

Fig. 2. Temperature profiles for different exposure levels for 1995 Chicago heat event. (Sources: Midwestern Climate Center [1996] for outdoor conditions; Huang [1996] for indoor conditions)

air-conditioned. Fig. 2 shows temperature and Fig. 3 shows relative humidity for these 4 environments in Chicago for July 12 to 16, 1995.

Outdoor conditions were defined by the hourly temperature and humidity obtained from the Midwestern Climate Center (1996), which represented conditions at Chicago O'Hare Airport. For the indoor conditions, we used results from Huang (1996), who used the DOE-2 building energy simulation program to estimate indoor temperatures for various non-air-conditioned building types during the Chicago heat wave. We defined 'indoor unventilated' and 'indoor ventilated' conditions directly from Huang's representative temperatures from a top floor unit in a 1970s-era structure, with relative humidity calculated assuming the same absolute humidity as outdoors. The oppressive indoor conditions are evident in Figs. 2 & 3, as indoor unventilated temperatures were as much as 15°C (27°F) above outdoor temperatures. Even with ventilation, Huang's model predicted indoor temperatures to be as much as 5°C warmer than outdoors, particularly at night. This prediction supports the Chicago experience, where the vast majority of the victims were found indoors and elevated indoor temperatures were thought to contribute to their mortality (Semenza et al. 1996). The air-conditioned environment was defined as having a constant temperature and humidity, isolated from outdoor conditions.

2.2. Core body temperature

Prediction of core body temperature for a given set of exposure conditions is influenced by physiological predisposition. We defined 2 physiological scenarios for use in the Chicago analysis: 'healthy' and 'compromised' individuals. To predict the core temperature of a normal, healthy individual on an hour-by-hour basis, we used a physiological model developed by Givoni & Goldman (1972) based on experiments conducted on healthy young adult males. The relationships are shown in Eqs. (1) to (6). The equilibrium core temperature (T_f) in Eq. (1) is that which would be achieved if the specified activity level and conditions are carried out over a long period.

The long-run equilibrium temperature for healthy individuals is given by:

$$T_f = 36.75 + 0.004M_{\text{net}} + (0.025/clo) \times (T_a - 36) + 0.8 \exp[0.0047(E_{\text{req}} - E_{\text{max}})] (\text{°C}) \quad (1)$$

The second term of this equation is the net internal metabolic heat load, defined as the internal heat generated (M) minus the energy expended in external work (W_{ex}), e.g., moving the body or external loads:

$$M_{\text{net}} = M - W_{\text{ex}} (\text{W}) \quad (2)$$

For a resting individual doing no external work, Givoni & Goldman gave an internal load of $M_{\text{net}} = 150 \text{ W}$.

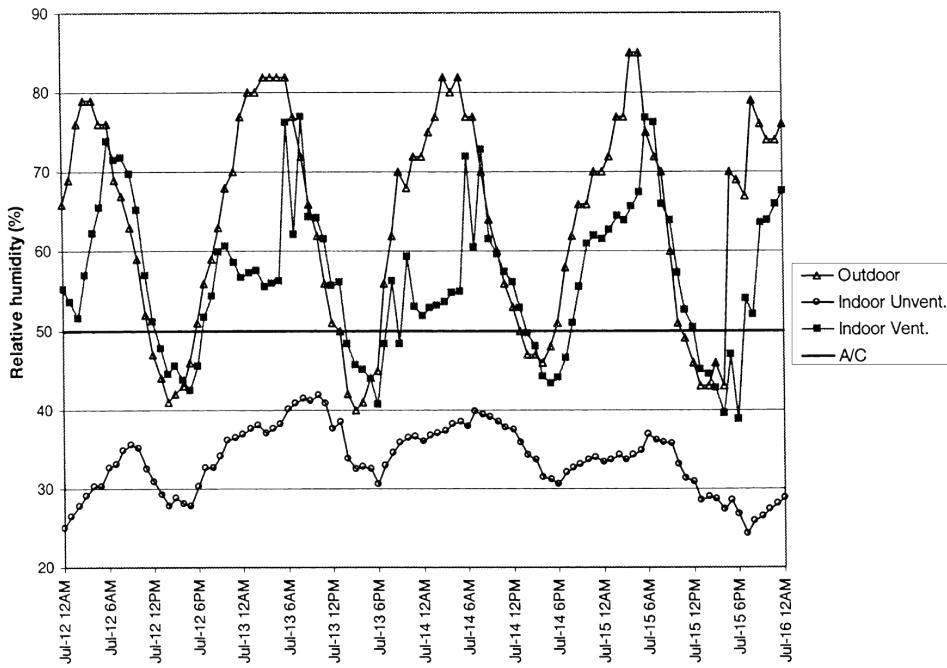


Fig. 3. Humidity profiles for different exposure levels for 1995 Chicago heat event. Outdoor conditions from Midwestern Climate Center (1996). Indoor conditions were calculated from outdoor assuming the same absolute humidity but temperature as shown in Fig. 2

The third term represents the environmental heat load, assuming a skin temperature of 36°C. A dry-bulb ambient temperature (T_a) greater than 36°C will add to the body's heat load. The amount of thermal energy added is dependent on the thermal resistance of the individual's clothing (denoted as clo). Empirically, Givoni & Goldman developed parameters to describe these relationships. The clothing thermal resistance is:

$$clo = 0.74v_{air}^{-0.28} \text{ for shorts and short-sleeved shirt (W }^{\circ}\text{C}^{-1}) \quad (3)$$

where v_{air} = air velocity, assumed to be 0.5 m s⁻¹ for indoor individual at rest.

The fourth term represents the physiological effect of the evaporative difference, that is, between the required evaporative cooling and the evaporative cooling capacity of the environment. Experimental findings led to the exponential relationship shown in Eq. (1). The required evaporative cooling (E_{req}) is the sum of the metabolic and environmental heat loads, which again depend on the thermal resistance of clothing:

$$E_{req} = M_{net} + (R + C) (W) \quad (4)$$

where $(R + C) = (11.6/clo) \times (T_a - 36)$.

The maximum cooling capacity of the environment (E_{max}) is governed by the vapor pressure of the air

(a function of saturation vapor pressure and relative humidity) and the vapor pressure of the skin (44 mm Hg assuming fully saturated and at skin temperature of 36°C). It also depends on the vapor permeability of the clothing (i_m/clo), which was determined empirically.

$$E_{max} = 25.5 (i_m/clo) \times (44 - \varphi_a P_a) (W) \quad (5)$$

where $(i_m/clo) = 0.94v_{air}^{0.28}$ [W (mm Hg)⁻¹] for shorts and short-sleeved shirt, $v_{air} = 0.5$ m s⁻¹ assumed, φ_a = relative humidity, and $P_a = 0.057279 T_a^2 - 1.54435 T_a + 26.05975$ mm Hg, saturated vapor pressure, valid from 20 to 50°C. Quadratic function fitted to saturation pressures from Sonntag & Van Wylen (1982, their Table A.1.1).

For 'compromised' individuals, the rise in T_f above normal is increased by 18%, for reasons discussed in subsequent sections.

In reality, people's ambient conditions and activity levels vary over the course of a day. Givoni & Goldman (1972) gave several formulas to determine the changes of core temperature over time under different 'phases': (1) no activity but a change in environment; (2) beginning activity, and (3) recovery after activity. Combined with the equilibrium temperature, these formulas give an hour-by-hour profile of core body temperature. For the baseline Chicago analysis, we assumed a sedate individual (no physical activity), i.e., phase (1) above,

because it seems likely that most individuals would avoid physical activity during a heat wave. We also assumed 'shorts and short-sleeved shirt' as the appropriate clothing level. The core temperature over time, $T(t)$ is then given by:

$$T(t) = T(0) + T_f \times 0.1^{0.4(t-0.5)} \quad (6)$$

at time t (h) after a change in T_f .

$T(0)$ is the core temperature at the time that the equilibrium temperature T_f starts to undergo a change.

There are limited data to predict the core body temperature of compromised individuals. Drinkwater & Horvath (1979) studied the effect of age on women's physiological response to exercise in elevated temperatures; fitted curves show rectal temperature, skin temperature, and sweat rate as functions of age. We assumed that a 'compromised' physiology corresponds to that of a 65 yr old. Based on Drinkwater & Horvath's data, we assumed that 'compromised' individuals would have a rectal temperature elevation approximately 18% greater than that of a 'healthy' individual, for the same environmental, exposure, and activity conditions.

Clearly it is an oversimplification to categorize the population into only 'healthy' and 'compromised' individuals and equate these with 'under 65' and '65 and over.' As more information becomes available, any number of physiological segmentations can be accommodated within the framework. In Section 3.4.1, we examine the effects of varying some of these physiological parameters.

2.3. Adaptive behavior responses

An individual experiencing heat stress may have options for reducing heat exposure. The victims of the Chicago heat wave were mainly elderly and poor with quite limited options. Many did not have access to air-conditioning or transportation; others could not open their windows (Changnon et al. 1996). Given even a limited range of choices, some people may still choose to do nothing. For example, the elderly may be able to open their windows or turn on the air-conditioning, but may choose not to. Changnon et al., as well as T. Shen of the Public Health Department in Springfield, Illinois (pers. comm. 1997), suggested that some of the Chicago elderly were perhaps unwilling to open windows or go to neighborhood cooling centers due to a fear of crime.

For the Chicago analysis, we considered the key behavioral response to an elevated core temperature to be reducing the heat exposure by moving to an air-conditioned space or by opening windows. We developed 3 adaptive response scenarios, which are described later.

2.4. Equivalent temperature over time

Core temperature is a primary measure of the potential adverse consequences of exposure to heat, but by itself is insufficient to describe heat strain, because the length of time at an elevated core temperature is an important determinant of the effects of heat. Bynum et al. (1978) cite a number of studies showing that at elevated temperature 'subclinical cascades of pathological events are initiated and cellular dysfunction and tissue damage occur.' Bynum et al. plotted cell culture lethality data from 3 other studies and developed a relationship for normalizing a unit time at a given temperature to an equivalent time at 42°C. So, we combined time and core body temperature into a single metric (denoted t_{42}) using Bynum's relationship:

$$t_{42} = 60 \times \int_0^{24} [2.1196 \times 10^{-25} \exp(1.353 \times T(t))] dt \text{ (min)} \quad (7)$$

where $T(t)$ is the actual realized core temperature at time t . We calculated t_{42} over each consecutive 24 h period and chose the largest t_{42} as a metric for heat strain.

2.5. Health effects index (HEI)

The final step in the framework is to combine the calculated t_{42} with the physiological scenario to arrive at a HEI:

$$HEI = 100 \times [L(t_{42}) - L(0)] / [1 - L(0)] \quad (8)$$

where $L(x) = 1/[1 + \exp(a - bx)]$, with parameters $a = 3.45$, $b = 0.53$ for healthy physiology and $a = 2.0$, $b = 0.7$ for compromised physiology.

As in the calculation of core temperature, we categorized individuals into 'healthy' and 'compromised.' We defined the HEI to take on values between 0 and 100. For healthy individuals, there are only sparse quantitative data on adverse heat-related health effects which could be used to develop the HEI relationship to t_{42} . Wyndham et al. (1965) and Schwartz et al. (1977) presented the proportion of men who 'dropped out' of groups heat tested under various conditions. Final average rectal temperatures were reported for each group tested. We estimated the trajectory of core temperature for each group using the relationships given by Givoni & Goldman (1972). From this trajectory, we calculated the corresponding average t_{42} for each group, defined the corresponding HEI as the proportion of subjects who dropped out due to exhaustion or other symptoms, then correlated the t_{42} with the HEI. Although there is considerable scatter, we manually fitted a logistic (S-curve) function to the data from Wyndham et al. (1965) and Schwartz et al. (1977), as

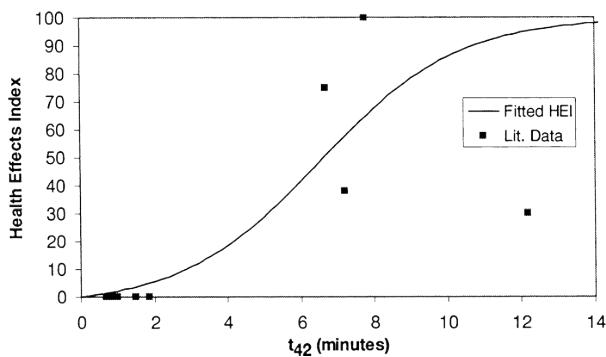


Fig. 4. Health effects index (HEI) as a function of t_{42} for healthy individuals. (■) Data points derived from Wyndham et al. (1965) and Schwartz et al. (1977). Solid curve represents fitted logistic function

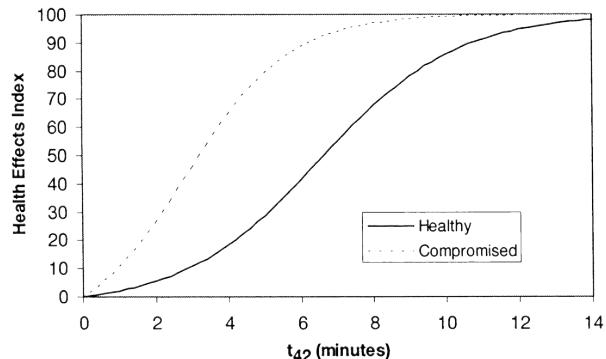


Fig. 5. Comparison of HEI as a function of t_{42} for healthy and physiologically compromised individuals

shown in Fig. 4 and Eq. (8). This function was chosen because it seemed intuitive that a small increase in t_{42} should lead to only a slight increase in the HEI, i.e., the slope of the curve at $t_{42} = 0$ should be very flat; and the curve should be asymptotic to 100 (the maximum possible HEI) as t_{42} becomes large.

