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Review

Effect of Temperature on Emergency Ambulance Call-Outs for Cardiovascular Causes: A Scoping Review

Santiago Gestal Romaní, Adolfo Figueiras, and Dominic Royé*

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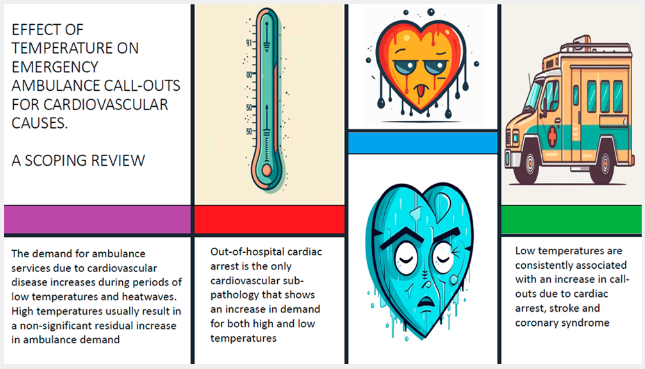
ABSTRACT: Climate change has increased interest in the effects of the thermal environment on cardiovascular health. Most studies have focused on mortality data. However, pre-hospital care data are better able to evaluate these effects, as they can register the full spectrum of the disease in real time. This scoping review aims to synthesize the epidemiological evidence regarding the effects of the thermal environment on cardiovascular morbidity in the pre-hospital setting, evaluated through ambulance calls. A staged literature search was performed using the PubMed database for the period between 1st January 2000 and 30th March 2023, using the MeSH terms "Weather" AND "Emergency Medical Services". A total of 987 publications were identified that examined the correlation between the thermal environment and ambulance call-outs for cardiovascular causes. The studies were mostly ecological time series, with significant variability in the methodological aspects employed. An increase in the number of ambulance call-outs has been observed in association with low temperatures, both for overall cardiovascular pathologies and for certain pathological subtypes. For high temperatures, no effect has been observed in overall call-outs, although an increase has been observed during heat waves. The demand for ambulances for cardiac arrests is increased by both low and high temperatures and during heat waves. Ambulance call-outs for cardiovascular causes increase with low temperatures and heat waves, with no significant increase in the overall demand associated with high temperatures. Ambulance call-outs for cardiac arrests are the only subtype that is increased by high temperatures.



KEYWORDS: *cardiovascular diseases, weather, cold exposure, heat exposure, ambulance call-out, emergency medical services*

INTRODUCTION

Cardiovascular disease is the leading cause of morbidity-mortality worldwide.[1] Available evidence indicates that both low and high temperatures are associated with increases in risk of presenting cardiac decompensation.[2−6]

In the current context of climate change, interest in assessing the impact of extreme temperatures on cardiovascular health has grown.[7] Most studies analyzing the effect of temperature on cardiovascular health make use of mortality, hospitalization or emergency department care data. Assessing the morbidity of cardiovascular ambulance call-outs (ACO) can help to understand the full range of diseases in real time, from minor cases which do not require hospital care to severe cases which do not reach hospital emergency services, as seen in Figure 1.

In recent years, awareness of the effects of climate change on cardiovascular health has increased, along with the appearance of the figure of the climate cardiologist, a specialist in the effects of climate on cardiovascular health who seeks opportunities to implement protective measures for patients and the planet.[8]

The purpose of this scoping review was to gather and summarize all of the available information relating to the impact of temperature on ACO for cardiovascular causes. Acquiring knowledge of the relationship between temperature and demand for ambulances due to cardiovascular causes will aid in allocating the necessary resources to the population based on weather forecasts and promote preventative public health measures to mitigate the anticipated effects of high or low temperatures.

MATERIALS AND METHODS

Search Strategy

A rigorous search was carried out to identify studies assessing the effects of the thermal environment on ACO for cardiovascular causes. To ensure that the search was comprehensive and up-to-date, the decision was taken to use PubMed as the database of

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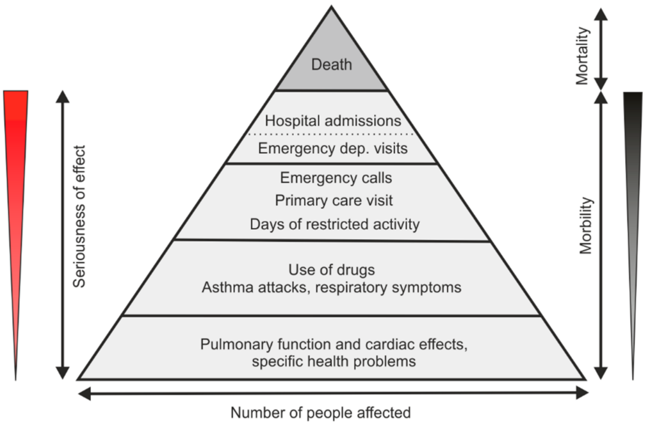

