Scandinavian Journal of Public Health, 2010; 38: 826–837

REVIEW ARTICLE

High temperatures and health outcomes: A review of the literature

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Abstract

Aims: To analyse the relationship between high temperatures and population health impacts, in terms of mortality and morbidity. *Methods:* A literature search was conducted using Medline to collect data from studies on heat waves, temperature–health impacts curve, risk factors, and preventive measures. All the data collected was published in English and available up to December 2009. *Results:* Numerous studies carried out in Europe following the 2003 heat wave, as well as those conducted prior to this date in Europe and North America, showed an increase in heat wave-related excess mortality. Recent studies have demonstrated that a forward shift of deaths may only explain a very limited quantity of the excess mortality observed (up to 15%) during major heat waves. Moreover, the results seem to exclude that ozone acts as a confounding variable, whilst it remains a potential effect modifier. ***Conclusions:*** **Future research needs to explore the consistency of results in new settings, to quantify the burden of heat-related morbidity and in particular to evaluate the effectiveness of the implemented preventive measures.**

Key Words: *Heat stress disorders, hot temperature, mortality*

Background

The global average temperature has increased by 0.4°C in the past 25 years and is forecast to further increase by between 1 and 3°C by the year 2100. Moreover, even a small shift in the mean temperature will entail a non-linear large increase in the frequency of extreme weather events, such as heat waves [1].

Global warming could determine physical environment alterations, social-economic disruptions, and adverse health consequences for human health on a large scale [2]. Heat-related illness is almost certain to be among the diverse potential heath impacts of global warming and the easiest to quantify [3].

The epidemic occurrence of heat illnesses was documented in Rome in 1694 and in Beijing in July 1743, when 11,000 people died during a 10-day heat wave [4,5]. In August 2003 an exceptional heat wave affected Europe causing some 40,000 deaths and gained the attention of the public and of the policy makers [6].

The aim of the study was to review the epidemiological evidence of the health impact of high temperatures on the population, as well as the associated risk and protective factors.

Materials and methods

The Medline database was searched for published literature up to 18 December 2009. The search was limited to human studies and English language articles, using the following MeSH terms "heat wave", "hot temperature/adverse effects", "heat stress disorders/epidemiology", "heat stress disorders/mortality", and "heat stress disorders/prevention and control". The bibliography of the extracted articles was also checked and the search engine Google was used to identify grey literature.

A total of 113 studies were selected and included in the analysis. Studies of heat-wave episodes and analytic studies of the relationship between

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(Accepted 13 June 2010)

DOI: 10.1177/1403494810377685

temperature and mortality/morbidity in the general population or large segments of it (such as the elderly) were included. Studies of cases or descriptions of cases of heat stroke, studies of effort heat stroke (e.g. in athletes) or heat stroke in particular environments (e.g. factories or desert), as well as those focusing mainly on winter or the effects of cold temperatures, were excluded.

A few more papers concerning the general issues associated with heat disease and climate change were also included in order to provide background information and support the discussion of the results.

Exposure

The intensity of exposure is usually measured at population level from data provided by weather stations, assuming that all persons who live in a specified geographical area experience the same exposure.

The most commonly used measure of exposure is maximum temperature; although some authors have suggested that minimum temperature would be a better indicator of human thermal stress. The minimum temperature index takes into account high night-time temperatures, which are typical of the "urban heat island" and do not allow for recovery from the severe stress experienced during the day. Results from a study in London seem to suggest that average temperature would be an even better indicator [7]. Several other weather variables have been used, including thermal discomfort indices that combine air temperature and humidity (such as heat index, etc.), but it remains difficult to identify a single variable to depict the health impacts of high temperatures.

A study in Toronto and four surrounding cities with similar weather conditions found that the variables having the strongest association with the increase in mortality (maximum temperature, minimum temperature, heat index, number of hours with heat index $>32.0^{\circ}$C) were not consistent for each city [8].

Several approaches have been used to study the relationship between high temperatures and health outcomes. Most commonly, studies have focused on individual heat-wave episodes. A heat wave is a prolonged period of extremely high temperatures, which is variously defined in reference to usual local weather conditions [9].

A secondary approach is aimed at studying the relationship temperature–heath outcomes over the summer months or the entire year and producing a curve to illustrate the different mortality/morbidity rates at various temperatures.

Outcome

The great majority of studies have investigated heat-associated mortality, while only a few have quantified high-temperature morbidity (either as number of accident and emergency department visits or hospital admissions).