For physiologically compromised individuals, we expect a higher chance of adverse effects for any given t_{42} as compared to healthy individuals. We modeled this by shifting the curve in Fig. 4 to the left, so that the HEI is higher for each t_{42} . We chose an approximate 50% shift to the left and then adjusted the parameters (Fig. 5). This factor was chosen because tolerance time was approximately halved for elderly (65 and over) women exercising in the heat compared to young adults (Drinkwater & Horvath 1979). Because there is a continuum of curves among the population, other factors could be chosen. However, we did not find sufficient data in the literature to justify anything more precise than

the approximate 50% factor. The sensitivity of our results to these assumptions are presented in Section 3.4.1.

Note that a compromised individual is at a disadvantage compared to a healthy individual in at least 2 ways. A compromised individual is likely to have a greater rise in core temperature in response to unfavorable environmental conditions, and for a given core temperature, a compromised individual will have a greater HEI. The severity of this 'double jeopardy' effect will vary among individuals.

Although we fitted the HEI based on the fraction of subjects who dropped out of the studies and although the index takes values between 0 and 100, it should not be thought of as a probability of clinical symptoms. This is because the men who 'dropped out' of the studies did so because of discomfort, onset of heat stress symptoms, or core temperature or heart rate exceeding predetermined limits. These manifestations indicate possible heat-related effects, but do not imply that medical attention was required. In fact, a (probably large) fraction of the potentially affected population in a heat wave would avoid any actual adverse health consequences by taking offsetting measures, such as altering their usual activity pattern. Thus, the affected population will consist of a combination of individuals who (1) altered their lifestyle to avoid a clinical effect, perhaps experiencing reduced work productivity or other economic or lifestyle inconvenience; (2) suffered discomfort but not illness while continuing a somewhat modified activity pattern; (3) suffered actual heat stress related effects and required medical attention but recovered; and (4) required medical attention and did not recover.

Thus, the index is best thought of as a relative severity metric, i.e., an HEI of 80 is twice as severe as 40, but it does not mean that 80% of the population subjected to these conditions would necessarily experience heat-related illness. Because the index is a relative severity, it is appropriate to compare HEI values for different population subgroups in the same manner as the Semenza et al. (1996) comparison of mortality odds ratios (defined as the ratio of mortality rate in deaths per 100 000 in one subgroup as compared to another).

3. APPLICATION OF THE HEI TO THE 1995 CHICAGO EVENT

We calculated the HEI for the 1995 Chicago event under different scenarios of ambient conditions, exposure, and adaptive responses. We defined the time for the study to start as midnight before July 12 and to end as midnight before July 16. We analyzed the hourly

Table 1. Health effects index (HEI) for population subgroup and (constant) exposure level, no activity, 1995 Chicago heat event

	Indoor unventilated	Indoor ventilated	Outdoors	Air-conditioned
Healthy	21.2	9.6	5.6	0.0
Compromised	77.3	45.1	30.3	0.0

environmental exposures and physiological core temperature responses.

3.1. Comparison of individual exposure conditions

For both healthy and compromised individuals, we calculated the index for each of the 4 exposure conditions: indoor unventilated, indoor ventilated, outdoor, and air-conditioned. Each individual was assumed to be in the same exposure condition for the full 4 d time period. The resulting HEI values are shown in Table 1. A healthy person in the most extreme exposure condition, indoor unventilated, was 3.8 ($21.2 + 5.6$) times more likely to experience an adverse effect than a healthy individual who was outdoors. The act of opening the windows was protective by a factor of about 0.45 ($9.6 + 21.2$) for healthy individuals. Compromised individuals have a much higher HEI under all exposure conditions, resulting from the 'double jeopardy' effect.

3.2. Effect of individual adaptive behavior

Next, we considered the effect of a particular adaptive behavior. We postulated that individuals in unventilated indoor spaces would turn on their air-conditioners (or move to an air-conditioned location) as an adaptive response to heat when their core temperature rose above a certain level. We assumed that persons would begin to sense heat discomfort when their core temperature reached 0.50°F (0.28°C) above normal, where normal is defined as core temperature at rest in air-conditioning (25°C , 50% relative humidity). This calculation resulted in a threshold temperature of

Table 2. Impacts of adaptive behavior on HEI, indoor unventilated starting condition, 1995 Chicago heat event

	No adaptation	Go to air-conditioning	
		1 h	3 h
Healthy	21.2	4.1	1.3
Compromised	77.3	21.4	13.8

37.17°C . We assumed that individuals would remain in the air-conditioned location until either their body temperature dropped back below threshold or a certain amount of time passed (denoted as the 'reversion time'), whichever was longer, after which they returned to their previous exposure condition. We evaluated reversion times of 1 and 3 h in air-conditioning. The resulting HEI values are shown in Table 2. The HEI for compromised individuals was reduced by a factor of 5.6 ($77.3 + 13.8$) by moving from indoor unventilated conditions to air-conditioning for 3 h. This compares well with the results of Semenza et al. (1996), who reported that access to air-conditioning was protective with a risk odds of 0.2 (reduction in mortality risk by a factor of 5). Semenza et al. arrived at their results by comparing potential predictive factors associated with the decedents versus the geographically nearest control subject of the same age.

3.3. Population-weighted ratios

The next analysis combined exposure situations with estimated population segments to arrive at a population-wide HEI. As shown in Table 3, we calculated the HEI for each of 4 mutually exclusive exposure situations: those having air-conditioning, those without air-conditioning but with immediate access to air-conditioned spaces, those without air-conditioning who opened their windows, and those without air-conditioning who did not open windows. We estimated the fraction of the population in each group and calculated a weighted average HEI for healthy and compromised populations.

As mentioned previously, we defined the 'compromised' population to be the individuals at least 65 yr old; the 'healthy' population was the remainder. The US Department of Housing and Urban Development (1997) reported that 88% of households in the Chicago area having at least 1 resident 65 yr or older had either central or room air-conditioning. The rate was 84% with air-conditioning for 'nonelderly' households. Of the case and control groups analyzed by Semenza et al. (1996), about 55% of those who did not have air-conditioning visited neighborhood cooling centers or other air-conditioned places (55% of 12%, or 6.6%). Because the case and control groups in Semenza et al.'s study were primarily elderly, we used the 55% fraction for our 'compromised' population. For the healthy population, we assumed 70% of the persons without air-conditioning would go to air-conditioning. We assumed that the elderly would be less likely than healthy persons to leave home to find air-conditioning because of more restricted mobility, fear of crime, and

Table 3. Estimated population distribution and HEI for healthy and compromised individuals, 1995 Chicago heat event

		% of group	HEI	Weighted average HEI
Healthy	Air-conditioning	84.00	0.0	
	Go to air-conditioning	11.20	1.3	
	Open windows	4.80	9.6	
	Do not open windows	0.00	21.2	0.61
Compromised	Air-conditioning	88.00	0.0	
	Go to air-conditioning	6.60	13.8	
	Open windows	5.26	45.1	
	Do not open windows	0.14	77.3	3.39
				Ratio = 5.6

reduced awareness that the heat was causing them additional physiological distress.

Finally, we needed to estimate the fractions of people without air-conditioning who would and would not open their windows for ventilation. We assumed that 100% of the healthy population without air-conditioning would open their windows. Of the persons in the US 65 yr or older, 1.3% were home health care patients needing assistance with light housework (US Department of Commerce 1993). We assumed that twice this fraction, 2.6% of the persons without air-conditioning, would either not be able to open their windows or would choose not to open windows because of fear of crime and would remain in their unventilated dwellings. The remaining fraction of compromised individuals were assumed to open their windows.

Table 3 shows the population-weighted averages of the subcategory HEIs. The ratio of the compromised to the healthy HEI was 5.6, which is substantially less than the actual mortality rate ratio of 11.6 (NOAA [1996] indicated 104 deaths per 100 000 age 65 yr and over, compared to 9 deaths per 100 000 for those under 65 yr). This difference may result because the HEI is intended to measure potential adverse effects which may not lead to clinical symptoms or death. There may be many more 'healthy' than 'compromised' individuals who experience a heat-related effect of discomfort or symptoms, but subsequently recover. Because the same exposure may lead more directly or rapidly to serious illness and death in the physiologically compromised, the mortality ratio would be larger than the HEI ratio. For example, suppose that a healthy population had an incidence of health effects (as measured by the HEI) of 100 cases per 1000 persons, of which 5% (5 cases) led to death. Further, suppose that a compromised population had an incidence of 400 per 1000 persons, of which 20% (80 cases) led to death. Then, the mortality ratio would be $80 + 5 = 16$, while the HEI ratio would be $400 + 100 = 4$.

Heat-related effects in the healthy population, although much less likely to lead to death, nevertheless

are more common and may have significant adverse consequences, such as direct heat illness, fatigue leading to illness or accidents, and reduced work productivity. This supports the notion that mortality rates may not be the best metrics for evaluating the effectiveness of risk-reduction strategies.

3.4. Sensitivity analysis

The framework described in this paper utilizes a preliminary set of models for each of the mechanistic steps leading from ambient conditions through to the HEI. Uncertainties in models and data will propagate through to the resulting HEI values, so it is important to understand the sensitivity of the HEI to these uncertainties. We examined the sensitivity of the HEI results of the Chicago analysis to variations in certain model parameters and inputs. This may help focus research efforts in targeting model improvements to the links in the framework. We did not investigate uncertainty in the population segmentation or the resulting population-weighted HEI values.

3.4.1. Sensitivity to physiological parameters

The HEI S-curve is based on a sparse set of data reported in the literature. We examined the results of different assumptions regarding the 'steepness' of the S-curve for both healthy and compromised physiologies. The 'steep' and 'shallow' S-curves for healthy and compromised physiologies are shown in Fig. 6. The steep and shallow curves for healthy physiology were chosen to respect the small number of data points in Fig. 4 but with different degrees of curvature. The corresponding curves for compromised physiology were then chosen to achieve approximately a 50% shift to the left, as was done for the nominal curve. The shape of the curve evidently has a significant impact on the resulting HEI values (top part of Table 4); the differ-

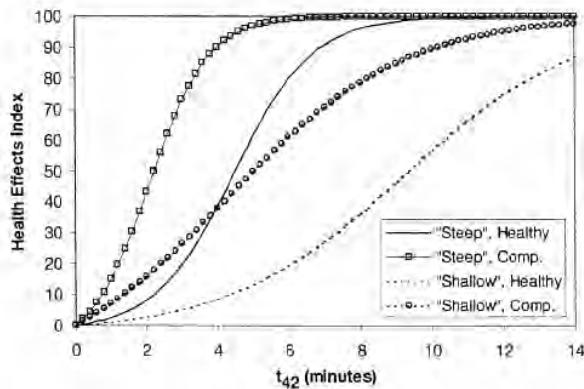


Fig. 6. Sensitivity scenarios for HEI S-curve

ence in HEI can be up to a factor of 4 between the shallow and steep curves describing healthy physiology. Clearly there is a need to develop additional data for use in predicting the likelihood of adverse effects given the time-at-temperature.

For compromised individuals, the literature indicates that the core temperature rise for a given adverse ambient condition will be greater than that for a healthy individual. We examined the sensitivity of the results to different assumptions of this rise, using the nominal S-curve. The results are shown in the lower portion of Table 4. While there is an effect, it is not as pronounced as the differences resulting from the different S-curve shapes.

3.4.2. Sensitivity to adaptive response threshold

The Chicago analyses examined the effect of the adaptive response of moving to an air-conditioned location. The threshold for individuals taking action was set at a core body temperature of 37.17°C, which

corresponded to 0.50°F above normal. We examined the sensitivity of the HEI to differing threshold assumptions, as shown in Table 5; the additional scenarios were adaptive response thresholds at 1.0 and 1.5°F above normal (37.45 and 37.72°C, respectively). As expected, as the threshold increases, so does the HEI, because persons do not move as readily to escape the heat. The additional relief provided by going to air-conditioning for 3 h instead of 1 h also diminishes as the threshold temperature increases.