Figure 1. Environmental effects on cardio-respiratory diseases. Reprinted with permission from ref 9. Copyright 2017 Springer.

reference. PubMed is a widely recognized and reputable medical database with advanced search tools, making it the most appropriate database for the present study, which specifically focuses on studies published in medical journals. To conduct the search, advanced search tools and the primary MeSH terms "Weather" and "Emergency Medical Services" were used, along with the Boolean command "AND" to limit the number of studies that assess the two principal characteristics targeted by our review. The only restriction established in the search was the date of publication, which was required to be between 1st January 2000 and 30th March 2023.

Inclusion and Exclusion Criteria

The eligibility criteria of the studies were as follows:

1. Papers were to be written in English.
2. Papers were to use temperature (in all its forms of assessment) as a variable of exposure.
3. Papers were to assess ACO for cardiovascular causes or their disease subtypes.
4. The exposure-response was to be expressed as relative risks (RR), odds ratios (OR), regression coefficients, attributable fraction (AF) or excess of events studied.

RESULTS

Search Results and Methodological Aspects

The search was conducted on 30th March 2023 using the predefined MeSH terms and retrieved a total of 987 papers from the PubMed database. The initial screening of titles was performed by one author, who excluded 879 papers as irrelevant to the study topic. In cases of uncertainty, the abstract or full text was read and the other authors were consulted before deciding upon the inclusion or exclusion of the paper in question. Subsequently, the abstracts and full texts of the remaining 108 papers were reviewed and 81 were excluded for not meeting the inclusion criteria. The most frequent causes of exclusion were a failure to assess a cardiovascular cause and a lack of ACO

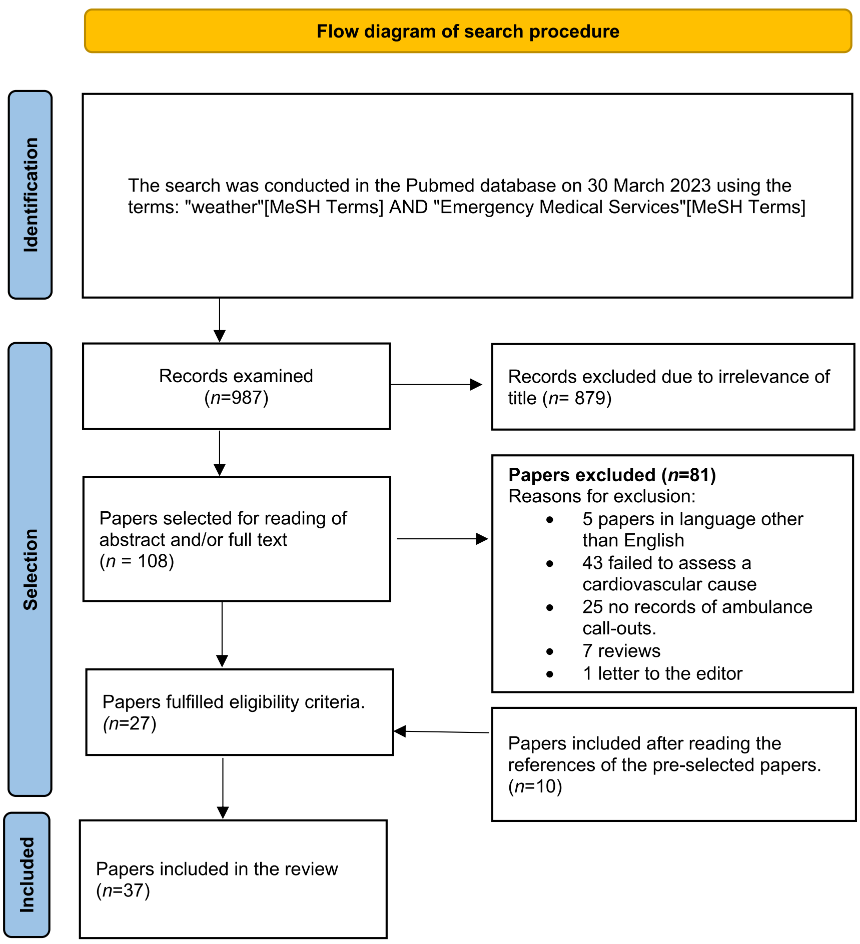

Figure 2. Flowchart.

