Heat Exposure and Cardiovascular Health:

A Summary for Health Departments



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Heat Exposure and Cardiovascular Health: A Summary for Health Departments

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Executive Summary

Extreme heat events (EHEs) are a leading cause of weather-related injury and death in the United States, and under a changing climate, these meteorological episodes are predicted to increase in both frequency and intensity. Prolonged heat exposure from EHEs places an increased strain on the heart and may lead to heat-related illness if the cardiovascular system fails to properly thermoregulate internal body temperature. Every individual is susceptible to heat-related illness, however, those with reduced cardiovascular function and pre-existing cardiovascular diseases are at a greater risk for morbidity and mortality during EHEs. This document gives an overview of our current understanding of heat exposure and its impact on cardiovascular health outcomes, an overview of the medications that may exacerbate heat-related cardiovascular illness, and asummary of the interaction between extreme heat and air pollutants, and their collective impact on cardiovascular health. Additionally, this document summarizes epidemiologic evidence and identifies gaps in the extant peer-reviewed literature on the effectiveness of strategies and interventions to protect against heat-related cardiovascular disease and death. This information is intended to aid health departments and other health professionals in understanding and responding to the impacts of heat exposure on cardiovascular health.

Introduction and Background

The global annual average temperatures have been on the rise over the last 150 years.¹ This upward trend in temperatures has been magnified in the previous six decades and is likely a consequence of anthropogenic activities rather than ecological variation.^{1,2} In the United States, annual average temperatures have been increasing since the beginning of the 20th century and are projected to rise an additional 2.3°F by 2050.² If the radical shift in climate continues, there may be far reaching negative impacts on human health and quality of life.²

Climate change has increased the frequency and the intensity of extreme weather events, especially heatwaves, which are currently the leading cause of weather-related deaths in the United States.³ Extreme heat events are defined as periods of summertime weather that are substantially hotter and/or more humid than the typical for a given location at that time of the year.⁴ EHEs have historically triggered major public health crises. In the summer of 1995, the city of Chicago endured a heatwave that resulted in at least 469 heat-related deaths and 739 excess deaths during the most intense period (July 14th–July 20th).⁵ Additionally, in the summer of 2003, a heatwave that swept through Western Europe claimed an approximate 25,000 to 70,000 lives, with some estimates that place the death toll even higher.^{6,7}

Days that are hotter than the average seasonal temperature, or those with moderately high ambient temperature in combination with high humidity may cause increased levels of illness and death⁸ by compromising the ability of the human body to regulate its internal temperature, which is primarily mediated by the autonomic and cardiovascular systems.⁹ In a hot environment, the autonomic nervous system causes cutaneous vasodilation (widening of the blood vessels in the skin) to allow for greater heat transfer from the body to its surroundings¹⁰ A portion of blood from abdominal organs, and in severe cases all organ systems, is redirected to the skin to accommodate the dissipation of internal heat.¹⁰ Maintaining a steady blood pressure during such significant vasodilation requires an increased cardiac output which is achieved through an elevation in heart rate and myocardial contractility.¹⁰ Usually the cardiovascular systems of the young and healthy can adapt to such demands with respect to heat. However, in the elderly and those with pre-existing cardiovascular conditions (e.g., ischemic heart disease (IHD), coronary heart disease (CHD), heart failure (HF)), the heart is not as proficient at meeting the increased demand required to rid the body of the excess heat. Individuals that belong to these populations are more susceptible to adverse health outcomes from extreme heat exposure.¹¹

Studies have shown that although the general population may be less vulnerable to extremely hot temperatures due to better infrastructure and improved availability of air conditioning, there is still a risk of increased heat-related morbidity and mortality in the future.¹²⁻¹⁴ One study found that if the climatic conditions from the European heat wave of 2003 were to be transposed onto the city of Chicago today, the heat-related deaths would be on the order of ten magnitudes higher than the current annual average heat-related deaths.¹⁵ In addition, a study of 12 major US cities projected that 200,000 heat-related deaths will occur by end of this century due to increasing temperatures, even when accounting for increased human resiliency to extreme heat.¹⁶



There are several interventions that public health departments, government and non-profit organizations can adopt to protect their citizens from extreme heat exposure¹⁷ but there is limited research and evidence on the interventions specifically to protect cardiovascular health, especially in the face of climate change. Given the increasing incidence and prevalence of heart disease in the United States,¹⁸ information on successful strategies for preventing adverse cardiovascular impacts associated with extreme heat could help shape policies and bring about meaningful public health actions to mitigate heat-related health burden in the future.