In some of the studies, in particular some of the earlier ones, heat mortality cases were identified using the relative International Classification of Disease (ICD) code [10–12]; unfortunately, even during exceptional heat waves, very few deaths are directly attributed to heat and the majority are instead ascribed to other causes, such as cardiovascular and respiratory diseases [13].

Alternatively, "ad hoc" study-specific definitions including clinical signs or symptoms (e.g. premortem or postmortem evidence of a body temperature higher than 40°C) have been adopted to correctly identify the cases; the use of these definitions is limited to small-scale studies for practicality [14–17].

Therefore, a compromise reached between misclassification and practicality is that of using all-cause mortality or natural mortality, i.e. all causes other than accidents or violence.

Results

Heat-wave episodes

Table I lists heat-wave events that have been reported so far in literature. Early studies found a relation between a peak of daily mortality and a concomitantly peak of temperatures [18,19]. Therefore, in order to obtain quantitative estimates of the mortality anomalies, the death counts were compared with a baseline. Reference periods were derived from surrounding days [18,20–22], from the same time period in the previous year [15,16], or the average of 2 or more prior years [23–26]. Eventually, regression analyses, using long-time series, were developed to estimate the expected number of daily deaths adjusted for numerous potential confounders [10,27–35].

Studies on morbidity outcomes have often shown contrasting patterns with no [32,34] or very limited [26,36–38] increases during heat waves. Even more surprisingly, two recent studies showed an increase in morbidity, while no increase in mortality was recorded [39,40].

Mortality displacement

Studies of long temporal periods demonstrated a greater mortality in those years or months with heat

Table I. Studies of mortality during heat-wave episodes.

Reference	Study population	Baseline measure	All-cause mortality
Goldsmith 1986 [18]	Los Angeles (persons >49 years), 1939, 1955 and 1966	Periods of 33 days before and after the heat waves[a]	1939: +546; 1955: +946; 1963: +580
Henschel et al. 1969 [12]	Saint Louis, 9–14 Jul 1966	Jul 1961–1965[a]	246 deaths[b]
Jones et al. 1982 [26]	Saint Louis and Kansas City, Jul 1980	Jul 1978–1979[a]	Saint Louis: +56.8%; Kansas City: +65.2%
CDC 1984 [16]	Rome, Jul–Aug 1983	Jul–Aug 1982	Jul: +408 (+23.0%); Aug: +66 (+4.5%)
Katsouyanni et al. 1988 [11]	Athens, 21–31 Jul 1987		About +2000 deaths
Ramlow and Kuller 1990 [25]	Pittsburgh, 4–18 Jul 1988	Jun–Aug 1983–1987[c]	+107 (+18.2%)[d]
Wainwright et al. 1999 [22]	Philadelphia, 6–16 Jul 1993	8–12 Jun 1993	+26%
CDC 1996 [15]	Chicago, Jul 1995	Jul 1994	+540 (+84.8%)
Whitman et al. 1997 [35]	Chicago, Jul 1995	Jun-Sep 1979–1994[e]	+696 (+31.2%)
Pirard et al. 2005 [112]	France, Aug 2003	2000–2002[a]	+14,800 (+60%)
Vandentorren et al. 2004 [24]	France (13 cities), Aug 2003	1999–2002[a]	Paris: +1854 (+142%); Dijon: +168 (+93%); Marseilles: +571 (+25%); Lille: +200 (+4%)
Conti et al. 2005 [88]	Italy (21 cities), Jun–Aug 2003	Jun–Aug 2002	+3134 (+15.2%)[f]
Michelozzi et al. 2005 [84]	Italy (4 cities), Jun–Aug 2003	1995–2002[a]	Rome: +944 (+18.6%);[d] Turin: +577 (+32.9%);[d] Milan: +559 (+23.3%);[d] Bologna: +175 (+13.9%)[d]
CDC 2004 [85]	Rome, Jun–Aug 2003	1995–2002[a]	+1094[g] (+22.8%)[d]
Simon et al. 2005 [33]	Spain (50 cities), Jun–Aug 2003	1996–2002[e]; 1990–2002[e]	+4151 (+10.6%)
Johnson et al. 2005 [31]	England and Wales, Aug 2003	1998–2002[a]	+2139 (+16%)[d]
Johnson et al. 2005 [32]	England, Aug 2003	1998–2002[e]	+2091 (+17%)[d]
Nogueira et al. 2005 [21]	Portugal (31 cities), 30 Jul–12 Aug 2003	15–28 Jul 2003; 1–14 Jul 2003; 1–28 Jul 2003	+1317; +1228; +1272 according to the three baseline periods
Garssen et al. 2005 [54]	Netherlands, 31 Jul–13 Aug 2003	1995–2002[e]	31 Jul–13 Aug 2003: about +500; Jun–Aug 2003: +1400–2200 (+3–5%)
Empereur-Bissonnet et al. 2006 [40]	France, Jul 2006	2003, 2004, 2005	No increase
No author cited 2006 [23]	England and Wales, 16–28 Jul 2006	2001–2005[a]	+680 (+4%)[d]
Ostro et al. 2009 [13]	California, 15–26 Jul 2006, 15–31 Jul 2006	Jul 2006 non-heat wave days; Jul 1999–2005[a]	Between +160 and +505 according to various models and reference periods
Hoshiko et al. 2009 [20]	California, 15 Jul–1 Aug 2006	8–14 Jul and 12–22 Aug 2006	+655 (+6%)[d]