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records. Last of all, the references of the 27 papers selected were reviewed and 10 papers not detected in the initial search were included. In the end, the review comprised 37 papers (Figure 2).

Of the 37 papers included in our review, 16 were devoted to evaluating the impact of the thermal environment on cardiovascular-related events and are summarized in Table S1. The remaining 20 papers investigated the effect of the thermal environment on specific subtypes of cardiovascular events and are summarized in Table S2. One paper,[10] which investigated both the overall effect and the effect on individual disease subtypes, is included in both tables (Table S1 and Table S2).

Geographical Location of Studies

The studies were conducted in different geographical locations with different climatic and socio-demographic conditions. The majority (17) were undertaken in Asian countries (China (7), Japan (8), Iran (1) and Korea (1)); 13 were carried out in European countries (Spain (1), England (1), France (2), Germany (2), Lithuania (3), Italy (3) and Sweden (1)); 6 were undertaken in Oceania (all in Australia); and 1 was undertaken in the USA (King County). The search located no studies carried out in Africa or Central or South America.

Methodology of Analysis

The *time series study* was the most widely used design (in a total of 31 studies) to assess the effects of temperature on cardiovascular morbidity. The methodology for statistical analysis used in each one of them varied, although most of them used regression models, distributed lag nonlinear models (DLNM), and/or correlation coefficients.

Other designs included case-crossover studies,[11−13] a prospective observational study,[14] a cross-sectional study[15] and an ecological time series study.[16] The method of statistical analysis varied with the study design.

Spatial Setting

The spatial setting of the studies covered differing levels, ranging from ***suburban***[17] to ***urban***[14,15,18−33] and ***regional***[10−12,16,34−40] to ***national***,[41−46] providing information on the effects of temperature in different climatic areas.

Exposure Measures

Most of the studies used ***mean daily temperature*** as the variable of exposure,[11,13,15−18,20,23−28,30−32,34,41,43−46] with the ***minimum daily temperature***[12,17,19,24,25,28] and ***maximum daily temperature*** also being used.[17,19,24,25,29,36−39] Others used ***mean hourly temperatures***,[13,14,21,42] ***biometeorological indices***, combining temperature and humidity, such as the **apparent temperature**,[22,40] **heat index**[10] or **Humidex**[35] and, in one study, **diurnal temperature range (DTR)**[33] (intraday difference between maximum and minimum temperature). To date, no method of thermal environment measurement has been demonstrated to be superior to another in predicting the effect. This has resulted in variability in the exposure measurement used across different studies.[47]

Lagged Effects

It is known that after exposure to a given thermal environment, risk is neither linear nor immediate in time. Some studies have assessed the effects at the **date of exposure**,[11,12,14,15,17,23,28−32,34−39] some **in hourly ranges closely following exposure**[21,42] and others in the **short *(0−48 h)***,[10,33] **medium *(0−7 days)***[13,18,24−26,41,46] or **long-term *(0−14 days)***[19,40] or ***0−21 days or more***[16,20,22,27,43−45]).

Definition of the Effect

There were fundamentally two types of results assessed by the studies: the effect on cardiovascular events overall (Table S1) and the effects on different cardiovascular sub-diseases (Table S2), such as stroke, out-of-hospital cardiac arrest, acute coronary syndrome, atrial fibrillation, hypertension, chest pain or suspicion of cardiovascular disease. One study assessed the effects overall and by sub-disease[10] and is, therefore, included in both tables.

To estimate the effects, most studies used the minimal morbidity temperature (MMT) calculated after obtaining exposure−response models,[13,16,18−21,27,30,33,39,41−46] with the rest using temperature ranges or selecting arbitrary thresholds to estimate the effects in some versus others[12,14,15,17,22−26,28,30−32,35,40] or assessing increases in risk by referring to comparable time periods during which the target exposure did not take place.[10,29,34,36−38]

Confounding Factors and Covariates

The majority of the studies (59%) took confounding factors into consideration, such as environmental air pollution (PM2.5, PM10, O_3), other atmospheric parameters (humidity, barometric pressure, wind speed), day of the week/weekend/public holidays, season, or coincidence with influenza epidemics.[11,13,16−22,24−27,39−46]