This document will outline the consequences of extreme heat on cardiovascular health and assess evidence and gaps on the approaches health departments can take to prevent harmful cardiovascular impacts of EHEs. While the intended audience is public health professionals, it may also be useful for other stakeholders such as healthcare providers.

Temperature and Cardiovascular Morbidity

This section summarizes the impacts of temperature on cardiovascular morbidity (degree of cardiovascular illness) with respect to heat exposure, based on a search of peer-reviewed literature through 2019 (see appendix for literature search methodology).

The negative influence of extreme heat on cardiovascular health has been observed across various geographical locations. In the state of New York, utilizing the definition of "extremely hot day" as a daily mean temperature of >95th percentile of regional monthly mean temperature, researchers discovered a significant increase in cardiovascular disease (CVD) related emergency department (ED) visits on day 5 and 6 (odds ratio (OR) = 1.02, 95% confidence interval (CI): 1.01-1.04, and OR = 1.01, 95% CI: 1.00-1.03 respectively) in older adults (≥ 65 years old) following an extremely hot day during the months of April – October, 2005-2013.¹⁹ A similar lag effect of extreme heat was observed by Li et al. on the hospitalizations due to CVDs in New York City throughout June, July and August of 1991–2004. This study reported an 1.4%–3.6% increase in 3-day delayed CVD hospital admissions with every 1°C above the temperature-health effect curve (29°C-36°C).²⁰ Aggregated county-level ED visits from six regions of California from July 15th, 2006 to August 1st, 2006 showed a significant increase (relative risk (RR) = 1.05, 95% CI: 1.02–1.09) in CVD visits due to the 2006 California heat wave compared to a reference period (July 8th–14th, 2006 and August 12th–22nd, 2006).²¹ The above three studies considered CVD ED visits and hospital admissions that included the following International Classification of Disease 9th version (ICD-9) principal diagnoses: hypertension (401–405), ischemic heart diseases (410–414), cardiac dysrhythmias (427), heart failure (428), cerebrovascular diseases (430-434, 436-438) and chronic rheumatic heart diseases (393-396).

Additionally, conditions such as hypertrophic cardiomyopathy (HC), an illness in which the muscle of the heart (myocardium) becomes abnormally thick (hypertrophied), are also affected by heat exposure. A telephone survey of HC patients (n=173) evaluated at Mayo Clinic (Rochester, MN) found that 72 respondents observed a deterioration of their baseline HC symptoms (shortness of breath, chest pain, loss of consciousness) due to a change in ambient temperature. 21% (n=17) of those patients reported an exacerbation with an increase or decrease in ambient temperature. More notably, the other 79% (n=57) attributed their symptom exacerbations to heat alone.²²

In Ontario, Canada, an analysis of all those who were hospitalized for CHD from 1996 to 2013, found a 6% (95% CI: 1%–11%) increase in CHD related admissions on days with high temperatures (daily mean temperature >99th percentile) relative to the optimal temperature (noted in the study as daily mean temperature that is at the 79th percentile). Overall, out of the 1.4 million CHD admissions included in the study, 1.20% (16,000) were attributable to heat.²³ Consistent data was also revealed in an investigation into the relationship between daily mean temperature and rheumatic heart disease (RHD) hospital admissions in Shanghai, China from 2013–2015. Following a day with moderate (>28°C, 90th percentile regional mean temperature) or extreme regional temperatures (>33.5 °C, 99th percentile regional mean temperature) the cumulative relative risks for RHD hospital admissions were 2.55 (95% CI: 1.14–5.73) and 3.22 (95% CI: 1.36–7.61) over lag 0–5 days

correspondingly, when compared to the reference temperature of 0°C.²⁴ Another study conducted in China, estimated that the cumulative relative risk of having an out-of-hospital cardiac arrest (OHCA) following a day with extreme heat (>99th percentile daily mean temperature) in Guangzhou from 2008-2015 was 2.45 (95% CI: 1.15–5.33) over lag days 0–21, compared with the reference temperature (28°C).²⁵ Analogous impacts of extreme heat on cardiovascular morbidity have also been observed in Australia and Korea.^{26,27}