[a]Average.
[b]Heat-related causes (ICD codes) of mortality.
[c]Moving average.
[d]Significant at $p < 0.05$.
[e]Regression.
[f]Not significant at $p < 0.05$.
[g]Non-traumatic mortality (i.e. all causes except external causes).

waves [41,42]. On the other hand, some other studies of time series with the subsequent identification of heat episodes, according to temperature threshold, observed a deficit of deaths in the following days [43–47].

The deficit in mortality is explained by the hypothesis of the "forward mortality" effect, commonly known as "harvesting". The heat wave does not determine a true excess mortality, but just the short-term displacement of deaths among terminally ill patients who would have died anyway within a few weeks. In the absence of the heat wave, those same deaths would have been more evenly spread out in the weeks thereafter without causing alarm. Results are contradictory showing that there is at least a partial compensation (Table II).

Ozone

The excess mortality is alternatively explained by the hypothesis of the various ambient pollutants, in particular tropospheric ozone, acting as a confounding variable or effect modifier [48].

The photochemical reaction that produces ozone from the precursors (such as volatile organic carbons, carbon monoxide, and methane) is catalysed by temperature. As precursors derived from car pollutions are in abundance in urban areas, solar radiation is the limiting factor of the reaction. Consequently daily variations in temperature and ozone levels can be highly correlated making it difficult to establish their independent and/or synergistic effects on mortality.

While in some of the studies there was no evidence of confounding by air pollution, with results only slightly changed [7,49–51], in other studies ozone and particulate matter were found to account for a substantial part of the excess mortality observed [52–54]. The result of a study in nine French cities is illustrative of results heterogeneity: the relative contribution of ozone varied largely according to the city, ranging from 2.5% to 85.3% [27].

An investigation of the 1987 heat wave in Athens, using a multiple linear regression, showed that while the main effects of air pollutants were not statistically significant, their interaction with high temperature was significant for sulphur dioxide and suggestive for ozone [55].

A significant interaction between air pollution and high temperature on health outcomes was also reported by some subsequent studies [56–59]. However, this has not been confirmed by other studies [48,60,61] and generally evidence is sparse and results are heterogeneous across geographical regions, as demonstrated in a study in 60 American communities [62].

Temperature–mortality curve

The relationship between temperature and mortality appears graphically as a "V" shape with an excess risk for exposure to temperatures above a threshold of minimum mortality (Table III).

The curve has been reported across different latitudes [63–65], and even in subtropical cities [66–68]. Multi-site studies have shown that the threshold is usually higher in southern cities, indicating that residents of colder locations are more susceptible to high values of temperature [49,50,69–75].

Some researchers modelling the relationship in the following period have demonstrated that the association is higher after 0–3 days and then suddenly disappears to be followed by a temporary drop in the number of deaths thereafter; these results seem to support the "harvesting" hypothesis [49,51,73,76–78].

Risk and protective factors

Man has the capacity to acclimatise to high temperatures and behavioural changes can reduce heat exposure; consequently, those individuals who are physiologically fragile or socially disadvantaged are more vulnerable. Published studies refer to risk factors either in heat-wave episodes or in days with temperatures above a specified threshold, as in time-series analyses.