Effects of Low Temperatures on Ambulance Call-Outs for Cardiovascular Causes

The effect was assessed in seven studies.[16,18,19,21,22,43,45] Although none assessed it during cold waves, they did assess it at extremely low centiles. The effects due to low temperatures were studied over the course of the year in four studies[16,19,21,43] and during the cold season in three.[18,22,45] Generally, the effects were assessed by reference to the MMT,[18,19,21,43,45] and, in one case,[22] by reference to an arbitrary centile (25th centile). The centiles of comparison also varied, with low (<10th centile,[18] <5th centile,[18,19] <2.5th centile[21,43]) or extreme (<1st centile[18,22,45]) centiles being used. The effect was likewise assessed for each degree of decrease in the minimum morbidity temperature[16] and by calculating the attributable fraction to non-optimal temperatures.[19,43] The increase in risk was observed under different climatic conditions, i.e., subtropical,[18] temperate oceanic,[19] temperate monsoon,[21] humid subtropical,[22] and subtropical oceanic[16] and even in different regions of Japan with different climatic conditions.[43,45] The increase in risk varied according to the lag at which the effect was studied. Overall, studies examining the lag time between exposure to temperature and ACO found that the increase in risk was not immediate. In fact, two studies[16,21] even showed a reduced risk in the first 24 h after exposure (Cui et al., 2020,[21] reported RR = 0.87, 95% CI: 0.78 to 0.98, at lag 0−48 h, and Turner et al., 2012,[16] reported a decrease in attendance of −0.625% (95% CI: −1.17% to −0.06%), at lag 0−1 days. It takes at least 2−3 days post-exposure for the risk to start rising, although the values remain only slightly higher than one. The increase in risk was usually maintained until 7−15 days after exposure (see Table S1). Two studies[19,43] investigated the fraction of emergency call-outs attributed to non-optimal temperatures. Onozuka and Hagihara (2015)[43] reported that 18.02% of emergency call-outs were attributable to non-optimal temperatures, with the majority of these (17.93%) being due to low non-optimal temperatures. Of this fraction, 16.92% were attributed to moderately low temperatures (between the 96th and 2.5th centiles), with only 1.08% being due to extremely low

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temperatures. A similar effect was found by Romani et al. (2020)[19] in two cities in the northwest of Spain, with a significant effect of low temperatures observed in only one city (A Coruña).

Effects of High Temperatures on Ambulance Call-Outs for Cardiovascular Causes

Ten studies investigated the effect of high temperatures.[16,18,19,21,22,28,35,40,43,44] Of these, six assessed the effect during the hot season,[18,22,28,35,40,44] while four papers investigated the effect throughout the year.[16,19,21,43]

Most studies compared high centiles of temperature with a calculated MMT to assess the effects.[16,18,19,21,35,43,44] However, Pourshaikhian et al. (2019)[22] arbitrarily employed the 75th centile as a comparison, while Alessandrini et al. (2011)[40] used percentage increases in the risk of call-outs for each degree of apparent temperature across different temperature ranges and Petralli et al. (2012)[28] compared different climate conditions. The choice of comparison centiles varied across studies, with some using high centiles, such as >90th centile,[18] >95th centile,[18,19] and >97.5th centile[21,44] or even extreme high centiles, such as >99th centile.[18,22,35,43] The effects of high temperatures were found to be less consistent compared to those observed for low temperatures. While some studies, including Wang et al. (2021),[18] Romani et al. (2020),[19] Pourshaikhian et al. (2019),[22] Calkins et al. (2016),[35] Onozuka and Hagihara (2017)[45] and Turner et al. (2012),[16] did not report a significant increase in risk, others, such as Cui et al. (2020),[21] Alessandrini et al. (2011),[40] Petralli et al. (2012)[28] and Onozuka and Hagihara (2015),[43] found an increased risk associated with high temperatures. Cui et al. (2020)[21] found an increased risk at lags of 0−12 h (RR = 1.04, 95% CI: 1.00−1.08) and 0−24 h (RR = 1.05, 95% CI: 1.00−1.11). Alessandrini et al. (2011)[40] reported that call-outs increased by 7.93% (95% CI: 3.76−12.10) for each one-degree rise above an apparent temperature of 30 °C, with a greater effect observed in patients over the age of 75 years (11.82%, 95% CI: 6.18−17.46). Petralli et al. (2012)[28] found a higher incidence of cardiovascular events during hot seasons with high temperatures compared to low temperatures (RR = 3.38, 95% CI: 0.22−6.51). Turner et al. (2012)[16] reported a decrease of −1.85% (95% CI: −3.06 to −0.64) in ACO at lags of 2−15 days post-exposure, a phenomenon known as the "harvesting effect" due to the fact that the events are assumed to have accumulated previously. Two studies examined the proportion of ACO that can be attributed to high, non-optimal temperatures,[19,43] with only Onozuka and Hagihara (2015)[43] reporting a slight increase of 0.10% (95% CI: 0.04−0.14) in risk. This increase is much smaller than the proportion of ACO attributed to low, non-optimal temperatures.