Some research indicates that temperature variability (large changes in mean temperature for a given region for a particular period of time) can also have an impact on cardiovascular morbidity. A nation-wide study in China that included 184 cities found that cardiovascular hospital admission rates increased 0.44% (95% CI: 0.32%–0.55%) for CVDs, 0.31% (95% CI: 0.20%–0.43%) for IHD, 0.48% (95% CI: 0.01%–0.96%) for HF and 0.34% (95% CI: 0.01%–0.67%) for heart rhythm disturbances for every 1°C increase in temperature variability at 0–1 days.²⁸ Additional investigations into the impact of temperature variability on cardiovascular morbidity are highly warranted as scientific evidence on this relationship remains inadequate.

Its critical to note that some peer-reviewed work found no statistically significant links between heat and cardiovascular morbidity. For example, after merging the daily CVD admissions (acute myocardial infarction, angina pectoris, congestive heart failure, hypertension, and stroke) data from four different hospitals in Thai Nguyen province in Vietnam with the daily weather, Giang et. al. observed no statistically significant association between hot temperatures and CVD-related hospital admission over the course of lag days 0–30.²⁹ A systematic review and meta-analysis of twenty-one studies also failed to detect a significant association between cardiovascular morbidity and ambient temperature (-0.5% (95% CI: -3.0%–10.1%)).³⁰ Contradictory evidence was also found in epidemiological studies conducted across Europe.^{31,32}



Temperature and Cardiovascular Mortality

This section summarizes the impacts of temperature on cardiovascular mortality (deaths from cardiovascular illness) resulting from heat exposure, based on a search of peer-reviewed literature through 2019 (see appendix for literature search methodology).

There is strong evidence that exposure to extreme heat directly leads to greater cardiovascular mortality. More specifically, a 26-study systematic review and meta-analysis found a 1.3% (RR = 1.013; 95% CI: 1.011–1.015) increase in cardiovascular mortality with exposure to heat across the included studies.³³ Consistent findings for MI mortality were reported by Sun et al.³⁴ In Bavaria, Germany, a 10% increase (95% CI: 5–15%) in cardiovascular mortality was observed with a 2-day rise in daily mean temperature from 20.0°C (90th percentile) to 24.8°C (99th percentile) from 1990–2006.³⁵ An examination of 16,559 IHD deaths in China (cities included Beijing, Tianjin, Shanghai, and Guangzhou) from 2004-2008 uncovered an 18% greater IHD mortality when the temperature was at 99th percentile compared to the 90th percentile (e.g., higher IHD deaths observed at abnormally hot temperatures).³⁶ Similar results were published on temperature and CHD mortality in Beijing.³⁷ Huang et al. discovered that each day with a mean temperature above 32°C in Brisbane, Australia resulted in 45 (95% CI: 22–67) years of life lost, a measure of premature mortality.³⁸

It's important to bear in mind that many localities and regions have heat actions plans or implement protective strategies such as cooling centers during periods of extreme heat. Likewise, individuals may take steps to reduce their exposure to heat when local governments or weather bureaus declare a heat warning. The studies described above did not assess the existence or impact of such adaptations. This potential source of bias may lead to an underestimate of the effect of exposure to high temperatures on cardiovascular morbidity and mortality.

Air Pollution and Cardiovascular Disease

In addition to the direct impacts of heat exposure on cardiovascular health, there is evidence of combined effects of air pollution and extreme heat on cardiovascular mortality, especially in urban areas.^{39,40} According to the Fourth National Climate Assessment, 100 million Americans are currently residing in regions which have air pollution levels that are harmful to human beings.⁴¹ Human-driven climate change has only augmented the levels of air pollutants in communities across the United States. One way in which this occurs is through air stagnation, a phenomenon where air remains stationary over a particular locality entrapping any pollutants underneath. Emissions from automobiles, power plants and refineries⁴¹ can then react with one another during air stagnation to produce ground level ozone in the lower atmosphere of that region. Other climate-related disasters such as drought and wildfires can contribute to air pollution as well. A full assessment of health effects of exposure to ground level ozone is outside the scope of this document. A few articles that studied the combined effects of air pollution and heat on cardiovascular health are described below.