4. FUTURE DIRECTIONS AND REFINEMENTS

The framework presented in this paper provides a structure in which transdisciplinary research results may be integrated. Sensitivity analyses on each module can point out the areas of greatest uncertainty or research potential. As new information and more sophisticated models become available, they can be readily incorporated, refining or replacing existing modules. The models presented herein, and the spe-

Table 5. Sensitivity of HEI to core temperature threshold for adaptive behavior, indoor unventilated starting condition

	Core temp. threshold (°C)	Go to air-conditioning	
		1 h	3 h
Healthy	37.17	4.1	1.3
	37.45	11.5	9.8
	37.72	21.2 ^a	21.2 ^a
	No adaptation	21.2	21.2
Compromised	37.17	21.4	13.8
	37.45	49.2	43.5
	37.72	70.2	69.8
	No adaptation	77.6	77.6

^aCore temperature does not exceed threshold, so no adaptive behavior occurs

Table 4. Sensitivity of HEI to physiological parameters

Physiology	Parameter	Parameter value	Exposure condition			Air-conditioned
			Indoor unventilated	Indoor ventilated	Outdoors	
Healthy	HEI	Shallow	9.9	4.8	2.9	0.0
	S-curve	Nominal	21.2	9.6	5.6	0.0
		Steep	45.6	17.3	8.6	0.0
Compromised	HEI	Shallow	47.8	25.8	17.8	0.0
	S-curve	Nominal	77.3	45.1	30.3	0.0
		Steep	96.2	71.8	49.5	0.0
Compromised	Core temp. rise over healthy	0 %	69.6	42.5	27.2	0.0
		18 %	77.3	45.1	30.3	0.0
		30 %	82.0	46.9	31.3	0.0

cific application to the Chicago event, are only first steps in this process. The HEI framework and these initial modules should be applied to other heat events in other cities to determine their robustness and forecasting potential. More sophisticated segmentation of physiological condition is warranted when more data become available, including varying degrees of compromised health and varying levels of heat acclimatization. Additional research into physiological response, such as the shape of the HEI S-curve, is needed to put the models on a firmer experimental foundation.

The framework is flexible and can be used to model possible future events in addition to retrospective analyses of historical heat waves. It also can be used to evaluate various scenarios of changing demographics, environment, or behavior. Examples include: What is the beneficial effect (in terms of reduced HEI) for the elderly to go to an air-conditioned location for the hottest hours of the day? How might adverse effects have increased if the heat wave were an additional 1°C hotter throughout? How might heat stress and strain have been lower if excessive heat had only occurred during the daytime, with the nights remaining 'normal'? How would healthy persons performing physical activity be at risk from the heat, and how much might this risk be lessened by reducing or ceasing activity? The framework and models, appropriately modified to target specific exposure, physiology, activity, and behavior scenarios, can address these and other questions. The population can be broken into as many categories as desired, as long as data are available to perform the segmentation and to develop the required relationships for each category in each step of the model. Once the functional relationships and segmentation are in place, any number of comparisons and 'what if' scenarios can be analyzed. Various management and mitigation strategies could be examined to evaluate their potential benefits. Examples could include increasing the number or accessibility of local cooling centers, or developing a neighborhood system where residents could check-in with each other. The effectiveness of these programs could be evaluated through changes in demographics or exposure, e.g., a better warning system, or increased contact between social workers and the elderly, which could be modeled as an increase in the fraction of the population who move to air-conditioned locations.

The framework and model also could be used to investigate issues related to the potential effects of global climate change on heat-related illnesses through an increase in severity and duration of extreme temperature events. The exposure types used in this analysis represented the historical conditions (outdoor and indoor) which occurred during the Chicago

event. The model could also be used to evaluate different exposure conditions representing any number of 'day-types' with different probabilities of occurrence. These day-types could reflect, for example, a hot day and cool night, or a hot day and warm night with high humidity, etc. An individual's heat strain response would be different for each of these day-types and would still depend on physiology, activity level, exposure, and adaptive behaviors. Climate change would enter the model through the frequency of occurrence of the day-types. For example, global warming might increase the frequency of hot-day, warm-night occurrences, and reduce warm-day, cool-night incidences. The expected HEIs could be compared for different scenarios. The transparency and structure of the framework, allowing integration of models and relationships from different disciplines and refinement as new information develops, make it a valuable evaluation tool.

Acknowledgements. This work was supported by EPRI under Contract WO 4420-01.

LITERATURE CITED

- Bynum GD, Pandolf KB, Schuette WH, Goldman RF, Lees DE, Whang-Peng J, Atkinson ER, Bull JM (1978) Induced hyperthermia in sedated humans and the concept of critical thermal maximum. *Am J Physiol* 235:R228–R236
- Centers for Disease Control and Prevention (1995) Heat-related mortality—Chicago, July 1995. *Morb Mort Wkly Rep* 44:577–579
- Changnon SA, Kunkel KE, Reinke BC (1996) Impacts and responses to the 1995 heat wave: a call to action. *Bull Am Meteorol Soc* 77:1497–1506
- Drinkwater BL, Horvath SM (1979) Heat tolerance and aging. *Med Sci Sports* 11:49–55
- Givoni B, Goldman RF (1972) Predicting rectal temperature response to work, environment, and clothing. *J Appl Physiol* 32:812–822
- Huang J (1996) DOE-2 analysis of indoor temperatures in typical apartment buildings during the July 1995 Chicago summer heat wave. Lawrence Berkeley Laboratory, Berkeley, CA
- Japsen B, Moore JD Jr (1995) Heat wave scorches emergency services. *Mod Healthcare* 25:3
- Kattenberg A, Giorgi F, Grassl H, Meehl GA, Mitchell JFB, Stouffer RJ, Tokioka T, Weaver AJ, Wigley TML (1996) Climate models—projections of future climate. In: Houghton JT, Meira Filho LG, Callander BA, Harris N, Kattenberg A, Maskell K (eds) *Climate change 1995: the science of climate change*. Intergovernmental Panel on Climate Change. Cambridge University Press, Cambridge
- Kunkel KE, Changnon SA, Reinke BC, Arritt RW (1996) The July 1995 heat wave in the midwest: a climatic perspective and critical weather factors. *Bull Am Meteorol Soc* 77: 1507–1518
- Midwestern Climate Center (1996) July 10–17, 1995 hourly temp, wet bulb temp, and rel. humidity for Chicago O'Hare. Data printout index #111549, MCC, Champaign, IL

- NOAA (1996) July 1995 heat wave. Natural disaster survey report. National Oceanic and Atmospheric Administration, National Weather Service, Silver Spring, MD
- Schvartz E, Shibolet S, Meroz A, Magazanik A, Shapiro Y (1977) Prediction of heat tolerance from heart rate and rectal temperature in a temperate environment. *J Appl Physiol: Respir Environ Exercise Physiol* 43:684–688
- Semenza JC, Rubin CH, Falter KH, Selanikio JD, Flanders WD, Howe HL, Wilhelm JL (1996) Heat-related deaths during the July 1995 heat wave in Chicago. *N Engl J Med* 335:84–90
- Sonntag RE, Van Wylen GJ (1982) Introduction to thermodynamics, classical and statistical, 2nd edn. John Wiley & Sons, New York
- US Bureau of the Census (1993) Statistical Abstract of the United States, 113th edn. US Bureau of the Census, Washington, DC
- US Department of Housing and Urban Development (1997) American housing survey for the Chicago metropolitan area in 1995. Current housing reports H170/95-22, US Department of Housing and Urban Development, Washington, DC
- Voelke R (1995) Probe of heat wave deaths under way. *J Am Med Assoc* 274:595–596
- Wyndham CH, Strydom NB, Morrison JF, Williams CG, Bredell GAG, Maritz JS, Munro A (1965) Criteria for physiological limits for work in heat. *J Appl Physiol* 20: 37–45

Editorial responsibility: Laurence Kalkstein,
Newark, Delaware, USA

Submitted: February 17, 1999; *Accepted:* June 1, 2000
Proofs received from author(s): November 20, 2000

ANNUAL REVIEWS **Further**

Click here for quick links to Annual Reviews content online, including:

- Other articles in this volume
- Top cited articles
- Top downloaded articles
- Our comprehensive search

Heat Stress and Public Health: A Critical Review

R. Sari Kovats and Shakoor Hajat

Public and Environmental Health Research Unit (PEHRU), London School of Hygiene and Tropical Medicine, London WC1E 7HT, United Kingdom; email: Sari.Kovats@lshtm.ac.uk, Shakoor.Hajat@lshtm.ac.uk

Annu. Rev. Public Health 2008.29:41-55

First published online as a Review in Advance on November 21, 2007

The *Annual Review of Public Health* is online at <http://publhealth.annualreviews.org>

This article's doi:
[10.1146/annurev.publhealth.29.020907.090843](https://doi.org/10.1146/annurev.publhealth.29.020907.090843)

Copyright © 2008 by Annual Reviews.
All rights reserved

0163-7525/08/0421-0041\$20.00

Key Words

heat waves, early warning, mortality

Abstract

Heat is an environmental and occupational hazard. The prevention of deaths in the community caused by extreme high temperatures (heat waves) is now an issue of public health concern. The risk of heat-related mortality increases with natural aging, but persons with particular social and/or physical vulnerability are also at risk. Important differences in vulnerability exist between populations, depending on climate, culture, infrastructure (housing), and other factors. Public health measures include health promotion and heat wave warning systems, but the effectiveness of acute measures in response to heat waves has not yet been formally evaluated. Climate change will increase the frequency and the intensity of heat waves, and a range of measures, including improvements to housing, management of chronic diseases, and institutional care of the elderly and the vulnerable, will need to be developed to reduce health impacts.

F21-1172

148 of 282

41

INTRODUCTION

HHWS: heat health warning system

NWS: national weather services

Heat is a natural hazard, and much is known about the effects of high temperatures on the human body. Episodes of extreme temperature can have significant impacts on health and present a challenge for public health and civil protection services. Further, one of the more certain impacts of future anthropogenic climate change will be an increase in heat waves in many populations, and such heat waves will be more intense (35).

Human populations are acclimatized to their local climates, in physiological, behavioral, and cultural terms. There are clear and absolute limits to the amount of heat exposure an individual can tolerate. However, human capacity to adapt to varied climates and environments is considerable. Most homes have an indoor temperature of 63°F to 87°F, and people do not comfortably live in temperatures outside this range. The tolerance range of an individual is usually much less than this and will narrow with age or illness.

This article reviews the current epidemiological information on the impacts of heat waves and hot weather and the implications for public health. This topic has become a rapidly growing area of epidemiological research. Since the 2003 heat wave, most countries in Western Europe have implemented some public health measures for heat waves, mostly in the form of heat health warning systems (HHWS). The relatively rapid development of these systems is a success of public health. However, although more is known about who is most vulnerable to heat waves, there is very limited evidence on the most effective ways to prevent heat-related mortality, particularly in community settings.

The Effects of Heat on the Body

Healthy adult persons have efficient heat regulatory mechanisms, which cope with increases in temperature up to a particular threshold. The body can increase radiant, convective, and evaporative heat loss by vasodilatation and perspiration (43). Experi-

F21-1172

149 of 282

mental data have been used to describe a wide range of thermal indices (more than 300) (79) and for setting important occupational and other standards to limit exposure to heat and the associated health effects (77). The physiological effects of heat are reviewed extensively elsewhere, but there is a lack of evidence on heat tolerance in women, in the elderly, and in persons with chronic disease (see below).

High temperatures cause the clinical syndromes of heat stroke, heat exhaustion, heat syncope, and heat cramps (42). Severe heat stroke occurs when the core body temperature exceeds 103°F and leads to multiple organ dysfunction. Heat stroke has a substantial case-mortality ratio, and progression to death can be very rapid (within hours). In survivors, the permanent damage to organ systems (17) can cause severe functional impairment (16) and increase the risk of early mortality (97).