Effects of Heat Waves on Ambulance Call-Outs for Cardiovascular Causes

Six studies investigated the association between heat waves and cardiovascular disease.[10,11,36−39] However, the definition of heat wave varied between studies. For instance, Campbell et al. 2021[11] used the Australian Meteorology Office's new definition, which considers excess heat for a population in the average forecast for 3 days and classifies heat waves into three degrees of intensity.[48] On the other hand, Nitschke et al. 2007, 2011, and 2016[36−38] defined heat waves as $T_{max} \geq 35$ °C (95th centile) for three or more consecutive days. Meanwhile, Turner et al. 2013[39] defined heat wave as two or more consecutive days with T_{max} >37 °C, while Brunetti et al. 2014,[10] defined it as days with a Heat Index (HI) two standard deviations above the mean for the month (taking the mean temperature of the three previous years as reference).

Regarding the effects of heat waves on ACO for cardiovascular causes, Campbell et al. 2021 observed an insignificant increase in call-outs during low-intensity and extreme-intensity heat waves in Tasmania. However, there was no increase during high-intensity heat waves. In the Adelaide region of Australia, an area with a temperate Mediterranean climate, Nitschke et al. (2007)[36] assessed different heat wave periods. While they did not document a rise in call-outs in 31 heat waves registered between 1993 and 2006,[36] they did observe a 7% decrease in call-outs in the population subgroup aged ≥75 years. In 2011, they updated their data[37] until March 2009, observing an increased risk of call-outs in both the 2008 and 2009 heat waves, though not in previous heat waves. They also studied the effects of a 2014 heat wave in Adelaide following the adoption of public health prevention measures, finding a 56% decrease in call-outs compared to the 2009 heat wave.[38] In Brisbane (Australia), a city with a humid subtropical climate, Turner et al. (2013)[39] observed a significant increase in the number of call-outs, particularly in the subgroup of patients aged 65 years and over. During the 2011 heat wave in the Apulia region (Italy), an area with a predominantly Mediterranean climate, Brunetti et al. (2014)[10] observed a 46% increase in call-outs for electrocardiogram assessments due to suspected heart disease after 48 h of exposure.

Effects of Temperature on Different Subtypes of Cardiovascular Disease

Out-of-hospital cardiac arrest is the most widely studied cardiovascular event in ten studies.[12,13,15,20,23,27,29,41,42,46] The majority of these papers (7/10) focus on the impact of high and low temperatures, with only one exclusively assessing the effect of low temperatures[12] and two examining the effect of high temperatures and heat waves.[10,46] **An increased risk of cardiac arrest was consistently associated with low temperatures**. In eight out of nine studies, a rise in the number of call-outs following exposure to low temperatures was observed. This effect was noted on a local level in Hamburg (Germany),[23] Chinese Taipei,[41,42] Guangzhou (China),[27] Stockholm (Sweden)[13] and Osaka (Japan)[15] and on a regional level in North Tochigi (Japan),[12] albeit only in the elderly subgroup (≥75 years). However, a local study in London (England) by Sangkharat et al. in 2020[20] did not find any effects.

Studies that found a significant increase in risk with low temperatures and examined the lags post-exposure[27,41,42] reported the peak effect sometime after the exposure had passed. Therefore, an hourly assessment of risk by Wang et al. in 2021[42] showed that the risk reached a peak at a lag of 0−180 h, while Wang et al. in 2020[41] found an increased RR due to low temperatures (=<15 °C) of 1.61 (95% CI:1.47−1.77) in the 4 days following exposure. In addition, in 2016, Niu et al.[27] noted that the risk continued to rise as the time from exposure increased: at lag 0−14 the RR was 2.85 (95% CI: 1.44−5.63), at lag 0−21 it was 3.75 (95% CI: 1.63−8.63) and at lag 0−28 6.87 (95% CI: 2.43−19.43).