In eight Chinese cities, a 10 μ g/m³ increment in PM10 (particulate matter less than or equal to 10 μ m in aerodynamic diameter) caused an 0.56% (95% CI: 0.36%–0.76%) increase in cardiovascular mortality on days with normal temperatures (5th–95th percentile regional temperature). On days with higher temperature (>95th percentile regional temperature) the cardiovascular mortality soared to 1.57% (95% CI:, 0.69–2.46).⁴³ Another study, this one conducted in 95 large US cities, observed a 10 part per billion (ppb) rise in ozone increased the cardiovascular mortality by 0.41% (95% posterior interval (PI): –0.19%–0.93%), 0.27% (95% PI: –0.44%–0.87%), and 1.68% (95% PI: 0.07%–3.26%) in low, moderate, and high levels of temperature.

Additionally, there is published data on the sole effect of air pollution on cardiovascular health. A notable study that looked at the relationship between PM 2.5 (particulate matter that have an aerodynamic diameter of 2.5 µm or less) and CVD hospitalizations in New York City, Long Island, and Hudson found that morbidity increased by 1.37% (95% CI: 0.90% - 1.84%) for every 10 µg/m³ rise in PM 2.5. Additional information on the effect of air pollution on cardiovascular health (not specific to heat and thus not included in this document) is available in the following studies:

Authors	Title
Dominic, et al (2013) ⁴⁶	Fine Particulate Air Pollution and Hospital Admission for Cardiovascular and Respiratory Diseases
Mustafic, et al (2012) ⁴⁷	Main Air Pollutants and Myocardial Infarction: A Systematic Review and Meta-Analysis
Rich, et al (2019) ⁴⁸	Triggering of Cardiovascular Hospital Admissions by Source Specific Fine Particle Concentrations in Urban Centers of New York State
Ueda, et al (2009) ⁴⁹	Effects of Fine Particulate Matter on Daily Mortality for Specific Heart Diseases in Japan

Vulnerable Populations

This section highlights populations, not mutually exclusive, that are at an increased risk of heat-related cardiovascular illness and death.

Pre-existing cardiovascular illness

Exposure to extreme temperature causes illness and death by compromising the ability of the human body to regulate its internal temperature which is mediated through the autonomic and the circulatory systems.⁹ Consequently, individuals with already weakened cardiovascular systems have a heightened risk of heat-related morbidity and mortality, as their hearts may not be able to meet the increased demand required to rid the body of the excess heat.⁹ For example, studies have demonstrated that patients with cardiovascular conditions such as heart failure have a decreased cardiac reserve and have diminished levels of blood flow to the skin to allow for heat dissipation during periods of extremely high temperatures.^{50,51}

Elderly

The capability of the cardiovascular system of the human body naturally decreases with age.⁵² Older adults, especially over the age of 60, with pre-existing cardiovascular illnesses in non-cooled environments are particularly vulnerable to increasing temperatures of climate change.^{53,54} One study found that elderly men (average age of 66 among 5 participants) were more sensitive to extreme heat due to a diminished vasodilatory reflex when compared to younger men (average age of 27 among 10 participants).⁵⁵ This data is consistent with the findings of Minson et al. and Kenney et al. on the cardiovascular responses of aged men to heat exposure.^{56,57}

Children

There is very limited peer-reviewed information on heat-induced exacerbation of cardiovascular illness in children and infants. However, the higher-body surface-area-to-mass-ratio and lower sweating capacity of children makes them more susceptible to heat illnesses during extreme heat events as compared to adults.^{58,59} Caregivers of children and infants should exercise cautionary judgment and react accordingly to extreme heat events such as increasing fluid intake, dressing for the hot weather, seeking cooler environments and reducing outdoor activity.⁶⁰

Studies have also shown that the fetuses of pregnant mothers are vulnerable to increasing temperatures, and that exposure to extreme heat during pregnancy can cause congenital heart defects especially if experienced during particular weeks of gestation.⁶¹⁻⁶⁵

