial pressure [29], mean pulmonary artery pressure, pulmonary capillary wedge pressure [31], left atrial volumes [32], aortic mean pressure, stroke volume [33] and total peripheral resistance (–52%) [29].

Heat stress also reduces right- and left-ventricular end-diastolic volumes and the volume of blood in the heart (18%), thorax (14%), inferior vena cava (23%), liver (22%) and spleen (27%) [34]. The hepatic-splanchnic blood flow declines and the estimated total splanchnic vascular resistance rises by 38% [30]. During orthostatic challenge, decreases in CV pressure are correlated with decreases in pulmonary capillary wedge pressure under all thermal conditions [31]. However, an experimentally induced lower body negative pressure induces a greater

decrease in stroke volume during heating than during normothermia and cooling, causing the operating point on the Frank-Starling curve to move to a steeper portion where small changes in pulmonary capillary wedge pressure result in large changes in stroke volume [35]. To meet the increased demand for cardiac output, systolic function is increased. The septal and lateral mitral systolic annular velocities are also significantly increased [28, 32]. Peak twist, peak systolic circumferential strain [31] and left-ventricular ejection fraction all increase [34], but stroke volume nevertheless decreases [32], and hence the augmentation of cardiac output is entirely the result of an increased heart rate.

Despite the known associated lowering in filling pressures, the preservation of the diastolic indices during heat stress suggests that diastolic function is also impaired [29]. Indeed, early diastolic mitral inflow velocity (E wave) remains unchanged, but late inflow velocity due to atrial contraction (A wave) is significantly increased, with a significantly decreased E/A ratio [29]. Early diastolic mitral annular septal and lateral velocities (e') are unchanged (in contrast to studies of unloading with lower body negative pressure) with unchanged E/ e' ratios, and the late diastolic annular velocities (a') are increased. The left-ventricular untwisting rate is significantly increased [29]. Heat stress also results in increases in the counts of platelets, red blood cells and neutrophils, cholesterol levels and blood viscosity [18]. The physiological response to heat in patients with HF is abnormal, with a significantly lower flow of blood in the skin and lower cutaneous vascular conductance responses, even if the maximal vasodilatation induced by sodium nitroprusside is not reduced [36, 37].

Physiological Adaptations to Temperature Extremes

Most of the effects described above in response to heat stress were observed during acute exposure. Epidemiological evidence demonstrates that populations less likely to be exposed to either temperature extreme are more vulnerable [38]. The first heat wave of the summer is usually associated with the greatest number of heat-related fatalities. Repeated exposure to heat stress greatly increases maximum sweat production and a given submaximal rate is achieved at a lower skin or core temperature [4]. Cellular adaptation and self-preservation from heat stress are associated with the accumulation of heat-shock proteins (Hsp) [39, 40] that act as molecular chaperones, protecting the process of protein assembly and folding [41]

and playing an anti-apoptotic role. Thermal pre-conditioning refers to tolerance to a subsequent thermal stress that is induced by a sublethal first thermal stress. It is conferred within hours and can last for up to 5 days [41]. Several cyto-protective mechanisms other than the sole induction of Hsp are probably involved in heat-stress pre-conditioning. Exercise can induce Hsp production, but an increase in core temperature is an essential component [41]. In animal models, the protection conferred by heat-stress pre-conditioning also protects against myocardial ischemia [42]. Polymorphisms in Hsp could also be implicated in different individual risks for ischemic heart disease [43].

Possible Cardiac Health Co-Benefits due to Greenhouse Gas Reductions

Ongoing efforts to mitigate the extent of climate change by reducing the emission of greenhouse gases appear to be an opportunity to modify lifestyle habits on a large population scale. For instance, reducing the use of motor vehicles by increasing physical activity as an alternative means of short-distance commuting would be expected to have general and cardiac health benefits [44–46]. The reduction in emissions of transportation-related greenhouse gases would be accompanied by a diminution of other air pollutants that have direct adverse effects on health.

In a review of the different ‘health co-benefits’ of greenhouse gas reduction policies [45], reduced consumption of ruminant meat (e.g. from sheep and cows) would also contribute to the mitigation of climate change and the protection of cardiac health. The same applies to an increase of the use of renewable sources of energy (e.g. solar radiation or wind power) for the production of electricity [45], the result being less urban air pollution from fossil fuel-powered electricity and lower risks of heart disease [45].

The term ‘active transport’ refers to self-transport that involves more physical activity than in transport by private motor vehicle. It encompasses walking and cycling, often in combination with public transport. The use of public transport such as buses, trains, underground trains or boats often requires a significant distance to be covered by walking or cycling as part of the trip. Daily exercise can be performed in manners other than by active transport, but for many people, including this in their daily commuting is the simplest and most natural method. According to a study in the USA, each extra kilometer that a per-

son walks per day is associated with a 4.8% reduction in the likelihood of obesity, hence the slogan coined by the American Heart Association in 2012: ‘Every day, walk the dog. If you have no dog, walk anyway.’ Each extra hour a person spends in a car per day is associated with a 6% increased risk of obesity [46].