As far as high temperatures are concerned, the results were less consistent, with only six out of nine studies[27,29,41,42,46] reporting a significant increase in risk. The remaining three studies[13,15,20] did not find significant effects. Two of the studies that reported a heat-related effect examined the impact during heat waves. In Korea, a 14% increased risk was observed during heat wave

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versus high-temperature periods,[46] while in Paris (France), the risk was 2.5 times higher during the heat-wave period than during the control period.[29]

Stroke was the second most widely studied event in five studies.[14,17,32−34] ***The effects due to low temperatures were relatively consistent*** in the different studies, with an increase in call-outs in response to low temperatures being documented in 3/5 studies, namely, in the Gifu Prefecture (Japan),[34] on a local level in Hamburg (Germany)[14] and in Yokohama (Japan).[32] On a suburban level in Paris[17] (France), the multivariate analysis showed no relationship. ***The effect of high temperatures*** was assessed in four studies, of which two reported significant findings: Matsumaru et al. (2020)[34] observed a 9% reduction in risk (OR = 0.91 (95% CI: 0.88−0.94)) for each one-degree rise in temperature, while Ohshige et al. (2006) reported a reduction in events with the rise in temperature. Wang et al. (2022)[33] assessed the incidence using DTR, observing a significant increase in risk for every degree of decrease in DTR below 10 °C, but not for increases.

Coronary disease in its different forms of clinical expression (chest pain and acute coronary syndrome) was assessed in eight studies.[10,14,20,26,29,30,41] In Chinese Taipei,[40] ACO due to *chest pain* showed an increased risk of RR = 1.14 (95% CI: 1.05−1.25) with low temperatures (<5th centile) but not with extremely high temperatures (>99th centile); in London,[20] while no relationship was observed with low temperatures, it was observed with high temperatures, with an RR = 1.684 (95% CI: 1.320−2.150), although this study estimated a MMT of −2 °C for chest pain. *Acute coronary syndrome* call-outs were consistently associated with low temperatures across different regions. In Hamburg[14] (Germany), a 26% increase in risk was observed at temperatures below 10 °C. Similarly, in Kaunas (Lithuania),[26] increased risk was found over three consecutive days of low temperatures. In Beijing (China),[30] a maximum 43% increase in call-outs was observed with a daily temperature of 2 °C and in Hiroshima (Japan)[31] an increase in demand was observed at temperatures below 10 °C. No significant increase was reported for high temperatures or heat waves.

Arrhythmias and hypertensive emergencies were the least studied sub-diseases, with arrhythmias showing inconsistent findings. Sangkharat et al. (2020)[20] in London and Hensel et al. (2017)[14] in Hamburg found no relationship between temperature and arrhythmias. In Kaunas, Vencloviene et al. (2017)[25] observed an increase in ACO during the 14:00−21:59 time period after 2−3 days when the temperature fell below 1.9 °C. In Apulia (Italy), Brunetti et al. (2014)[10] reported an increase in the number of diagnoses of atrial fibrillation after 24 h on heat wave days (HI > 44). *Hypertensive emergencies* were studied by Hensel et al. (2017)[14] in Hamburg, with an 18% increase in ACO (95% CI: 10%−25%) being observed at temperatures below 10 °C, and by Vencloviene et al. (2015)[24] in Kaunas, with a fall in ACO being observed for every 10 °C increase in temperature.

■ DISCUSSION

The relationship between ambient temperature and cardiovascular morbidity and mortality has been extensively studied and reviewed in the scientific literature, with several meta-analyses and reviews having been conducted to date.[49−53] However, these reviews have focused solely on evaluating a portion of cardiovascular pathology, specifically, that which results in patient death, hospitalization or emergency department care. A recent review and meta-analysis[54] evaluated the effects of temperature on ACO globally but did not focus specifically on cardiovascular alerts.

It is important to note that ACO may be more sensitive in capturing the effects of ambient temperature on the cardiovascular system in real time. This is due to the fact that they can capture the full spectrum of disease, ranging from minor decompensations, which can be resolved without requiring hospitalization, to events that may or may not require hospital care (such as patient death, patient decision or access to private healthcare services). Therefore, ACO are considered a valuable tool in gaining a better understanding of the relationship between ambient temperature and cardiovascular morbidity and mortality.