Gender

There is inconsistent data on whether women or men may be more sensitive to heat-related cardiovascular illness.⁶⁶ One analysis in Czech Republic found that women had a much greater heat-related IHD mortality compared to men during hot spells.⁶⁷ However, an assessment of ED visit rates for heat stroke in the United States found that the incidence in males was 1.99 per 100,000 (95% CI: 1.81–2.16) while incidence in females was 0.71 per 100,000 (95% CI: 1.43–1.79).⁶⁸ Additionally, a systematic review and meta-analysis derived that

the overall rate of heat-related illness is significantly increased in men compared to women.⁶⁹ The variations in health outcomes by sex can be influenced by several factors such as sex-associated behavioral and exposure differences, occupational, or regional factors.⁶⁶ More research is warranted on the difference in the vulnerability of men and women to heat-induced cardiovascular illness to inform future adaptation strategies and guidelines.

Other populations

There are other populations also considered to be more susceptible to heat-related illness and death than the general public, but with limited peer-reviewed evidence of specific threats to cardiovascular health. Individuals living in some highly urbanized areas that experience the heat island phenomenon, some minorities, people in certain outdoor jobs, and those with less education and of lower socio-economic status are more affected by heat-induced cardiovascular illness.^{70,72} Athletes spending extended periods exercising and performing in the heat are also more likely to suffer heat-related cardiovascular impacts which may even result in death.⁷³ Näyhä et al. identified agricultural workers, unemployed persons, pensioners and individuals having only basic education as those who are more prone to heat-related cardiorespiratory symptoms.⁷⁴ Furthermore, individuals who are socially isolated are more vulnerable to heat-related illnesses than those who enjoy the company of family and friends.^{75,76}

Additional information on vulnerable populations are outlined in two previous CDC technical reports:

Authors	Title
Widerynski et al. ⁷⁷	The Use of Cooling Centers to Prevent Heat-Related Illness: Summary of Evidence and Strategies for Implementation
	https://www.cdc.gov/climateandhealth/docs/UseOfCoolingCenters.pdf
Abbinett et al. ⁷⁸	Heat Response Plans: Summary of Evidence and Strategies for Collaboration and Implementation
	https://www.cdc.gov/climateandhealth/docs/HeatResponsePlans_508.pdf

Table 2: Additional information on vulnerable populations from CDC climate and health technical report series



Cardiovascular Medication Use and Heat Exposure

Within the context of CVD, it is important to take note of how medications prescribed for cardiovascular conditions, namely angiotensin converting enzyme inhibitors (ACEI), ACE receptor blockers (ARBs), beta blockers, and diuretics, can compound the deleterious effects of extreme heat on the human body.

Sommet et al. compared adverse drug reactions (ADRs) during two years with heatwaves (2003 and 2006) to two reference years without heatwaves (2004 and 2005) and found that most frequent ADRs during the years with heatwaves were from cardiovascular medications such as diuretics, ACEIs and ARBs, although the authors do note that the total number of ADRs in heatwave years and the reference years were not significantly different.⁷⁹ A multi-center multi-variate analysis of 1,456 patients that were admitted to hospitals in Paris during the 2003 heatwave (Aug 5th–Aug 14th) demonstrated that longtime use of diuretics (OR=1.26, 95% CI: 1.04–1.54) as one of the 11 prominent variables that could negatively affect prognosis of patients that suffered from non-exertional heatstroke.⁸⁰ Severity at the time of presentation was also reported to be related to diuretic usage.⁸¹ Research on cardiovascular drugs exacerbating heat-induced cardiovascular morbidity and mortality was limited and more studies are required to better understand this relationship and to inform how healthcare practitioners should advise their patients regarding such medications during EHEs.

Interventions and Preventative Strategies for Extreme Heat Events

Based on the peer-reviewed literature, there is evidence that exposure to high temperatures, along with regional temperature variability and air pollution, has significant deleterious effects on cardiovascular morbidity and mortality, especially for vulnerable populations as described earlier. Very limited data was available on the interventions specifically aimed at reducing the cardiovascular morbidity and mortality from heat. The information summarized in this section includes an assessment of the generalized preventative measures that are commonly recommended during heat waves. Strategies that have been supported by peer-reviewed articles to decrease overall heat-related illness can be applied by public health departments to prevent or reduce heat-related cardiovascular illness and death.