HEAT WAVES AND THEIR IMPACTS ON HEALTH

This review focuses on the prevention of heat-related impacts in community settings. The heat wave in France in August 2003 caused 14,802 deaths in a 20-day period (33). A major heat wave in Athens in 1987 was associated with more than 2000 deaths (39). Other well-studied heat waves include several in the U.S. Midwest region, particularly the 1995 Chicago event (46). What constitutes a heat wave event is loosely defined, and national weather services (NWS) have developed their own definitions on a national or local basis. In practice, the term heat wave is applied to a wide range of meteorological conditions, from moderate to severe. Heat waves, in terms of a disaster or emergency, i.e., that involve some aspect of the overwhelming of public services, are rare. Unfortunately, best practice guidelines had not been developed in Europe or the United States until recently. **Table 1** lists the heat wave events in Europe that have been reported in the health literature. Major heat wave events are also associated with other health hazards such as air pollution episodes,

Table 1 Heat wave events and attributed mortality in Europe (adapted from Reference 52)

Heat wave event	Attributable mortality (% increase)	Baseline measure
1976—London, UK	9.7% increase England and Wales and 15.4% Greater London	31-day moving average of daily mortality in same year
1981—Portugal	1906 excess deaths (all cause, all ages) in Portugal, 406 in Lisbon (month of July)	Predicted values
1983—Rome, Italy	35% increase in deaths in July 83 in 65+ age group	Compared with deaths in same month in previous year
1987—Athens, Greece	estimated excess mortality >2000	Time trend regression adjusted
July 21–31		
1991—Portugal	997 excess deaths	Predicted values
July 12–21		
1995—London, UK	11.2% (768) in England and Wales, 23% (184) Greater London	31-day moving average of daily mortality in previous two years
July 30–August 3		
1994—Netherlands	24.4% increase, 1057 (95% CI 913, 1201)	31-day moving average of previous 2 years
July 19–31		
2003—Italy,	3134 (15%) in all Italian capitals	Deaths in same period in 2002
June 1–August 15		
2003—France	14802 (60%)	Average of deaths for same period in years 2000 to 2002
August 1–20		
2003—Portugal	1854 (40%)	Deaths in same period in 1997–2001
August 1–31		
2003—Spain	3166 (8%)	Deaths in same period 1990–2002
August 1–31		
2003—Switzerland,	975 deaths (6.9%)	Predicted values from Poisson regression model
June 1–August 31 (3 months)		
2003—Netherlands	1400 deaths	Number of degrees above 72°F multiplied by the estimated number of excess deaths per degree (25–35 excess deaths)
June 1–August 23		
2003—Baden-Wuerttemberg, Germany	1410 deaths	Calculations based on mortality of past five years
August 1–24		
2003—Belgium	1297 deaths for age group older than 65	Average of deaths for same period in years 1985–2002
August 4–13		
2003—England and Wales	2091 (17%). Mortality in London region: 616 deaths (42% excess)	Average of deaths for same period in years 1998 to 2002

wild fires, and water and electricity supply failures, which also have implications for public health action.

The excess mortality attributed to a heat wave event is the short-term increase in the numbers of deaths (**Figure 1**), a peak in mor-

tality similar to that seen for very severe pollution episodes. The estimated number of deaths therefore depend on the definition of the heat episode. Reviews have shown that the total impact of a heat wave event will be dependent on a number of factors including

heat wave magnitude, timing in season, population experience of heat wave events, and public health responses (47).

The majority of heat wave studies have considered impacts on mortality because daily deaths data are generally readily available in high-income countries. Heat waves are also associated with increases in emergency hospital admissions (36, 51, 84). During the August 2003 heat wave, an increase in admissions was reported in Spain (10) and in France, where many hospitals were overwhelmed (27, 54, 96). Heat-related increases in emergency admissions are most apparent for particular outcomes, including renal and respiratory disease, particularly in the elderly (51). Higher temperatures are not associated with increases in admissions for cardiovascular disease (51, 76), although some effect is apparent in the United States (83). Health system factors, such as admission thresholds, may explain some of this difference. Studies so far have indicated that increases in hospital admissions during heat waves are not as severe as those seen in mortality data. One reason for this may be that people who die during heat waves die suddenly or do not reach the attention of the medical services. The latter hypothesis has implications for health protection measures.

METHODS OF CHARACTERIZING THE TEMPERATURE-MORTALITY RELATIONSHIP

Assessment of Heat Wave Effects

Heat waves have a greater impact on mortality than shown in the reported number of deaths or cases certified as classic heat illness. **Figure 1** illustrates the peak in all-cause mortality associated with an acute heat episode. Heat episode analyses are traditionally used to investigate health—usually mortality—during specific heat wave events (39, 48, 60, 95, 100). These describe mortality counts or rates during a heat wave and are compared with a baseline usually derived from the same

F21-1172

151 of 282

time period in surrounding years. The period of interest should differ from the comparison period only with regard to a heat wave occurrence, and so any general trends in the baseline mortality series need to be avoided or allowed for. An alternative is to create the expected values using a regression model with explicit control for season and other time-varying confounders (see below).

Assessment of Heat Effects

Time-series regression. Regression models of time-series data are often used to quantify the general heat-mortality relationship observed throughout the summer (8, 14, 41, 101). The general principle of a time-series regression approach involves assessment of any short-term associations between regular measurements of the health outcome and the exposure (e.g., daily death counts and daily temperature over a number of years). The methods used are closely related to those developed for air pollution epidemiology (40, 81). Unlike air pollution, however, the effects of temperature on mortality cannot be assumed to follow a general linear form. In populations with a temperate climate, a general U- or V-shaped relationship exists between daily mortality counts and temperature, with deaths increasing as temperatures fall, but also as temperatures rise above population-specific threshold values (14).

Multiple lag times are associated with temperature exposures, especially in relation to cold weather, but heat effects may also be delayed by up to a few days. The effects of exposure on multiple days, including any negative risks arising from mortality displacement (see below) may be modeled using a distributed lag curve (7, 8, 30, 99). The use of cross-basis functions has also recently been proposed to allow for flexible modeling of changes in coefficients with changing lags (3). The key issue with the time-series design is the optimal control for confounding by season and other causes of fluctuations in mortality over time.

Case-crossover study. If individual-level mortality records are available, an increasingly common alternative to time-series regression is the case-crossover design, in which the day of death of each individual is considered a case, and proximate days (e.g., $+/-7$ days) are controls (89). The analytic approach is then akin to that for a matched case-control study. This approach circumvents the need for seasonal control but can be subject to other biases, especially in relation to the choice of controls (58). The method is being increasingly refined as knowledge accumulates about potential biases, and investigators have recently favored a time-stratified design in which control days are chosen from time strata *a priori* (4, 57). Such designs are a special case of the general case-series approach (22). An important consideration in both time-series and case-crossover studies is serial correlation, the nonindependence of consecutive days.

Effect Modification

In addition to the approaches above, other designs have also been used to assess effect modification of the heat-mortality relationship. The case-only approach has been used to quantify the modification effect of several risk factors but does not give an indication of the overall effect (2, 82). Several case-control studies have also been undertaken on heat wave events in Chicago (85) and Paris (94). These studies used live controls and therefore may also be estimating factors that determine the risk of death per se, rather than the determinants of a heat-related death. Stratifying daily mortality series by subgroup can also be used to investigate effect modification (26, 31). However, the number of subgroups that can be investigated using this method is normally quite limited.

Mortality Displacement

A key consideration about the correct public health interpretation of the heat effect estimates is the extent to which heat deaths occur

in already frail individuals whose death may be hastened by the heat exposure by only a matter of days—so-called short-term mortality displacement or early harvesting. Studies using heat episode analysis rarely consider post-heat wave periods to assess whether excesses in mortality during a heat wave are followed by subsequent deficits in expected deaths, consistent with a harvesting process. Recent studies using penalized splines to model the mortality pattern during the Paris (56) and Chicago (37) heat waves suggested little evidence of harvesting during these very extreme heat wave events. Assessment of a harvesting process during a heat wave is complicated by the fact that any negative risk following an initial hot day may be masked by an increased risk from further heat exposure from subsequent hot days; these two opposing effects cannot be separated. Time-series studies have demonstrated some degree of mortality displacement following general heat-related deaths (7, 30).

AT: apparent temperature

Other Issues

Also of importance is the correct parameterization of the exposures measure. Mean temperature is commonly used, with control for confounding with humidity. Combined indices of temperature and humidity, such as apparent temperature (AT), are also used as a construct that characterizes the physiological experience better than just temperature alone (90). However, in a recent assessment, AT was not a better predictor of mortality than was mean temperature in two of three European cities (29).

Although some major studies of temperature and mortality have not controlled for the effects of air pollutants (7, 14), more recent work has suggested that heat effects are likely to persist even after control for air pollution, and weather risk assessments are best informed by analyses that account for PM10 and ozone in particular (73). Heat episode analyses implicitly control for the air pollution concentrations because the baseline represents the seasonal norm; however, there exists the

F21-1172

152 of 282

www.annualreviews.org • Heat Stress and Public Health

45

possibility of synergistic effects of air pollution with heat, which may be more apparent during extreme events (38).

DETERMINANTS OF HEAT-RELATED MORTALITY AND MORBIDITY

Public health interventions are often targeted at high-risk groups, and it is therefore important to identify those most vulnerable to dying in a heat wave. Published epidemiological studies are available for three main categories of exposure related to hot weather:

- individual heat wave events;
- days with temperatures above a specified heat threshold, as described in time-series regression studies; and
- classic heat stroke related to hot weather (note that a significant proportion of heat stroke is not related to heat wave days).

Risk factors can also be categorized as intrinsic (age, disability) and extrinsic (housing, behaviors); the latter vary according to location and adaptations to the local climate. The risk factors can operate at many stages along the causal chain from high ambient temperature to death (Figure 2). Effective health protection measures have important effects

on the risk of heat-related mortality at the population level and may modify the risks by age group (15). However, there is little published information as yet on the effectiveness of such measures, although it is clear that the mortality response can change dramatically from one heat wave to the next (see below) (65).

Climate itself is an important determinant of population sensitivity to temperature because it influences the level of acclimatization in individuals. The threshold temperatures for heat-related mortality effects are related to the local summer temperature. That is, the temperature above which mortality increases with increasing temperature is higher in warmer climates compared with cooler climates. The slope of the temperature-mortality response, however, is rather heterogeneous and in general not predicted by latitude, as shown by comparisons of cities within the United States (14), Europe (66), and around the world (62).

Age and Aging

Vulnerability to heat in old age occurs because of changes in the thermoregulatory system (23, 28, 93). Epidemiological studies of heat-related mortality show a larger effect in

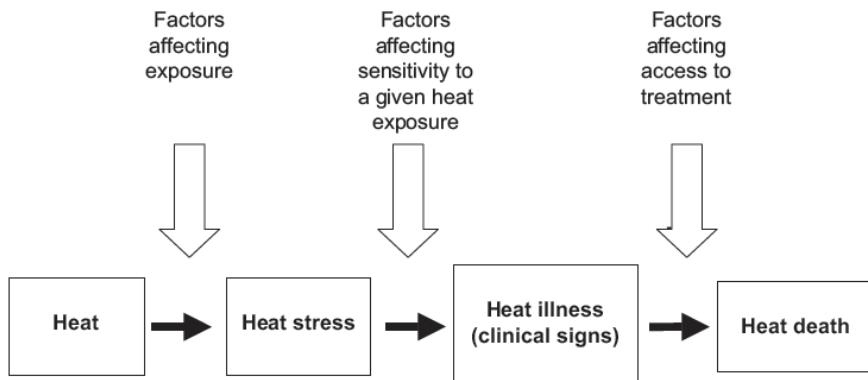


Figure 2

Points along the causal chain from heat exposure to heat death.

F21-1172

153 of 282

the elderly; the risk increases with increasing age above ~50 years old.

Children and babies also have limited ability to thermoregulate. Children are also more at risk of dehydration than are adults. Child deaths from heat stroke occurred in France during the heat waves in 2003 (78) and 2006 (21). However, time-series and episode analyses indicate no excess in mortality in children due to heat waves [except the 1981 and 1991 heat waves in Portugal (25, 74)].

Heat mortality risk varies by both age and sex. The majority of European studies have shown that women are more at risk, in both relative and absolute terms, of dying in a heat wave. There maybe some physiological reasons for an increased risk in elderly women (9, 32), but social factors are also important. In Paris, the heat risk increased for unmarried men, but not unmarried women (11). In the United States, elderly men are more at risk in heat waves than are women, and this was particularly apparent in the Chicago events (85, 100). This vulnerability may be due to the level of social isolation among elderly men (46). Evidence for the importance of social contact as protective for heat wave mortality is based on case control studies conducted in the United States (68, 85). Men are also more at risk of heatstroke mortality because they are more likely to be active in hot weather (13).

Clinical or Pathophysiological Factors

In addition to the natural patterns of aging (or senescence) on homeostatic mechanisms, several medical conditions increase vulnerability to heat stress. Epidemiological studies indicate that people with depression, cardiovascular and cerebrovascular conditions (89), and diabetes (82) need to take extra care in hot weather. The literature on pathophysiology of heat is incomplete. Many deaths that are attributed to heat are not caused by heat stroke nor are they even in persons that exhibit the clinical signs of heat stress. There

are likely several mechanisms by which a person may die during a heat wave because the environmental temperatures place extra strain on the cardiovascular system. The person becomes dehydrated, displaying increased blood viscosity and other physiological changes. In theory, any illness that compromises thermoregulation will increase the risk of heat-related death. If the exposure to heat is severe enough, even healthy people will succumb to heat stroke. Illnesses also compromise mobility, awareness, and behavior. Therefore, some conditions, such as dementia and Parkinson's disease, are also important risk factors for heat mortality. In addition, a range of common medications interfere with thermoregulation (anticholinergics interfere with sweating, diuretics can cause dehydration, etc.) (17, 19, 44).