Both women [28,44,50,79–86] and, less frequently, men [15,19] have been reported to be more affected than the opposite gender. However, many of these studies did not adjust for age and much of the difference may be explained by the greater longevity of women; therefore, it is not unlikely that the risk is substantially similar as has been reported by some other studies [87–89].

The elderly have consistently been reported as being at the highest risk, particularly those living in large cities [31]. Despite usually being considered in the low-risk group, a disproportionate number of heat-related deaths were recorded in adults during the 2003 heat wave [28,30,81,90,91]. Excess adult mortality was also reported in other periods [69,86,92]. In early studies, infants were also found to be at high risk [19]; but later studies have usually not confirmed this result [26,79,87].

Populations at greater risk are physiologically frail people, such as people with underlying diseases [93,94], being confined to bed [14,17,93,94],

Table II. Studies investigating deaths displacement.

Reference	Study population	Deaths displacement
Laschewski and Jendritzky 2002 [45]	Baden-Wurttemberg, 1968–1997	Large (about 90%) in the short term (by 2 weeks)
Rey et al. 2007 [81]	France, Jun–Sep 1971–2003	Moderate for four of the six heat waves in the short term (by 20 days). Absent for the Jul 1983 and Aug 2003 heat waves
Hajat et al. 2006 [70]	London, Jun–Sep 1976–2003; Budapest, Jun–Sep 1980–2000; and Milan, Jun–Sep 1985–2002	Some degree of "deaths displacement" in the short term (by 3 days) may have occurred (particularly in London)
Huynen et al. 2001[46]	Netherlands, 1979–1997	Large in the short term (by 15–30 days)
Kysely 2004 [44]	Czech Republic, 1982–2000	Large (up to 90%) in the short term
Hajat 2005 [77]	Delhi, Sao Paulo and London, 1991–1994	High in London (complete compensation) but absent in Delhi in the short term (by 4 weeks). An intermediate pattern was observed in Sao Paulo
Kaiser et al. 2007 [10]	Chicago, 21 Jun–10 Aug 1995	Medium (26.4%) in the short term (by 3 weeks)
Revich and Shaposhnikov 2008 [43]	Moscow, Jan 2000–Feb 2006	Heterogeneous (9% in 15–27 Jul 2001 heat wave, 39% during three heat waves of 2002) in the short term (by 2 weeks)
Conti et al. 2007 [60]	Genoa (persons >74 years), 16 Jul–31 Aug 2003	None in the intermediate term (Sep–Dec 2003). Some deficit of deaths was recorded in the long term (in Jan–Apr 2004)
Fouillet et al. 2006 [28]	France, Aug 2003	None in the intermediate term (by 30 Nov)
Toulemon and Barbieri 2008 [91]	France, 1–21 Aug 2003	Absent in the short term (by Aug 30th), and limited (not more than 15%) in the long term (Jan–Dec 2004)
Le Tertre et al. 2006 [29]	France (9 cities), Aug 2003	Limited (on average 8%; between 1% in Strasbourg and 30% in Lille) in the short term (by 2 Sep)
Hoffman et al. 2008 [113]	Essen, 6–12 Aug 2003	Absent in the short term (by 1 week), except for neoplasm (complete compensation) and cardiovascular disease
Grize et al. 2005 [30]	Switzerland, Jun-Aug 2003	None in the intermediate term (by 31 Dec)

Table III. Studies of the relationship between temperature and mortality.