Hydration

Being well hydrated remains one of the most important ways in which the dangers of extreme heat can be avoided.^{82,83} When humans first perceive the sensation of thirst, the body is already in a mildly dehydrated status which, in turn, means the cardiovascular system is already being strained.⁸⁴ Therefore, in general, healthcare providers and public health departments can encourage residents to drink water before the feeling of thirst. Water should be consumed before participating in any mild to moderate outdoor activity. Sole consumption of water during the exertional activity is inadequate for replenishing the losses from the activity itself.^{82,83} The CDC recommends that individuals should drink 8 ounces of water for every 15–20 minutes spent working in the heat as consuming large quantities of water at once is ineffective.⁸²

However, individuals must take note to avoid excess consumption of water as over-hydration can quickly lead to electrolyte imbalances (hyponatremia) which can carry its own set of adverse effects and may even lead to coma and death.⁸⁵ While hydration may be protective against the cardiovascular impacts of heat exposure, we were unable to find peer-reviewed literature on the impact of health department activities (e.g. a communication campaign reminding people to stay hydrated) on cardiovascular outcomes.

Air conditioning

Along with adequate hydration, readily available air conditioning has a strong protective effect against the negative impact of severe heat.⁸⁶⁻⁸⁸ Although there are no guidelines on the length and temperature of air conditioning, individuals can stay in cooled areas until normal core temperature (98.6°F) is achieved.⁶⁰ To further improve the access to air conditioning, public health departments can work to implement cooling centers by following guides published by governmental organizations.⁷⁷ If access to air-conditioned areas is lacking, individuals can seek out other publicly air-conditioned places, shaded areas or try to take cool showers and baths to help stave off heat stress during extreme heat events.^{89,90}

Clothing

During extreme heat events, wearing one layer of light colored (to minimize heat absorption) and loose-fitting clothing (to allow for air permeability) seemed to provide most protection against heat-illnesses.^{91,92} Ideally, the clothing material should be cotton.⁶⁰ However, there is mixed evidence for wearing clothing made

of polyester. ⁹³⁻⁹⁵ Although, synthetic "breathable" fibers are widely available and may keep individuals drier and cooler, little peer-reviewed research exists to support such claims. Wearing wide brimmed hats (at least 7.5 cm or greater) is another potential source of protection against heat-related illness, but individuals should not choose hats that would retard evaporative heat loss.⁹⁶

Electric fans

Convection (one of the modes of heat transfer) and evaporation of sweat are both mechanisms through which the human body reduces its core temperature. Use of an electric fan can facilitate these processes. Fans are low-cost, accessible, and require less energy to operate than air conditioning, and are thus an attractive option as an intervention to reduce heat-related illness.⁹⁷ Current guidelines in medicine recommend treating exertional heat stroke with ice water immersion while treating non-exertional heat stroke with evaporative cooling (spraying the patient with water in combination with a fan).⁹⁸ However, there is mixed evidence about the efficacy of fans for prevention of heat stroke during extreme heat events. Factors such as temperature, humidity, hydration levels, and current health status could all impact the effectiveness of fan use in reducing body temperature and protecting health.⁷⁸

There are relatively few peer-reviewed research articles on fan effectiveness. A Cochrane review published in 2012 concluded that existing evidence did not resolve uncertainties about the health effects of electric fans during heat waves and suggested that randomized trials would help to fill the knowledge gap.⁹⁹ Other studies have continued to find mixed and sometimes contradictory evidence for the use of fans to prevent heat-related illness.^{86,87} For additional information on this topic, see the "fan distribution and use" section of Heat Response Plans: Summary of Evidence and Strategies for Collaboration and Implementation.⁷⁸ We were unable to find peer-reviewed literature on the health impacts of fan distribution programs.

Alcohol intake

Typically, water is regarded as the safest way to restore fluid volume when mildly dehydrated and as heat-stroke prophylaxis. Usage of alcohol has been known to have a diuretic effect on the human body¹⁰⁰, although there is limited peer-reviewed data on their impact on heat-induced cardiovascular morbidity and mortality. Some studies have demonstrated that excessive alcohol consumption is a considerable risk factor for heat stroke and heat-related mortality during periods of extremely hot weather.⁸⁷ Alcohol alters mental status and impairs judgement, and may render excessive consumers less capable to react to extreme heat and prevent them from seeking out cooler environments.⁶⁰ However, we did not identify studies examining the effects of moderate to low alcohol consumption on heat-related cardiovascular illness during EHEs.