Living in Institutions

The heat wave in 2003 caused a severe impact on elderly persons in hospital and in residential homes. The mortality rate doubled in the 75+ age group for persons living in retirement homes in France (24). In 2003, such institutions in France, Italy, and the United Kingdom were generally without air conditioning. A study in the United Kingdom also found a much higher heat risk in nursing home patients and care home patients, with lowest risk in persons living at home (31). The residents of institutions therefore represent an important high-risk group for heat wave interventions. In France, the government has since recommended that homes for the elderly have at least one cool room (63). In the southern United States, there appears to be little heat-related mortality in care homes, except when an air conditioning system failure occurs (92). The risk of heat-related mortality is determined by level of disability (or frailty) in the nursing home setting (5); however, a study in France suggested that as medical care during the heat wave was directed toward the most vulnerable patients, the less-frail patients made the largest contribution to F21-1172

the excess mortality estimated during the heat wave (34).

Housing Characteristics and Air Conditioning

People spend the most of their time indoors. There is relatively little evidence that certain types of housing (and thermal behavior) increase vulnerability. Brick houses (with a high thermal mass), top floor apartments with no through ventilation, and closed windows are associated with an increased risk of mortality during a heat wave (67, 94).

U.S. studies indicate that air conditioning is an important protective factor for heat-related mortality (85). In Europe, there is little domestic air conditioning, although this is expected to change in the coming decades. Lack of air conditioning may explain the risk of heat stroke in poor urban elderly persons in some U.S. inner cities because the role of high energy costs and the loss of income support was an issue during the Chicago heat wave (46). Power failures are common during heat waves because of sudden increases in electricity demand (20). Some investigators suggest that widespread use of air conditioning may reduce physiological acclimatization and can therefore make people more susceptible to heat waves, but the evidence is unclear (72).

Socioeconomic Factors

Deprivation, particularly in the inner city, is an important determinant of heat wave and heat stroke mortality risk in the United States, but the evidence is less clear for European populations. The heat wave in Phoenix in 2006 was responsible for 13 heat stroke deaths, of which 11 were homeless people. Most studies of the 2003 heat wave in Europe report little or no effect of deprivation on mortality risk (6, 31, 89), although one study in Italy reported a higher risk in low-income groups (64). Individuals on low incomes are more likely to have a chronic disease or other

F21-1172

155 of 282

medical risk factors, such as obesity or mental illness, and less adequate types of housing, which will all modify the risk of heat-related mortality.

Urban Heat Islands

Urban heat islands are a factor in many cities and refer to the difference in temperatures measured inside and outside the city (70, 71). Heat islands have been particularly important in some heat wave events (53, 98). Heat islands are dynamic in time and space and are therefore hard to quantify either spatially or temporally for individual heat wave events. Although much is known about the factors of the built environment that increase temperatures, when estimating health effects within a city, confounding by housing type and socioeconomic factors becomes very important. Several studies show that mortality is more sensitive to heat in urban areas compared with rural and suburban areas. One reason for this could be that urban heat islands magnify night time temperatures. The importance of heat islands may vary, as cities in southern Europe are more adapted to heat than are those in northern Europe (31, 87). A study in Spain found that excess mortality during the August 2003 heat wave was comparable in rural villages and in the provincial capital (61).

IMPLICATIONS FOR PUBLIC HEALTH

Many public health lessons were learned following the August 2003 heat wave in western Europe (86). The key problems identified by the French government included the lack of an intervention plan for heat waves and the lack of coordination between the social services and health agencies. In addition, very few care homes ("maison de retrait") had air conditioning or other space cooling, and these buildings simply became too hot. However, the heat wave itself was so severe that it could not have been anticipated (59, 86). It is very

likely that anthropogenic climate change has doubled the likelihood of such events (91).

Many countries now have heat wave plans that dictate a range of public health interventions. Health education about prevention and identification of the first signs of heat stress is the most important public health strategy, but such campaigns must be repeated at the beginning of every summer. HHWSs use meteorological forecasts to reduce the impact of heat waves on human health (49). The challenge lies in determining at which point the weather conditions become sufficiently hazardous to human health in a given population to warrant intervention (47, 50, 69, 88). Warnings issued to the general public during a heat wave reinforce the general heat-avoidance messages. The active components of HHWSs, such as identifying and contacting high-risk individuals, vary from city to city. Intervention plans should be best suited for local needs through coordination between the local health agencies, social services, voluntary agencies, and the NWS.

The heat wave in 2006 in western Europe was associated with much less impact on mortality than that in 2003 (21). It is not straightforward to compare directly the impacts of heat waves in terms of numbers of deaths, either in different cities or in the same city over time (15, 50). Fewer heat-related deaths occurred in Chicago during the 1999 heat wave compared with the earlier 1995 event. Although some of this reduction in mortality was attributed to the successful implementation of prevention measures, such as the opening of cooling centers (75), as well as an increase in air conditioning use, there would also have been a significant increase in the general level of awareness of heat waves impacts and public health messages.

Another important issue that has been identified is lack of health surveillance for heat wave mortality. In 2003, surveillance of general mortality on a daily basis was not available, and systems could not detect an increase in deaths in the elderly from nonspecific causes (1). Some countries consequently

Table 2 Recent trends, assessment of human influence on the trend, and projections for extreme weather events for which there is an observed late-twentieth-century trend [Fourth Assessment Report of the Intergovernmental Panel on Climate Change (35)]

Phenomenon and direction of trend	Likelihood that trend occurred in late twentieth century (typically post-1960)	Likelihood of a human contribution to observed trend	Likelihood of future trends based on projections for twenty-first century
Warmer and fewer cold days and nights over most land areas	Very likely	Likely	Virtually certain
Warmer and more frequent hot days and nights over most land areas	Very likely	Likely (nights)	Virtually certain
Warm spells/heat waves. Frequency increases over most land areas	Likely	More likely than not	Very likely
Heavy precipitation events. Frequency (or proportion of total rainfall from heavy falls) increases over most areas	Likely	More likely than not	Very likely
Area affected by droughts increases	Likely in many regions since 1970s	More likely than not	Likely
Intense tropical cyclone activity increases	Likely in some regions since 1970	More likely than not	Likely
Increased incidence of extreme high sea level (excludes tsunamis)	Likely	More likely than not	Likely

now use other indicators that are available on a 24-hour basis, such as a telephone advice line in the United Kingdom, to support decision making for heat wave alerts (55).

CONCLUSION

Heat health protection strategies require further research to identify those that are truly effective in reducing mortality due to heat waves. The limited epidemiological evidence suggests that targeting high-risk groups may not be the most effective strategy but that sustainable improvements in the health of the vulnerable elderly are required.

Global climate change is likely to be accompanied by an increase in the frequency and intensity of heat waves (**Table 2**). In the long term, as the climate changes, populations are likely to become less sensitive to temperature extremes owing to improvements in the underlying health of the population (12, 18). Conversely, populations are aging, and the number of elderly people susceptible to temperature extremes will increase. There is also a trend toward more energy-intensive buildings that need to be artificially cooled (80). Thus we must also build houses and cities that are cooler as well as more sustainable and energy efficient.

SUMMARY POINTS

1. A range of epidemiological study designs are used to quantify the effect of temperature on mortality and explore effect modification, including time-series regression; episode analyses; and case-control, case-only, and case-crossover methods.
2. Mortality due to heat waves is most pronounced among the elderly, but other groups are also at risk, including adults with chronic disease and children. The epidemiological evidence base needs to be reviewed regularly to determine the advice on targeting certain groups with prevention strategies.
3. A range of measures, including health advice and weather-based alerts, are used to prevent heat wave deaths, but there is no clear evidence about the most effective measures in community settings, particularly in targeting the very vulnerable.
4. Climate change will increase the frequency and the intensity of heat waves, and a range of measures—including improvements to housing, management of chronic diseases, and institutional care of the elderly and the vulnerable—will need to be developed to reduce future impacts of heat.

DISCLOSURE STATEMENT

The authors are not aware of any biases that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGEMENTS

The authors thank Bettina Menne and Franziska Matthies, and all participants in the EUROHEAT network. The authors' work was funded in part by the European Commission DG SANCO (agreement number 2004322).

LITERATURE CITED

1. Abenheimp L. 2005. Lessons from the heat wave epidemic in France (Summer 2003). See Ref. 45, pp. 161–66

F21-1172
157 of 282

2. Armstrong B. 2003. Fixed factors that modify the effects of time-varying factors: applying the case only approach. *Epidemiology* 14:467–72
3. Armstrong B. 2006. Models for the relationship between ambient temperature and daily mortality. *Epidemiology* 17:624–31
4. Basu R, Dominici F, Samet JM. 2005. Temperature and mortality among the elderly in the United States: a comparison of epidemiological methods. *Epidemiology* 16:58–66
5. Belmin J, Auffray J-C, Berbezier C, Boirin P, Mercier S, et al. 2007. Level of dependency: a simple marker associated with mortality during the 2003 heatwave among French dependent elderly people living in the community or in institutions. *Age Ageing* 36:298–303
6. Borrell C, Marí-Dell'Olmo M, Rodríguez-Sans M, García-Olalla P, Caylà JA, et al. 2006. Socio-economic position and excess mortality during the heat wave of 2003 in Barcelona. *Eur. J. Epidemiol.* 21:633–40
7. Braga ALF, Zanobetti A, Schwartz J. 2001. The time course of weather-related deaths. *Epidemiology* 12:662–67
8. Braga ALF, Zanobetti A, Schwartz J. 2002. The effect of weather on respiratory and cardiovascular deaths in 12 US cities. *Environ. Health Perspect.* 110:859–63
9. Burse RL. 1979. Sex differences in human thermoregulatory response to heat and cold stress. *Hum. Factors* 21:687–99
10. Cajoto VI, Peromingo JA, Vicdego GV, Leira JS, Frojan S. 2005. Health impact of 2003 heat wave at Hospital de Riveira (A Coruña). *An. Med. Int.* 22:15–20
11. Canoui-Poitrine F, Cadot E, Spira A, Groupe Rég. Canicule. 2006. Excess deaths during the August 2003 heatwave in Paris, France. *Rev. Epidemiol. Sante Publique* 54:127–35
12. Carson C, Hajat S, Armstrong B, Wilkinson P. 2006. Declining vulnerability to temperature-related mortality in London over the twentieth century. *Am. J. Epidemiol.* 164:77–84
13. Cent. Dis. Control (CDC). 2006. Heat-related deaths—United States, 1999–2003. *MMWR* 55:769–98
14. Curriero FC, Heiner KS, Samet JM, Zeger SL, Strug L, Patz JA. 2002. Temperature and mortality in 11 cities of the Eastern United States. *Am. J. Epidemiol.* 155:80–87
15. Delarozière JC, Sanmarco JL. 2004. Excess mortality in people over 65 years old during summer heat waves in Marseille. Comparison before and after preventive campaign. *Presse Med.* 33:13–16
16. Dematte JE, O'Mara K, Buescher J, Whitney CG, Forsythe S, et al. 1998. Near-fatal heat stroke during the 1995 heat wave in Chicago. *Ann. Intern. Med.* 129:173–81
17. Dixit SN, Bushara KO, Brooks BR. 1997. Epidemic heat stroke in a midwest community: risk factors, neurological complications and sequelae. *Wis. Med. J.* 96:39–41
18. Donaldson GC, Keatinge WR, Nayha S. 2003. Changes in summer temperature and heat-related mortality since 1971 in North Carolina, South Finland, and Southeast England. *Environ. Res.* 91:1–7
19. Ellis F. 1976. Heat wave deaths and drugs affecting temperature regulation. *Br. Med. J.* 2:474
20. Emerg. Manag. Aust. (EMA). 2002. EMA Disaster Events Data Tracking System (EMA-Track) <http://www.ema.gov.au/>
21. Empereur-Bissonnet P, Salines G, Bérat B, Caillère N, Josseran L. 2006. Heatwave in France, July 2006: 112 excess deaths so far attributed to the heat. *Eurosurveillance* 11:E060803.3
22. Farrington CP, Whitaker HJ. 2006. Semiparametric analysis of case series data. *Appl. Stat.* 55:553–94