Reference	Study population	Minimum mortality temperature (°C)	Increase in all-cause mortality for each °C (%)
Curriero et al. 2002 [74]	Eastern USA (11 cities), 1973–1994	Boston: 21.0; Chicago: 18.4; Tampa: 27.1; Miami: 27.2	Boston: +1.1; Chicago: +0.4; Tampa: +0.3; Miami: +0.7
Kunst et al. 1993 [51]	Netherlands, 1979–1987	16.5	
Nicholls et al. 2008 [66]	Melbourne (persons >64 years), 1979–2001	About 22	About +3
Vigotti et al. 2006 [71]	Milan, 1980–1989; and Palermo, 1996–1999	Milan residents born in Lombardy: 18.9; Milan residents born in Sicily: 23.6; Palermo residents: 23.2	
Alberdi et al. 1998 [78]	Madrid, 1986–1992	20.3	
Keatinge et al. 2000 [75]	Europe (persons 65–74 years in 6 regions), 1988–1992	North Finland: 15.8; London: 20.8; Athens: 24.2	North Finland: +6.2;[a] London: +3.6;[a] Athens: +2.7[a]
Baccini et al. 2008 [52]	Europe (15 cities), Apr-Sep 1990–2000	Mediterranean region: 29.4; north-continental region: 23.3	Mediterranean region: +3.1;[a] north-continental region: +1.8[a]
Ballester et al. 1997 [65]	Valencia, Apr-Sep 1991–1993	About 24	+2.6
Gouveia et al. 2003 [68]	Sao Paulo, 1991–1994	About 20	0–14 years: +2.6;[a] 15–64 years: +1.5;[a] >64 years: +2.5[a]
Kim et al. 2006 [63]	Korea (4 cities), 1994–2003	Between 27.0 and 29.7	Between +6.7 and +16.3
Zauli Sajani et al. 2002 [64]	Emilia-Romagna, 1995–1998	About 24	
El-Zein et al. 2004 [67]	Beirut, 1997–1999	27.5[b]	+12.3[a]
Revich and Shaposhnikov 2008 [115]	Moscow, Jan 2000–Feb 2006	About 18	+2.8[a,c]
Näyhä 2007 [116]	Finland, 2000–2005	About 12	+0.8
Rocklöv and Forsberg 2008 [76]	Stockholm, 1998–2003	11.5	+1.4[a]

[a]Significant at $p < 0.05$.
[b]Thom index.
[c]Non-traumatic mortality (i.e. all causes except external causes).

hospitalised [83], and beneficiaries of dependency stipends [95]. Also at higher risk are socially isolated people, such as people living alone [14,38], single, widowed, or divorced [28,83], and those not leaving home daily [14,93,96]. In fact, during the Chicago heat wave of 1995, anything that facilitated social contact was associated with a decreased risk of death [14].

Numerous socioeconomic factors found to be significant predictors of vulnerability to heat include: Afro-American ethnic group [10,15,35,87,89,97], low level of education [84,89], lower income [10,14,82], unemployment [69], and heavy physical labour [94]. Contrasting results are provided by the majority of recent studies carried out in Europe, where it has been reported that deprivation has little or no effect on mortality [80,82,83].

The excess mortality is substantially greater in urban areas [26,30,55,69,80,86], probably due to the combined effect of the "urban heat island" and socioeconomic deprivation [98]. Furthermore, populations of more deprived cities are at higher risk, even after adjusting for latitude [74]; and accordingly people in economically underprivileged neighbourhoods within a city are usually more vulnerable to heat [85,99]. Contrasting results have been frequently reported [68,69,80,79].

Housing characteristics also affect vulnerability to heat, with increased risks for those living on higher floors of multistorey buildings and decreased risks for those whose houses are surrounded by vegetation [14,17,94]. It is necessary to consider that housing conditions may serve as a marker of socioeconomic differences and, therefore, the confounding effect is likely to be very large.

Air conditioning is regarded as the strongest protective factor according to the results of one meta-analysis [93], some case–control studies [14,17,94,96], and other types of studies [18,72,100], despite the likely confounding by socioeconomic status.

Heat-wave response plans

The Chicago heat wave in 1995 and the European heat wave in 2003 raised awareness of the serious heat-wave consequences led to the setting up of heat watch warning systems in numerous cities in North America and in Europe [101,102].

Heat watch warning systems use local weather forecasts to predict heat-related effects on human health and activate a response plan [103]. Simple methods based on air temperature threshold and duration were used in the primordial systems, developed in the 1990s. Subsequently to implement the accuracy of the systems, more complicated approaches were adopted, such as models able to forecast specific weather types along with two or more stages of responses. Intervention plans include issuing mass-media alerts, opening an information "heat-line", facilitating access to air-conditioned shelter facilities, home visits to elderly, and increasing the number of emergency service staff [103].

Some surveys have demonstrated a growing awareness about the heat peril among the population following the adoption of response plans; however this does not apply to behavioural changes [104,105].

Meanwhile, other studies have compared the health effect of two successive heat waves before and after finding a reduction in mortality [106–109] and morbidity [110]. Alternative or supplementary explanations cannot be excluded and contrasting results have been reported [111].

Discussion

Despite the intrinsic limitations of the methodology, the numerous studies collated from the literature search provide evidence of excess mortality associated with heat waves and seem to exclude some of the more common alternative explanations.