Caffeine intake

Expert suggestions on the intake of caffeine during extreme heat is unclear. If adequate hydration is maintained, acute caffeine consumption in chronically consuming subjects has been shown to have no effect on thermoregulation.¹⁰¹ Other studies found similar results in non-habitual, non-heat acclimatized users with

an acute increase consumption of caffeine,¹⁰² even though non-regular caffeine users seemed to experience its diuretic effect to a greater extent than habitual consumers.¹⁰³ A 9mg/kg dose of caffeine was enough to slightly increase heat production and mean body temperature, but it was not sufficient for the participants to perceive and thus likely would not cause significant physiological strain.¹⁰² Healthcare providers and public health departments may choose to communicate to the general public and non-habitual consumers to decrease their intake of caffeine during excessively hot weather. However, current research is unclear on recommendations for habitual consumers on caffeine intake during EHEs and as such should not be advised.⁶⁰

Folic acid and L-Arginine Supplementation

Dietary supplements have been proposed as non-pharmaceutical prophylactic strategies against heat-related cardiovascular illness; however, the current body of evidence is limited, and it is unknown if they have a direct preventative effect. Gagnon et al. concluded that there was no significant difference before and after a 6-week supplementation (5 mg/day) of folic acid in cutaneous vascular conductance (before (CON): $54 \pm 8\%$ units/mmHg vs. after (FOLIC): $59 \pm 7\%$ units/mmHg, p = 0.22) and forearm vascular conductance (CON: 3.47 ± 0.76 mL/mmHg vs. FOLIC: 3.40 ± 0.56 mL/mmHg, p = 0.93) in nine healthy older adults subjected to extreme heat and humidity.¹⁰⁴ Similarly, a 10g supplementation of L-arginine before passive and active heat exposure in young, health males improves neither cardiovascular nor thermoregulatory responses (measures with p > 0.05 included systolic arterial pressure, diastolic arterial pressure, mean arterial pressure, cutaneous vascular conductance, rectal and skin temperature).¹⁰⁵



Summary

Healthcare providers and public health departments may recommend several interventions during extremely high temperature events. Advice to the general public can include maintaining adequate hydration, seeking out air-conditioned buildings, wearing light-colored clothing and wide brimmed hats, and recommending the restriction of outdoor activities during EHEs.¹⁷ In the workplace, a way to address heat stress is to reduce the physical workload per individual and encourage more frequent and longer breaks to allow the workers to dissipate their excess internal heat.^{106,107}

Intervention	Evidence
Hydration (drinking water)	Supported by literature
Air conditioning	Supported by literature
Light colored, loose fitting cotton clothing	Supported by literature
Electric fans	Mixed evidence
Reducing alcohol intake	Mixed evidence
Reducing caffeine intake	Mixed evidence
Dietary supplements (folic acid; L-arginine)	Not supported by literature

Table 3: Summary of strength of evidence of interventions to prevent negative cardiovascular health impacts

Dissemination

Information on these interventions can be quickly disseminated to the general population via integrated community hot weather warnings and heat action plans. Comprehensive heat action plans have been shown to reduce heat-related mortality, and therefore can likely be applied to similar affect in the context of cardiovascular health.^{108,109} Implementing heat response plans can also have great economic benefits that outweigh the costs of running and maintaining such strategies.¹¹⁰ To further increase cost-effectiveness of heat response systems, public health departments can also consider utilizing media platforms to disseminate information on extremely hot days. In cities and regions where there are no current heat-response plans, or in places with hotter climates, public health departments can employ syndromic surveillance systems and monitor web data from search engines on common symptoms of dehydration and heat-stroke to prepare for extreme heatwaves.¹¹¹ More information on the barriers, implementation and coordination of heat response plans can be found in the selected resources section, and in Abbinett et al., 2019.⁷⁸

Future planning

In the future, city planners, architects, and developers may choose to implement more reflective "cool" roofs into their designs to better mitigate the urban heat island effect. A study conducted in the UK found that reflective roofs can reduce up to 7% of the total heat-related mortality that is due to the urban heat island effect if enacted across a whole city.¹¹² More information on factors that should be considered by city planners and public health professionals when designing future cities and urban areas can be found in the selected resources section.