23. Flynn A, McGreevy C, Mulkerrin EC. 2005. Why do older patients die in a heatwave? *QJM* 98:227-29
24. Fouillet A, Rey G, Laurent F, Pavillon G, Bellec S, et al. 2006. Excess mortality related to the August 2003 heat wave in France. *Int. Arch. Occup. Environ. Health* 80:16-24
25. Garcia AC, Nogueira PJ, Falcao JM. 1999. Onda de calor de Junho de 1981 em Portugal: efeitos na mortalidade. *Rev. Port. Saude Pública* 1:67-77
26. Gouveia N, Hajat S, Armstrong B. 2003. Socio-economic differentials in the temperature-mortality relationship in São Paulo, Brazil. *Int. J. Epidemiol.* 32:390-97
27. Gremy I, Lefranc A, Pepin P. 2004. Conséquences sanitaire de la canicule d'août 2003 en Ile-de-France [Impact of the August 2003 heat wave: sanitary consequences in Ile-de-France]. *Rev. Epidemiol. Sante Publique* 52:93-98
28. Grundy E. 2006. Ageing and vulnerable elderly people: European perspectives. *Ageing Soc.* 26:105-34
29. Hajat S, Armstrong B, Baccini M, Biggeri A, Bisanti L, et al. 2006. Impact of high temperatures on mortality: Is there an added "heat wave" effect? *Epidemiology* 17:632-38
30. Hajat S, Armstrong B, Gouveia N, Wilkinson P. 2005. Mortality displacement of heat-related deaths: a comparison of Delhi, São Paulo and London. *Epidemiology* 16:613-20
31. Hajat S, Kovats RS, Lachowycz K. 2007. Heat-related and cold-related deaths in England and Wales: Who is at risk? *Occup. Environ. Med.* 64:93-100
32. Havenith G. 2005. Temperature, heat balance, and climatic stress. See Ref. 45, pp. 70-80
33. Hémon D, Jouglard E. 2004. La canicule du mois d'août 2003 en France. *Rev. Epidemiol. Sante Publique* 52:3-5
34. Holstein J, Canoui-Poitrine F, Neumann A, Lepage E, Spira A. 2005. Were less disabled patients the most affected by 2003 heatwave in nursing homes in Paris, France? *J. Public Health* 27:359-65
35. Intergov. Panel Clim. Change (IPCC). 2007. *Climate Change 2007: The Physical Science Basis. Contribution of Working Group I to the Fourth Assessment Report of the Intergovernmental Panel on Climate Change*. Cambridge, UK/New York: Cambridge Univ. Press
36. Johnson H, Kovats RS, McGregor GR, Stedman JR, Gibbs M, et al. 2005. The impact of the 2003 heatwave on mortality and hospital admissions in England. *Health Stat. Q.* 2005:6-11
37. Kaiser R, Le Tertre A, Schwartz J, Gotway C, Daley R, Rubin CH. 2007. The effect of the 1995 heatwave in Chicago on all-cause and cause-specific mortality. *Am. J. Public Health* 97:S158-62
38. Kosatsky T, Litvak E, Analitis A, Katsouyanni K, Menne B, Penteli X. 2007. The independent and synergistic short-term effects of temperature and air pollution on health: review of the evidence. In *Preparedness and Response to Heat-Waves in Europe: From Evidence to Action*, ed. B Menne, F Matthies. Copenhagen: World Health Org. In press
39. Katsouyanni K, Trichopoulos D, Zavitsanos X, Touloumi G. 1988. The 1987 Athens heatwave. *Lancet* 2:573
40. Katsouyanni K, Zmirou D, Spix C, Sunyer J, Schouten JP, et al. 1995. Short-term effects of air pollution on health: a European approach using epidemiological time-series data. The APHEA project: background, objectives, design. *Eur. Respir. J.* 8:1030-38
41. Keatinge WR, Donaldson GC, Cordioli E, Martinelli M, Kunst AE, et al. 2000. Heat-related mortality in warm and cold regions of Europe: an observational study. *Br. Med. J.* 321:670-73
42. Kilbourne EM. 1997. Heat waves and hot environments. In *The Public Health Consequences of Disasters*, ed. E Noji, pp. 245-69. New York: Oxford Univ. Press

F21-1172

159 of 282

43. Kilbourne EM. 1992. Illness due to thermal extremes. In *Public Health and Preventative Medicine*, ed. JM Last, RB Wallace, pp. 491–501. Norwalk, CT: Appleton Lang
44. Kilbourne EM, Choi K, Jones TS, Thacker SB. 1982. Risk factors for heatstroke. A case-control study. *JAMA* 247:3332–36
45. Kirch W, Menne B, Bertollini R, eds. 2005. *Extreme Weather Events and Public Health Responses*. Berlin: Springer-Verlag
46. Klinenberg E. 2002. *Heat Wave: A Social Autopsy of Disaster in Chicago*. Chicago: Univ. Chicago Press
47. Koppe C, Jendritzky G, Kovats RS, Menne B. 2003. *Heatwaves: Impacts and Responses*. Copenhagen: WHO
48. Kosatsky T. 2005. The 2003 European heat waves. *Eurosurveillance* 10:148–49
49. Kosatsky T, King N, Henry B. 2005. How Toronto and Montreal (Canada) respond to heat. See Ref. 45, pp. 167–71
50. Kovats RS, Ebi KL. 2006. Heatwaves and public health in Europe. *Eur. J. Public Health* 16:592–99
51. Kovats RS, Hajat S, Wilkinson P. 2004. Contrasting patterns of mortality and hospital admissions during heatwaves in London, UK. *Occup. Environ. Med.* 61:893–98
52. Kovats RS, Jendritzky G. 2005. Heat waves and human health. In *Climate Change and Adaptation Strategies for Human Health*, ed. B Menne, KL Ebi, pp. 63–98. Darmstadt: Steinkopff Verlag
53. Kunkel KE, Changnon SA, Reinke BC, Arritt RW. 1996. The July 1995 heatwave in the midwest: a climatic perspective of critical weather factors. *Bull. Am. Meteorol. Soc.* 77:1507–18
54. Lecomte D, de Penanster D. 2004. People living in Paris, dead during the August 2003 heatwave and examined in Medicolegal Institute. *Bull. Acad. Natl. Med.* 188:459–69
55. Leonardi G, Hajat S, Kovats RS, Smith GE, Cooper D, Gerard E. 2006. Syndromic surveillance use to detect the early effects of heat-waves: an analysis of NHS Direct data in England. *Prev. Med.* 51:194–201
56. Le Tertre A, Lefranc A, Eilstein D, Declercq C, Medina S, et al. 2006. Impact of 2003 heat wave on all cause mortality in 9 French cities. *Epidemiology* 17:75–79
57. Levy D, Lumley T, Sheppard L, Kaufman J, Checkoway H. 2001. Referent selection in case-crossover analyses of acute health effects of air pollution. *Epidemiology* 12:186–92
58. Lumley T, Levy D. 2000. Bias in the case-crossover design: implications for studies of air pollution. *Environmetrics* 11:689–704
59. Luterbacher J, Dietrich D, Xoplaki E, Grosjean M, Wanner H. 2004. European seasonal and annual temperature variability, trends and extremes since 1500. *Microb. Ecol.* 303:1499–503
60. MacFarlane A, Waller RE. 1976. Short-term increases in mortality during heatwaves. *Nature* 264:434–36
61. Martínez-Navarro F, Simón-Soria F, López-Abente G. 2004. Valoracion del impacto de la ola de calor del verano de 2003 sobre la mortalidad. Evaluation of the impact of the heat wave in the summer of 2003 on mortality. *Gac-Sanit.* 18:250–58
62. McMichael A, Wilkinson P, Kovats RS, Pattenden S, Hajat S, et al. 2007. International study of temperature, heat and urban mortality: the ‘Isotherm’ Project. *Int. J. Epidemiol.* Manuscript submitted
63. Michelon T, Magne P, Simon-Delaville F. 2005. Lessons of the 2003 heatwave in France and action taken to limit the effects of future heat waves. See Ref. 45, pp. 131–40
64. Michelozzi P, de Donato F, Bisanti L, Russo A, Cadum E, et al. 2005. The impact of the summer 2003 heatwaves on mortality in four Italian cities. *Eurosurveillance* 10:161–65

F21-1172

160 of 282

www.annualreviews.org • Heat Stress and Public Health

53

65. Michelozzi P, DeSario M, Accetta G, DeDonato F, Kirchmayer U, et al. 2006. Temperature and summer mortality: geographical and temporal variations in four Italian cities. *J. Epidemiol. Community Health* 60:417–23
66. Michelozzi P, Kirchmayer U, Katsouyanni K, Biggeri A, McGregor GR, et al. 2007. Assessment and prevention of acute health effects of weather conditions in Europe, the PHEWE project: background, objectives, design. *Environ. Health.* In press, doi:10.1186/1476-069X-6-12
67. Mirchandani HG, McDonald G, Hood IC, Fonseca C. 1996. Heat-related deaths in Philadelphia—1993. *Am. J. Forensic Med. Pathol.* 17:106–8
68. Naughton MP, Henderson A, Mirabelli M, Kaiser R, Wilhelm JL, et al. 2002. Heat related mortality during a 1999 heatwave in Chicago. *Am. J. Prev. Med.* 22:221–27
69. O'Connor M, Cardinal M-E, Shaykewich J, Kosatsky T. 2007. *Heat Health Warning Workshop Rep., Oct. 5–6, 2006, Montréal, Québec, Can.* Agence de la santé et de services sociaux de Montréal
70. Oke TR. 1995. *Boundary Layer Climates.* London: Methuen
71. Oke TR. 1973. City size and the urban heat island. *Atmos. Environ.* 7:769–79
72. O'Neill M. 2003. Air conditioning and heat-related health effects. *Appl. Environ. Sci. Public Health* 1:9–12
73. O'Neill M, Hajat S, Zanobetti A, Ramirez AM, Schwartz J. 2005. Impact of control for air pollution and respiratory epidemics on the estimated associations of temperature and daily mortality. *Int. J. Biometeorol.* 50:121–29
74. Paixao E, Nogueira PJ. 2002. *Estudo de onda de calor de 1991 em Portugal: efeitos na mortalidade.* Lisbon: Obs. Nac. Saudé (ONSA)
75. Palecki MA, Changnon SA, Kunkel KE. 2001. The nature and impacts of the July 1999 heatwave in the midwestern United States: learning from the lessons of 1995. *Bull. Am. Meteorol. Soc.* 82:1353–67
76. Panagiotakos DB, Chrysohou C, Pitsavos C. 2004. Climatological variations in daily hospital admissions for acute coronary syndromes. *Int. J. Cardiol.* 94:229–33
77. Parsons KC. 2003. *Human Thermal Environments: The Effects of Hot, Moderate, and Cold Environments on Human Health, Comfort, and Performance.* London: Taylor & Francis
78. Pascal L, Nicolau J, Ledrano M. 2005. *Evaluation de l'Impact de la Vague de Chaleur de l'Été sur la Morbidité Hospitalière Infantile. Rapport d'étude.* Paris: Inst. Veille Sanitaire
79. Quayle R, Doebring F. 1981. Heat stress: a comparison of indices. *Weatherwise* 34:120–24
80. Roaf S, Crichton D, Nicol F. 2005. *Adapting Buildings and Cities to Climate Change.* London: Archit. Press
81. Samet JM, Dominici F, Zeger SL, Schwartz J, Dockery DW. 2000. The National Morbidity, Mortality, and Air Pollution Study. Part I: Methods and methodologic issues. *Res. Rep. Health Eff. Inst. Rep.* 94: 5–14
82. Schwartz J. 2005. Who is sensitive to extremes of temperature? A case-only analysis. *Epidemiology* 16:67–72
83. Schwartz J, Samet JM, Patz JA. 2004. The effects of temperature and humidity on hospital admissions for heart disease. *Epidemiology* 15:755–61
84. Semenza JC, McCullough JE, Flanders WD, McGeehin MA, Lumpkin JR. 1999. Excess hospital admissions during July 1995 heat wave in Chicago. *Am. J. Prev. Med.* 16:269–77
85. Semenza JC, Rubin CH, Falter KH, Selanikio JD, Flanders WD, et al. 1996. Heat-related deaths during the July 1995 heat wave in Chicago. *N. Engl. J. Med.* 335:84–90
86. Sénat. 2004. *La France et les Français face à la canicule: les leçons d'une crise. Rapport d'information no. 195 (2003–2004) de Mme Letard, MM Flandre, S Lepeletier, fait au nom de la mission commune d'information du Sénat, déposé le 3 Février 2004.* Paris, Fr.
F21-1172
161 of 282