A study in Copenhagen showed that, after adjustment for age, sex, educational level, leisure time activity, body mass index, blood lipid levels, smoking and blood pressure, individuals who cycle to work had a relative risk for all-cause mortality of 0.72 [47]. In another study from Finland [48], women who walked or cycled ≥ 15 min to and from work had a significantly reduced risk for CV disease and all-cause mortality. Walking has been shown to be associated with substantial reductions in the incidence of CV events among post-menopausal women [49]. In the Shanghai Women’s Health Study [50], cycling for transportation was inversely and independently associated with all-cause mortality. Conversely, Wennberg et al. [51] demonstrated that after multivariate adjustment, regular commuting by car remained associated with an increased risk (OR 1.74) of myocardial infarction compared to commuting by bus or cycling or walking. A recent review [52] identified 3 high-quality studies [53–55] on the economic benefit/cost ratio of transport infrastructure and policies that include the health effects related to cycling and walking. They concluded in favour of a positive benefit/cost ratio. Sælensminde [54] estimated that the benefits of investments in cycle networks in three Norwegian cities were at least 4–5 times the cost.

CV Drugs and Heat Exposure

Very few studies have assessed the independent role of different CV drugs in the context of exposure to different climatic conditions. Only a few studies have assessed the effect of heat exposure on pharmacokinetics in humans [56]. Although it is biologically sound to consider that diuretics are harmful in the context of heat stress, we found very little evidence to support this.

In a retrospective study on 94 adult patients admitted with heat-related illness following the heat wave of February 1993 in Adelaide, South Australia, significantly more patients with heat stroke were taking diuretics at presentation than patients with only heat exhaustion [57].

Sommet et al. [58] analyzed serious adverse reactions to drugs that were reported to the network of the French pharmacovigilance centres during the summers (with

heat waves) of 2003 and 2006, and then compared these with reactions that occurred during the summers of 2004 and 2005 as a reference. The authors found that more serious adverse drug reactions were reported in 2003 and 2006, and they were able to link them to the two heat waves. The drugs most frequently involved were diuretics, serotonic antidepressants, angiotensin-converting enzyme inhibitors and proton-pump inhibitors. Diuretics were more frequently involved, and, in contrast, beta-blockers were less frequently involved in 2006 compared to 2003.

Hausfater et al. [59] performed a multi-center, observational cohort study of 1,456 patients with a core temperature of $\geq 38.5^{\circ}\text{C}$ who were admitted to 19 emergency departments in the teaching hospital network of Paris during the heat wave of 2003, i.e. between the 5th and 14th of August. The survival rate at 1 year was 57%. After multivariate analysis, 11 variables were found to be prognostic, among them: an age of >80 years (Hazard Ratio [HR] 1.45, $p = 0.004$), the use of diuretics (HR 1.25, $p = 0.037$), living in an institution (HR 2.07, $p = 0.006$) and a systolic blood pressure of <100 mm Hg (HR 2.36, $p < 0.001$).

How to Help Patients Cope with Intense Heat Exposure

A comprehensive review on the evidence base for the most commonly provided advice for protection from heat has recently been published [60]. As general advice, an increase in fluid intake, including drinking without waiting for thirst, in elderly people is encouraged. People at risk should avoid the hottest environments, wear loose-fitting clothes and take frequent showers or baths. It is also recommended that physical activity be reduced during hot weather, and exposed people should be made aware of the symptoms of heat exhaustion and heat stroke. Working people will thus reduce their hourly labour productivity; this could become a serious economic loss as climate change brings on more hot days [6]. The current evidence does not facilitate the issuing of clear-cut recommendations regarding the use of CV drugs, but it suggests increasing the monitoring of patients and adjusting doses of/temporarily suspending CV drugs accordingly, with special attention being given to diuretics, beta-blockers, renin-angiotensin-system inhibitors and anticholinergic drugs.

The largest mortality effect of high temperature occurs on the day of exposure continuing up to 3 days after ex-

posure. A predicted heat wave should prompt a rapid response to prevent heat-related deaths in vulnerable subgroups such as the elderly [22].

Conclusion

The ongoing climate changes are (at least in part) secondary to anthropogenic activities and are expected to continue, even in the 'best-case scenario'. Environmental stimuli do have an influence on cardiac health and there is a well-defined U-shape curve relation between temperature and cardiac event outcomes. Increased climate change-related heat exposure is associated with an increase in cardiac events. Elderly people, people with pre-existing cardiac diseases and people who perform heavy physical labour are among the most vulnerable. It should be emphasized that almost no studies have been carried out on the impact that daily climate has on cardiac health at locations characterized as very hot, during the hottest parts of the day and in the hot season (i.e. in the Tropics). The U-shaped relationship between temperature and cardiac disease occurrence will be more dominated by increased events at the hot end in such locations. These hot parts of the world are also where the majority of the global population lives and works.

With more intense heat waves of longer duration, mortality due to myocardial infarction as well as excesses in the mortality and hospitalization of HF patients are to be expected. Preventive measures may limit the expected effects of climate change on cardiac health, namely increased patient awareness, social networking, increased access to air-conditioned environments, physician and hospital preparedness and heat-wave alert response systems. Admittedly, such measures are rather limited in their beneficial potential impact.

Concerted actions to reduce emissions of greenhouse gases, such as changes in transportation patterns, a lower consumption of meat from cows and sheep and architectural solutions for the over-heating of indoor spaces, will have certain cardiac co-benefits and serve as an opportunity to modify lifestyle habits on a large population scale. Increased access to appropriate health care, particularly in low-income populations, is another important step towards coping better with the cardiac challenges of climate change.

Conflict of Interest

There were no conflicts of interest.

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