To our knowledge, this is the first review that specifically examines the relationship between ambient temperature and ACO for cardiovascular causes and subtypes. The results obtained demonstrate that ambient temperature influences the daily number of ACO for cardiovascular causes. These findings are consistent across different geographical regions with varying climatic zones, on different spatial scales (suburban, local, regional and national) and under different thermal conditions (extreme, non-extreme and during heat waves). There is significant heterogeneity in the methods applied by studies to assess the effect, yet they tend to show similar results: low temperatures increase the number of ACO for cardiovascular causes, whereas high temperatures are not consistently associated with an effect on ACO, although an increase can be observed during heat waves. Regarding subtypes of cardiovascular pathologies, low temperatures are consistently associated with an increase in call-outs due to cardiac arrest, stroke and coronary syndrome, while high temperatures increase the number of alerts for cardiac arrest, albeit to a lesser degree than low temperatures, and appear to decrease the number of alerts for stroke.

The findings of the present review are in line with other reviews that have investigated the impact of low temperatures on cardiovascular morbidity and mortality. For instance, Moghadamnia et al. (2017)[49] observed a 5% increase in cardiovascular mortality due to cold exposure, while Phung et al. (2016)[53] reported a 2.8% rise in hospitalizations related to cardiovascular conditions due to cold weather. Studies that examined visits to emergency departments for cardiovascular diseases also observed an increase in colder temperatures.[55] In the 2022 review by Wang et al.,[33] it was observed that low temperatures carried a higher risk of calls related to cardiovascular causes (RR = 1.209) compared to global calls (RR = 1.121). In contrast, the association of ACO with high temperatures was only observed globally (RR = 1.179) and not in those related to cardiovascular pathology (RR = 1.022).

There is considerable methodological variability among studies assessing the risk of hospitalization or emergency department visits for cardiovascular causes and those evaluating immediate or different lag (DLNM) effects. Since hospital referrals are typically required for ACO related to cardiovascular causes, the obtained lags should be similar or earlier. However, the lack of studies in the same regions makes it difficult to make comparisons or to determine the initial time of the effect or the maximum post-exposure effect. In England and Wales,[56] it was observed that for every degree of reduction in temperature, there was an increased risk of hospitalization due to acute coronary syndrome, with a stronger effect between 2 and 7 days of lag. In ACO for cardiovascular causes, an increase was observed in the number of calls associated with low temperatures from the same

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day after exposure, which lasts for 5 to 7 days. These effects are consistent with those of cardiovascular mortality, although their maximum delayed effects occur at 14 days.[49]

Exposure to low temperatures is known to bring about physiological prothrombotic changes, such as an increase in cholesterol levels, fibrinogen concentrations and platelet aggregation.[57,58] Peripheral vasoconstriction induced by low temperatures increases blood pressure and gives rise to a release of catecholamines, which leads to an increase in cardiac work and oxygen requirements, destabilization of vulnerable plaques and, on a respiratory level, an increase in the incidence of respiratory infections, which may also be the cause of an increase in the number of cardiovascular decompensations and events.[59]

The effects of high temperatures in our review on ACO for cardiovascular causes are heterogeneous (neutral effects or insignificant increases in risk), whereas, during heat wave periods, there is a majority observation of an increase in ACO. In the 2017 review by Moghadamnia et al.,[49] an increased risk of cardiovascular mortality of 1.3% was observed with high temperatures, with a higher risk at lag 7 (RR = 1.14). Our results are in line with those obtained in the review by Phung et al. (2016) assessing cardiovascular morbidity. After grouping the results, no relationship was observed with high temperatures. However, there was a relationship with exposure to heat waves, indicating an increase in the risk of hospitalization due to cardiovascular causes of 2.2% during these periods. The review by Cheng et al. (2019)[50] on morbidity and mortality during heat waves observed a significant association between heat waves and cardiovascular mortality (RE 1.149), although the association with morbidity was marginal (RE 0.999). In the study by Ponjoan et al. (2017),[62] carried out in Catalonia (Spain), no increases in hospitalizations due to cardiovascular causes were documented during heat waves. Among cardiovascular subtypes, out-of-hospital cardiac arrest exhibits the most consistent increase in risk associated with high temperatures and heat waves, albeit always with a lower magnitude of risk than that observed in the context of low temperatures.