87. Sheridan S. 2003. Heat, mortality and level of urbanisation. *Clim. Res.* 24:255–65
88. Smoyer-Tomic KE, Rainham DGC. 2001. Beating the heat: development and evaluation of a Canadian Hot Weather Health Response plan. *Environ. Health Perspect.* 109:1241–47
89. Stafoggia M, Forastiere F, Agostini D, Biggeri A, Bisanti L, et al. 2006. Vulnerability to heat-related mortality: a multi-city population based case-crossover analysis. *Epidemiology* 17:315–23
90. Steadman RG. 1984. A universal scale of apparent temperature. *J. Clim. Appl. Meteorol.* 23:1674–87
91. Stott PA, Stone DA, Allen MR. 2004. Human contribution to the European heatwave of 2003. *Nature* 432:610–14
92. Sullivan-Bolyai JZ, Lumish RM, Smith EW, Howell JT, Bregman DJ, et al. 1979. Hyperpyrexia due to air-conditioning failure in a nursing home. *Public Health Rep.* 94:466–70
93. Thomas ND, Soliman H. 2002. Preventable tragedies—heat disaster and the elderly. *J. Gerontol. Soc. Work* 38:53–66
94. Vandentorren S, Bretin P, Zeghnoun A, Mandereau-Bruno L, Croisier A, et al. 2006. August 2003 heat wave in France: risk factors for death of elderly people living at home. *Eur. J. Public Health* 16:583–91
95. Vandentorren S, Suzan F, Medina S, Pascal M, Maulpoix A, et al. 2004. Mortality in 13 French cities during the August 2003 heat wave. *Am. J. Public Health* 94:1518–20
96. Vanhems P, Gambotti L, Fabry J. 2003. Excess rate of in-hospital death in Lyons, France, during the August 2003 heat wave. *N. Engl. J. Med.* 348:2077–78
97. Wallace RF, Kriebel D, Punnett L, Wegmann DH, Amoroso PJ. 2007. Prior heat illness hospitalization and risk of early death. *Environ. Res.* 104:290–95
98. Watkins R, Palmer J, Kolkotroni M, Littlefair P. 2002. The London Heat Island—surface and air temperature measurements in a park and street gorges. *ASHRAE Trans.* 108:419–27
99. Welty LJ, Zeger S. 2005. Are the acute effects of particulate matter on mortality in the National Morbidity, Mortality and Air Pollution Study the result of inadequate control for weather and season? A sensitivity analysis using flexible distributed lag models. *Am. J. Epidemiol.* 162:80–88
100. Whitman S, Good G, Donoghue ER, Benbow N, Shou W, Mou S. 1997. Mortality in Chicago attributed to the July 1995 heat wave. *Am. J. Public Health* 87:1515–18
101. Zeger S, Irizarry R, Peng RD. 2006. On time series analysis of public health and biomedical data. *Annu. Rev. Public Health* 27:57–79

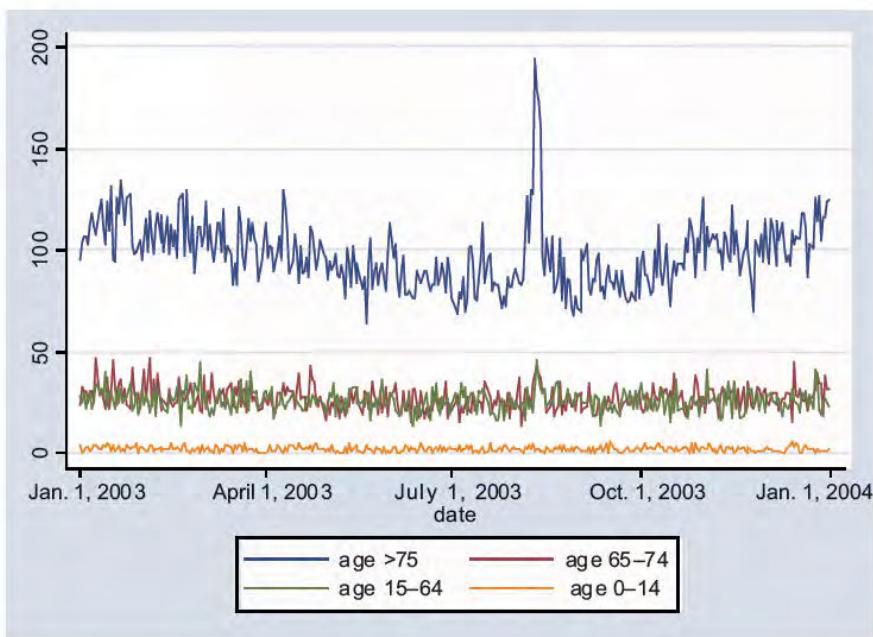


Figure 1

Daily mortality in Greater London, 2003, by age group.



Annual Review of
Public Health

Volume 29, 2008

Contents

Commentary

- Public Health Accreditation: Progress on National Accountability
Hugh H. Tilson xv

Symposium: Climate Change and Health

- Mitigating, Adapting, and Suffering: How Much of Each?
Kirk R. Smith xxiii

- Ancillary Benefits for Climate Change Mitigation and Air Pollution
Control in the World's Motor Vehicle Fleets
Michael P. Walsh 1

- Co-Benefits of Climate Mitigation and Health Protection in Energy
Systems: Scoping Methods
Kirk R. Smith and Evan Haigler 11

- Health Impact Assessment of Global Climate Change: Expanding
on Comparative Risk Assessment Approaches for Policy Making
*Jonathan Patz, Diarmid Campbell-Lendrum, Holly Gibbs,
and Rosalie Woodruff* 27

- Heat Stress and Public Health: A Critical Review
R. Sari Kovats and Shakoor Hajat 41

- Preparing the U.S. Health Community for Climate Change
Richard Jackson and Kyra Naumoff Shields 57

Epidemiology and Biostatistics

- Ecologic Studies Revisited
Jonathan Wakefield 75

- Recent Declines in Chronic Disability in the Elderly U.S. Population:
Risk Factors and Future Dynamics
Kenneth G. Manton 91

The Descriptive Epidemiology of Commonly Occurring Mental Disorders in the United States <i>Ronald C. Kessler and Philip S. Wang</i>	115
The Women's Health Initiative: Lessons Learned <i>Ross L. Prentice and Garnet L. Anderson</i>	131
U.S. Disparities in Health: Descriptions, Causes, and Mechanisms <i>Nancy E. Adler and David H. Rehkoppf</i>	235
Environmental and Occupational Health	
Industrial Food Animal Production, Antimicrobial Resistance, and Human Health <i>Ellen K. Silbergeld, Jay Graham, and Lance B. Price</i>	151
The Diffusion and Impact of Clean Indoor Air Laws <i>Michael P. Eriksen and Rebecca L. Cerak</i>	171
Ancillary Benefits for Climate Change Mitigation and Air Pollution Control in the World's Motor Vehicle Fleets <i>Michael P. Walsh</i>	1
Co-Benefits of Climate Mitigation and Health Protection in Energy Systems: Scoping Methods <i>Kirk R. Smith and Evan Haigler</i>	11
Health Impact Assessment of Global Climate Change: Expanding on Comparative Risk Assessment Approaches for Policy Making <i>Jonathan Patz, Diarmid Campbell-Lendrum, Holly Gibbs, and Rosalie Woodruff</i>	27
Heat Stress and Public Health: A Critical Review <i>R. Sari Kovats and Shakoor Hajat</i>	41
Preparing the U.S. Health Community for Climate Change <i>Richard Jackson and Kyra Naumoff Shields</i>	57
Protective Interventions to Prevent Aflatoxin-Induced Carcinogenesis in Developing Countries <i>John D. Groopman, Thomas W. Kensler, and Christopher P. Wild</i>	187
Public Health Practice	
Protective Interventions to Prevent Aflatoxin-Induced Carcinogenesis in Developing Countries <i>John D. Groopman, Thomas W. Kensler, and Christopher P. Wild</i>	187
Regionalization of Local Public Health Systems in the Era of Preparedness <i>Howard K. Koh, Loris J. Elqura, Christine M. Judge, and Michael A. Stoto</i>	205

The Effectiveness of Mass Communication to Change Public Behavior <i>Lorien C. Abroms and Edward W. Maibach</i>	219
U.S. Disparities in Health: Descriptions, Causes, and Mechanisms <i>Nancy E. Adler and David H. Rehkopf</i>	235
The Diffusion and Impact of Clean Indoor Air Laws <i>Michael P. Eriksen and Rebecca L. Cerak</i>	171
Public Health Services and Cost-Effectiveness Analysis <i>H. David Banta and G. Ardine de Wit</i>	383
Social Environment and Behavior	
Creating Healthy Food and Eating Environments: Policy and Environmental Approaches <i>Mary Story, Karen M. Kaphingst, Ramona Robinson-O'Brien, and Karen Glanz</i>	253
Why Is the Developed World Obese? <i>Sara Bleich, David Cutler, Christopher Murray, and Alyce Adams</i>	273
Global Calorie Counting: A Fitting Exercise for Obese Societies <i>Shiriki K. Kumanyika</i>	297
The Health and Cost Benefits of Work Site Health-Promotion Programs <i>Ron Z. Goetzel and Ronald J. Ozminkowski</i>	303
The Value and Challenges of Participatory Research: Strengthening Its Practice <i>Margaret Cargo and Shawna L. Mercer</i>	325
A Critical Review of Theory in Breast Cancer Screening Promotion across Cultures <i>Rena J. Pasick and Nancy J. Burke</i>	351
The Effectiveness of Mass Communication to Change Public Behavior <i>Lorien C. Abroms and Edward W. Maibach</i>	219
U.S. Disparities in Health: Descriptions, Causes, and Mechanisms <i>Nancy E. Adler and David H. Rehkopf</i>	235
Health Services	
A Critical Review of Theory in Breast Cancer Screening Promotion across Cultures <i>Rena J. Pasick and Nancy J. Burke</i>	351
Nursing Home Safety: Current Issues and Barriers to Improvement <i>Andrea Gruneir and Vincent Mor</i>	369

Public Health Services and Cost-Effectiveness Analysis <i>H. David Banta and G. Ardine de Wit</i>	383
The Impact of Health Insurance on Health <i>Helen Levy and David Meltzer</i>	399
The Role of Health Care Systems in Increased Tobacco Cessation <i>Susan J. Curry, Paula A. Keller, C. Tracy Orleans, and Michael C. Fiore</i>	411

Indexes

Cumulative Index of Contributing Authors, Volumes 20–29	429
Cumulative Index of Chapter Titles, Volumes 20–29	434

Errata

An online log of corrections to *Annual Review of Public Health* articles may be found at <http://publhealth.annualreviews.org/>

From: [Henderson, Sarah \[BCCDC\]](#)
To: [Henry, Bonnie \[EXI\]](#)
Cc: [McVea, David \[BCCDC\]](#)
Subject: RE: Briefing Minister on hot weather mortality in BC today
Date: Tuesday, July 06, 2021 1:22:50 PM
Attachments: [Ministry Brief - July 6 2021.pdf](#)

Hi Bonnie –

Thank you for facilitating the meeting today, and for your helpful comments. Based on the discussion I have updated the slide deck, attached.

Changes:

1) I have added outdoor temperatures from Abbotsford airport (YXX) to slide 7 – these temperatures aren't exactly representative of east Vancouver either, but they show the other regional extreme and the large daytime/nighttime outdoor variability compared with the indoor variability. Regardless, the key message remains that heat builds up indoors, and that indoor temperatures stay high overnight.

2) I have added the urban heat island map we created for greater Vancouver on slide 9

Please feel free to pass these along to the Ministers, as they seemed interested in this further information.

Thank you,

Sarah

Sarah B. Henderson, PhD

Scientific Director | Environmental Health Services | BCCDC

Associate Professor (Partner) | School of Population and Public Health | UBC

Office: 604.707.2449

Cell: [s.15\(1\)\(l\)](#)

[**#DifferentTogether**](#)

[**#InPlainSight**](#)

I often work outside of normal business hours because it suits me. There is no expectation that you will respond outside of your working hours.

From: Henry, Bonnie HLTH:[S.19\(1\)](#)

Sent: Tuesday, July 06, 2021 9:01 AM

To: Henderson, Sarah [BCCDC]; Gustafson, Reka [BCCDC]; Sandhu, Jat [BCCDC]

Cc: McVea, David [BCCDC]; Kosatsky, Tom [BCCDC]

Subject: RE: Briefing Minister on hot weather mortality in BC today

EXTERNAL SENDER. If you suspect this message is malicious, please forward to spam@phsa.ca and **do not** open attachments or click on links.

Thanks, Sarah, that is exactly what I was looking for. Just to bring him up to speed on what is in place and what we will be looking at. There may be opportunities to fund some of the work in the short term.

b

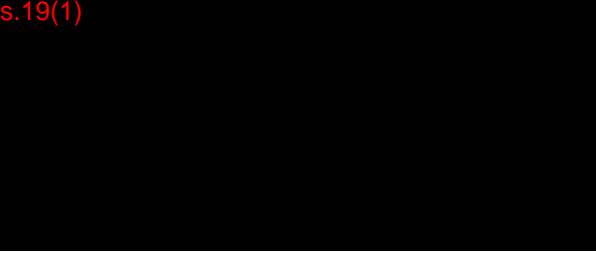
Dr Bonnie Henry

Provincial Health Officer

Office of the PHO

Ministry of Health

s.19(1)



I gratefully acknowledge that I live and work on the traditional unceded territory of the Lekwungen Peoples, specifically the Songhees and Esquimalt First Nations. Hay'sxw'qu Si'em

Warning: This email is intended only for the use of the individual or organization to whom it is addressed. It may contain information that is privileged or confidential. Any distribution, disclosure, copying, or other use by anyone else is strictly prohibited. If you have received this in error, please telephone or e-mail the sender immediately and delete the message.

From: Henderson, Sarah [BCCDC]

Sent: July 6, 2021 8:44 AM

To: Gustafson, Reka HLTH:IN ; Sandhu, Jatinder HLTH:IN

Cc: Henry, Bonnie HLTH:EX ; McVea, David [BCCDC] ; XT:HLTH Kosatsky, Tom

Subject: Briefing Minister on hot weather mortality in BC today

[EXTERNAL] This email came from an external source. Only open attachments or links that you are expecting from a known sender.