Outdoor and indoor air pollution compounds the deleterious effects of heat on cardiovascular health. Therefore, public health departments can also attempt to employ strategies to reduce air pollution both inside and outside the household to indirectly decrease the burden of heat exposure on cardiovascular health. For instance, during times of poor air quality, avoidance of outdoor activities for those with cardiovascular diseases or reducing dependence on solid fuels for cooking could prevent negative health outcomes.^{113,114} More information on this topic can be found in the selected resources section of this document.

Research Gaps

Although there is research on prevention strategies against general heat-related illness and death, studies on interventions that specifically target cardiovascular morbidity and mortality are lacking. Management of cardiovascular medications during EHEs also requires additional investigation.¹¹⁵ Furthermore, guidance and information on cross-sectoral collaboration, e.g., partnerships between public health departments and hospitals to prevent cardiovascular health impacts may be of great benefit. Finally, additional research could help quantify the potential impacts of a changing climate on cardiovascular health in the future.

Selected Resources

This section lists additional resources that public health departments might find helpful when developing and implementing strategies against heat-related cardiovascular deaths. This is not a comprehensive list and includes resources that are not peer-reviewed

Table 4: Selected resources on the development and implementation on protective strategies against
heat-related illness.

Category	Resource
Safety advice during heatwaves	Harvard Medical School ¹¹⁶ Heat is hard on the heart; simple precautions can ease the strain. <u>https://www.health.harvard.edu/blog/heat-is-hard-on-the-heart-simple-precautions-can-ease-the-strain-201107223180.</u>
Safety advice during heatwaves	Maricopa County, Arizona Department of Public Health ¹¹⁷ Extreme Heat. <u>https://www.maricopa.gov/1871/Extreme-Heat</u> .
Safety advice during heatwaves	Minnesota Department of Public Health ¹¹⁸ Extreme Heat Events. <u>https://www.health.state.mn.us/communities/environment/climate/ extremeheat.html</u> .
Development of heat action plans	World Health Organization ¹¹⁹ Heat–health action plans. <u>http://www.euro.who.int/en/publications/abstracts/heathealth-action- plans</u> . Published
General advice on interventions against heat-related illness	EPA ¹²⁰ Extreme Heat Guidebook. https://archive.epa.gov/epa/climatechange/extreme-heat-guidebook. html.



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Appendix

Methodology

An initial informal review on the relationship between cardiovascular health, temperature, and climate change was conducted using Google Scholar through August 1, 2019. This was followed by an informal search and review on interventions to prevent heat-induced cardiovascular morbidity and mortality. These informal reviews were used to guide a formal review utilizing the CDC library. Databases in the formal search included Medline, Embase, Environmental Science Abstracts and GreenFILE. The search was limited to English results.

Description of CDC library search

Climate and heat terms: Climate Change; Heat Exposure; Extreme Temperatures; Ambient Temperature; heat; heatwave; heat wave; hot; extremely hot weather; heat-related; Heat emergency

Health terms: Cardiovascular Health; Coronary Artery Disease; Myocardial Infarction; Congestive Heart Failure; Cardiovascular Mortality; Cardiovascular Morbidity; Cardiovascular Drugs; Diuretics; Non-communicable disease; heart disease; Cardiorespiratory; hospitalization; ACE inhibitors; Betablockers; Angiotensin receptor blockers; Cardiovascular dysregulation

Intervention/adaptation/action terms: Intervention; Prevent; Mitigat*; Control; Hydrat*; Heat Response Plans; Heat Warning Systems; adaptation; assessment; evaluat*; cooling; communication plan; strategy; education; awareness; insulation; protection.

The search yielded 592 unique articles. After reviewing title and abstracts, 187 were selected due to their relevance to the topic at hand according. Full text was reviewed for these articles. In addition, the references of these articles were reviewed to identify any additional relevant articles. These articles were used to inform this document.



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