The direct comparison of the estimated number of excess deaths across studies is made difficult by the heterogeneity of heat-wave definition and baseline calculations. In defining a heat wave, timing, intensity, duration, and the lag effect are of utmost importance in producing the estimated number of related deaths [46]. In general, the most marked effects have been observed in those episodes occurring early in the summer [7,44,77], where temperature percentiles were at their highest intensity [70] and had a longer duration, with non-linear growth of deaths [88]. The lag effect, that is to say the time interval, which results most significant when associating heat and mortality, is considered to be 0–3 days [69,73]; different type-specific causes of death show different lag times [46,73]. Nevertheless, there appears to be some consistency in the results. For example, the number of excess deaths during the 2003 heat wave in France, calculated using the most rudimentary (average August 2000–2002) and the most sophisticated (regression on 1979–2002 controlled for confounders) baseline, proved to be very similar (14,802 and 14,748 deaths, respectively) [91]. Other authors obtained similar results [28,112]: a consistency of results during the 2003 heat wave was also reported in England [31,32] and in Rome [84,85]. In addition, very similar results across different studies were also reported during the

heat waves which struck Chicago in 1995 [15,35] and California in 2006 [13,20].

The alternative hypotheses used to explain the excess mortality observed are mortality displacement and the confounding effect (or effect modification) caused by heavy air pollution and more specifically ozone pollution. With reference to the former hypothesis, recent studies carried out after the 2003 heat wave in Europe show that a forward shift of deaths account for only a limited fraction (up to 15%) of the excess mortality registered, at least in regard to the major heat-wave episodes [28,29,91,113]. Regarding the latter of the explanations, there is a growing body of evidence that the link between high temperatures and adverse health effects is independent from air pollution; while some results seem to indicate that ozone, particularly in high concentrations, may act as an effect modifier and amplify the effect of high temperatures on mortality [56].

Studies on the temperature–mortality relationship have highlighted a certain adaptation to high temperatures in warmer geographical locations and in the course of time [69,72,74]. The reduction in heat sensitivity observed in the last decades has usually been explained by a wider usage, particularly in the USA, of air conditioning. Air conditioning has also been confirmed by many analytic studies [100] to be the strongest protective factor, at least in North America.

On the other hand, the most important risk factors are old age, pre-existing illnesses, low socioeconomic status, living alone, and living in the "urban heat island". Hence population ageing (particularly in industrialised countries) and growing urbanisation (particularly in developing countries) may further accentuate the heat effects [3]. Therefore in order to counteract the adverse effects of extreme high temperatures, collective preventive measures are likely to be more appropriate and equitable. Among numerous public health actions implemented, the most important and comprehensive are represented by the health watch warning systems. Various systems, regarding criteria for issuing a warning and activities included in the response plan, have been instituted in numerous cities, but their effectiveness has yet to be demonstrated [114].

Conclusions

There is a necessity for more solid evidence regarding the correlation between high temperatures and adverse health effects and, needless to say, the impact on morbidity. It goes without saying that more research needs to be made on a wider geographical scale. This is reflected in the large number of studies carried out over the last 2 years in new settings such as Scandinavia and Russia [43,76,115,116] as well as regarding the morbidity outcomes [36,39,117,118]. Findings of the literature review suggest that in order to improve research quality and to allow for a comparison of results across heat-wave episodes, some minimum epidemiological criteria, such as those proposed by Whitman et al. [35], need to be followed. Whilst, for a more in depth understanding of the global warming effect on different geographical areas and diverse population groups, further analytic studies (such as case-crossover), health surveillance, and continuous evidence reviewing are fundamental [69]. Furthermore, multisite studies are particularly warranted [56] in order to define the role of ozone as an effect modifier.

Lastly, it is of the utmost importance that an evidence-based evaluation is performed to ascertain whether the response plan serves its function in public health and in order to assure its support and implementation. In this regard, Kovats and Kristie have proposed minimum criteria in order to evaluate the response plan's effectiveness [103].

In any case, response plans cannot be the only way to protect the population, because even short periods of high temperatures of 1–3 days (usually considered not long enough to define as a heat wave and activate a response plan) seem to cause excess mortality [69,119]. Some authors have estimated that heat waves account for as few as one-fifth of total heat deaths [70]. Therefore, more comprehensive strategies, which aim to enhance the health of the elderly and their involvement in social activities, along with projects aimed at reducing the "urban heat island" (such as promoting environmental education, and increasing green zones in cities), are also needed [2,9].

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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