Hi Reka, Jat –

Bonnie (cc'd) asked yesterday whether David and I could brief Minister Dix and others on hot weather mortality in BC, and potentially protective measures for susceptible populations. We still don't have complete Vital Statistics data for the event that has just passed, so we will focus on what we learned about the 2009 event and what we know about the 2021 event so far.

Please find the draft of our slides here, waiting on one figure to be updated and a few other tweaks:

s.15(1)(l)



My apologies for not bringing this to your attention sooner. It's been a bit of a whirlwind to prepare, and it just clicked for me now that you might not be aware of the request. The meeting is at noon. Please feel free to call if you have any questions.

Cheers,

Sarah

Sarah B. Henderson, PhD

Scientific Director | Environmental Health Services | BCCDC

Associate Professor (Partner) | School of Population and Public Health | UBC

Office: 604.707.2449

Cell: s.15(1)(l)

#DifferentTogether

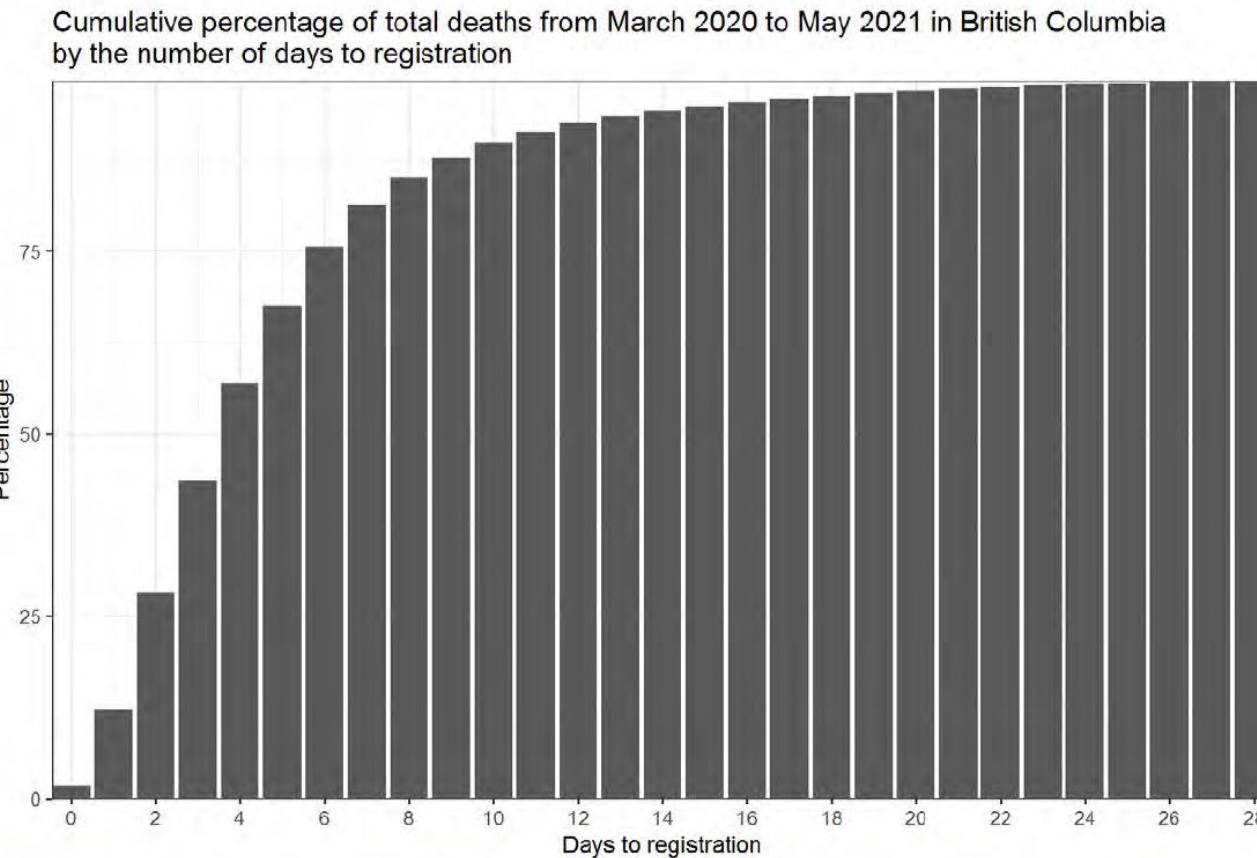
#InPlainSight

Extreme Hot Weather Events and Mortality in BC

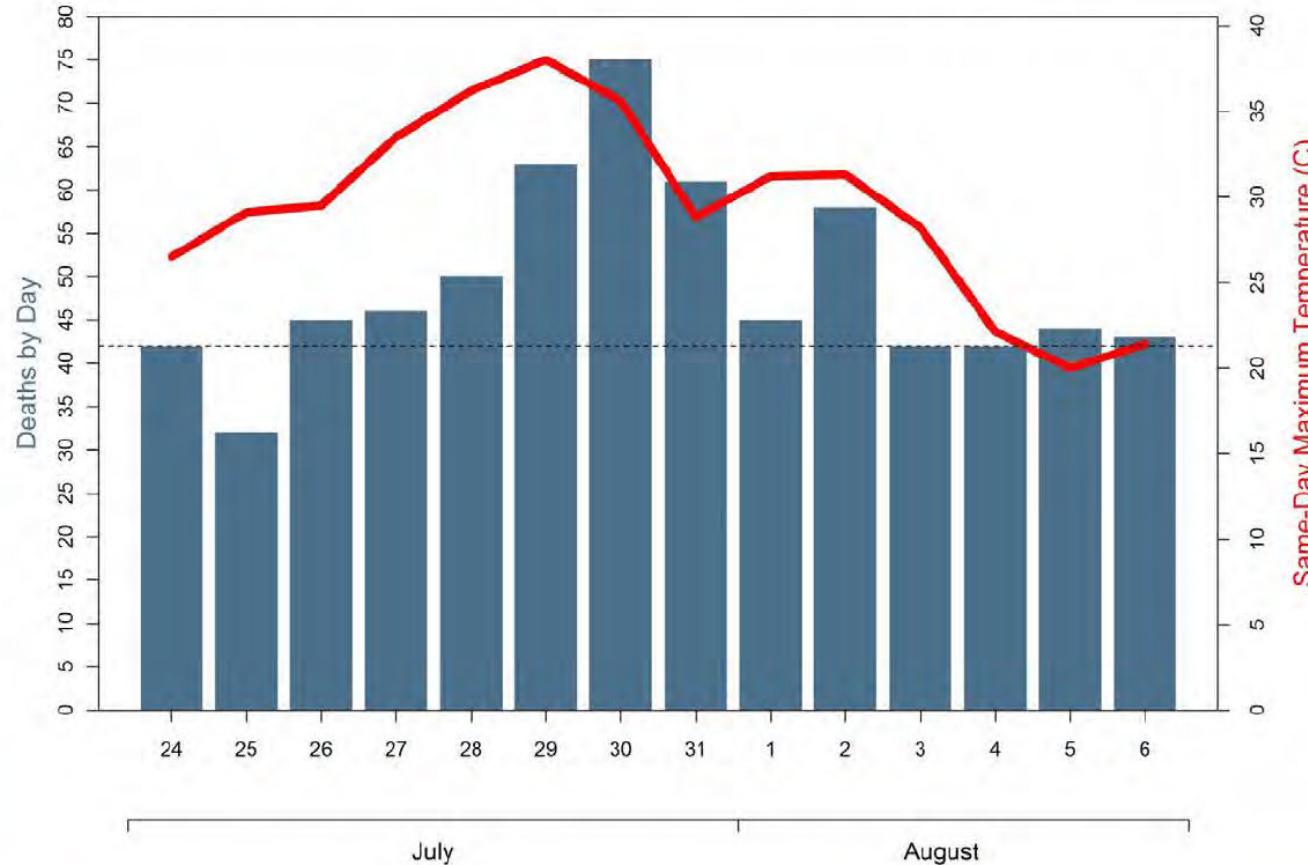
July 6, 2021

It takes time for all deaths to be registered with Vital Statistics. Deaths reported to the BC Coroners Service are a subset of all deaths.

- Data are usually 99% complete within 21 days
- Registrations happen more slowly when there are external pressures on the system (e.g. COVID-19)
- Coroners Service has reported ~500 excess investigations



The 2009 event in the Lower Mainland is our closest comparator to the provincial 2021 event.



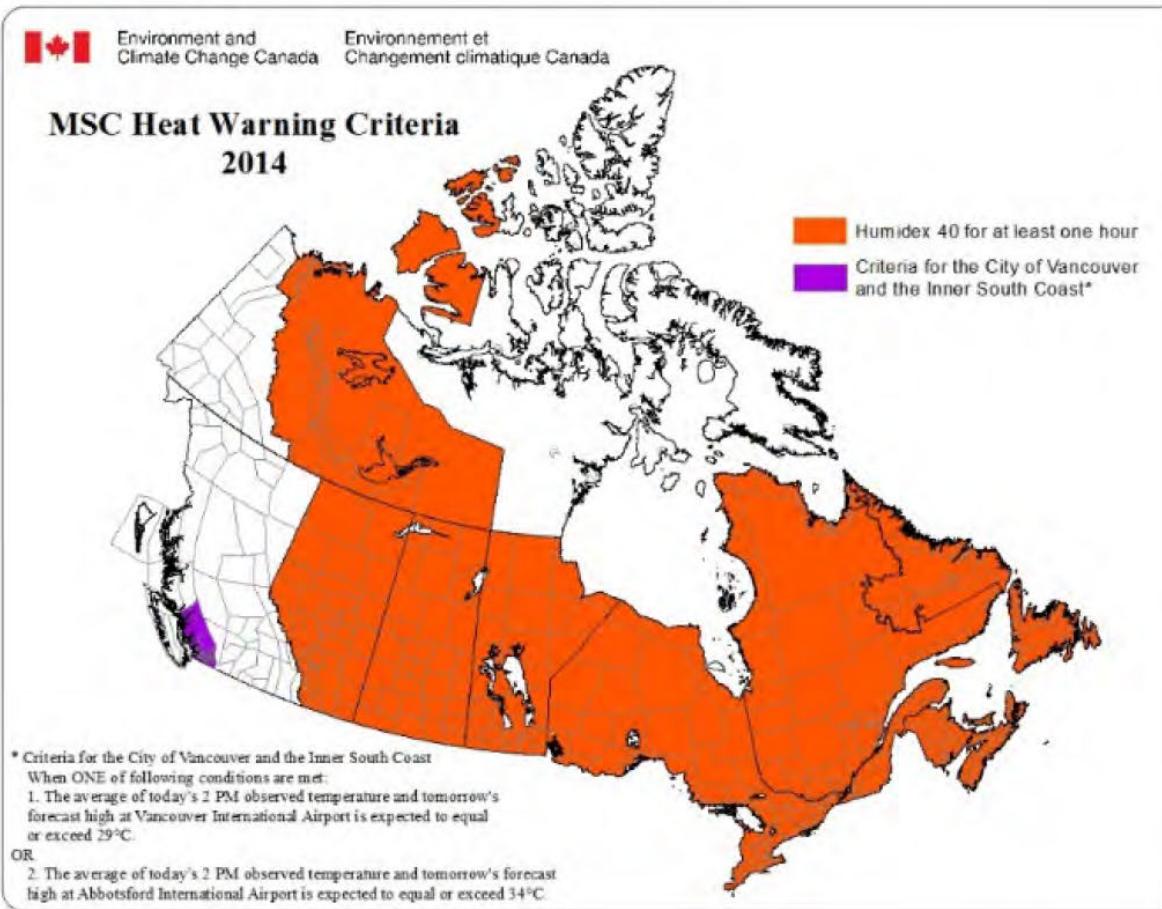
- Maximum observed at Abbotsford airport = 38°C (43°C in 2021)
- Maximum observed at Vancouver airport = 34°C (33°C in 2021)

BCCDC studied the 2009 event using available data. There were several interrelated risk factors for mortality.

1. Died at home or in the community (not hospital or LTCF)
2. Age between 65-75 (not the most elderly)
3. High neighborhood deprivation
4. Lack of surrounding neighborhood green space
5. Deaths due to cardiovascular causes
6. Deaths due to external causes (accidental poisoning from pharmaceutical and illicit drugs, suicide)

The Lower Mainland heat alert and response system (HARS) was first implemented in 2012.

- Heat not considered a risk in BC by ECCC until that time
- Data-driven risk thresholds set by BCCDC in partnership with VCH and FHA



BCCDC worked closely with ECCC to set risk-based thresholds for all of BC during the 2018 modernization project.

- BC granted FIVE alerting regions due to complex topography
- These thresholds used by ECCC to issue heat warnings during 2021 event