The physiopathological mechanisms involved in the increase in the number of cardiovascular events could be due to the fact that during high-temperature periods the circulating volume has to be redistributed to the skin in order to ease sweating, which, in turn, increases cardiac work, causes hemoconcentration, raises blood viscosity and alters the ionic composition of blood through kidney regulation mechanisms.[60] These changes increase the probability of developing cardiac decompensation, especially in patients with low myocardial reserve, and susceptibility to arrhythmias and out-of-hospital cardiac arrest.[61] In accordance with the studies by Empana et al. (2009)[29] and García-Lledó et al. (2020),[66] it seems that an ischemic origin can be ruled out as the cause of out-of-hospital cardiac arrests, as an increase in ST-segment elevation acute coronary syndromes is not observed and an increase in out-of-hospital cardiac arrests during heat waves is observed. However, more studies are required to identify the precise mechanisms of the effect due to high temperatures and heat waves.

The determination of the MMT has been a common approach in the studies included in our review, albeit with a considerable degree of variability. Consistent with previous research on minimum mortality and morbidity temperatures, a north−south gradient was observed in the threshold values. For instance, Baccini et al. (2008)[65] reported higher minimum mortality thresholds for Mediterranean cities (29.4°C) compared to continental cities in the north (23.3 °C). Similarly, a study conducted in different cities in China by Zhao et al. in 2017[64] also observed an adaptive effect to temperatures, with varying effects depending on the latitude of the city. In cities located farther north, there were more heat-related effects, while in more southern cities, there were more cold-related effects. It is also known that, depending on the sub-disease studied, this will present with a different minimum mortality temperature. Hence, when studying different sub-cardiovascular diseases attended at emergency departments, Wang and Lin (2014)[63] observed that each of them presented with a different minimum mortality temperature. Therefore, climate and geographical region are pivotal in these types of differences in threshold values and it would be of interest to establish at what point emergency systems should be placed on alert for increases in the risk of weather forecast-related events.

Our review has some limitations. The first is inherent in using pre-hospital diagnoses of ambulance medical services, since these are presumptive diagnoses which may vary after hospital care. The second is that most of the studies have been undertaken in southeast Asia or European regions, which would not enable us to draw global conclusions. The third is the methodological variability of the various studies, as well as their way of assessing the effects, which limits comparability among them. The fourth is due to the absence of studies in the same regions which separately assess the different spectra of the health pyramid. This makes it impossible to assess whether ACO show a MMT different from the other strata. The fifth limitation is the heterogeneity in the response of human populations to the thermal environment, which restricts global data synthesis. This variability across different populations may hinder the generalization of the findings of the present study, thus limiting the applicability of its conclusions to other geographical regions. Standardizing the method of assessing the effects in the different studies would be invaluable as far as improving comparability is concerned. More studies should be conducted in different regions to be able to predict at what time and with what intensity a peak in demand might take place in order to have the necessary resources ready to meet it.

■ CONCLUSIONS

This is the first scoping review exclusively using ACO records that synthesizes the real-time effects of the thermal environment on cardiovascular morbidity. The demand for ambulance services due to cardiovascular disease increases during periods of low temperatures and heat waves. High temperatures usually result in a insignificant residual increase in ambulance demand. Out-of-hospital cardiac arrest is the only cardiovascular subpathology that shows an increase in demand for both high and low temperatures. In the current context of climate change and an aging population, it is important to determine the thresholds at which increases in cardiovascular ambulance demand occur and implement public health plans based on meteorological predictions to mitigate the deleterious effects of the thermal environment on cardiovascular health.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge at https://pubs.acs.org/doi/10.1021/envhealth.3c00003.

Table S1 and Table S2 that document the studies included in the systematic review (PDF)

AUTHOR INFORMATION

Corresponding Author

Dominic Royé − *Spanish Consortium for Biomedical Research in Epidemiology and Public Health, (CIBER Epidemiology and Public Health-CIBERESP), 28029 Madrid, Spain; Climate Research Foundation, 28013 Madrid, Spain;* orcid.org/0000-0002-5516-6396; Email: dominic.roye@ficlima.org

Authors

Santiago Gestal Romaní − *Hospital Clínico Universitario de Santiago de Compostela, 15706 Santiago de Compostela, A Coruña, Spain*

Adolfo Figueiras − *Spanish Consortium for Biomedical Research in Epidemiology and Public Health, (CIBER Epidemiology and Public Health-CIBERESP), 28029 Madrid, Spain; Department of Public Health, Faculty of Farmacy, University of Santiago de Compostela, 15782 Santiago de Compostela, A Coruña, Spain; Health Research Institute of Santiago de Compostela (IDIS), University of Santiago de Compostela, 15706 Santiago de Compostela, A Coruña, Spain*

Complete contact information is available at: https://pubs.acs.org/10.1021/envhealth.3c00003

Notes

The authors declare no competing financial interest.

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