

An empirical mechanistic framework for heat-related illness

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ABSTRACT: A physiologically based, mechanistic framework was developed to understand key risk factors associated with adverse health effects from heat waves. The framework consists of a number of integrated transdisciplinary modules. Environmental conditions and behavioral responses link to a physiological model, which predicts core temperature. Core temperature over time is then converted into a time-at-temperature metric. The output of the framework is a heat-related health effects index (HEI), reflecting the potential relative severity of the heat stress on health. The framework is flexible, allowing the individual models to be adapted to conditions at specific locations and to be updated as new information becomes available. Scenario analyses are easily accommodated, enabling the framework to evaluate issues such as intervention strategies and the possible effects of global climate change on heat-related illnesses. The framework and an initial set of component models were applied to conditions during the 1995 Chicago event and the results compared with published studies. For individuals, there was reasonably good agreement between HEI ratios and actual mortality risk ratios when comparing indoor versus outdoor environments. When aggregating across populations, predicted HEI ratios were significantly smaller than actual mortality ratios when comparing healthy versus compromised populations, supporting the notion that mortality may not be the best indicator of heat stress effects. Future work should include refinement of the initial models and application to other cities and heat events.

KEY WORDS: Heat stress · Chicago heat wave · Risk factors · Physiological model · Integrated modeling approach

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1. INTRODUCTION

There is increasing interest in understanding the health impacts of heat waves, in part because it has been suggested that global climate change might increase the frequency and duration of hot day occurrences (Kattenberg et al. 1996). Most previous analyses of heat events used epidemiological and statistical techniques to infer patterns in heat-related morbidity

and mortality and to identify subpopulations with increased susceptibility to heat illness.

This paper presents a modeling framework for analyzing the potential health impacts of extreme temperatures. In contrast to purely statistical methods, this proposed framework is a linked series of modules, each representing a particular component, relationship, or driver in the onset of heat-related adverse health effects. In this paper, the term 'heat stress' will refer to the ambient environmental that can impact an individual's physiology; the term 'heat strain' will refer to the physiological response, including core tempera-

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ture and adverse health impacts. The modules are linked to reflect a step-by-step, mechanistic view of the physiological process of heat strain, driven by environmental conditions and physiological predisposition. The individual models and modules within the framework are based on empirical and statistical information. The model's output is a 'health effects index' (HEI) calculated for each specific combination of exposure and physiology. Demographic information is used to calculate population-wide HEIs using weighted averages.

The objective of this framework is to provide a structured skeleton on which may be placed modeling results from multiple disciplines. This structure allows the relationships to be examined independently and refined in light of new information. In this paper, we present preliminary models for each of the key steps in the framework, derived from a variety of sources. We exercise this framework and these models on a historical event. In July 1995, the midwestern United States was struck with an extreme heat wave. In Chicago, the maximum temperatures occurred on July 13 and 14, when maximum outdoor dry bulb temperatures exceeded 100°F (37.7°C) for 2 consecutive days and remained above 80°F (26.6°C) overnight. The Centers for Disease Control and Prevention (1995) reported 465 heat-related deaths over a 17 d period. Literature published on this event includes meteorological studies of environmental factors, sociological and demographic studies of the victims, and policy studies addressing interventions such as the availability of cooling centers and a heat impact warning system (Centers for Disease Control and Prevention 1995, Japsen & Moore 1995, NOAA 1996, Voelke 1995, Changnon et al. 1996, Kunkel et al. 1996, Semenza et al. 1996). The meteorological conditions, adaptive responses, and population segmentation were developed for this scenario to calculate the resulting HEI values for different subpopulations. This is only a first effort; much additional work is required to determine which links are the most sensitive or uncertain, to refine the corresponding modules, and to test the framework and models against other historical events.

2. FRAMEWORK FOR MODELING HEAT STRAIN

The framework is shown in Fig. 1 as a linked set of modules. Inputs to the framework include ambient conditions, physical activity, and adaptive behavior. Each of these is described by a set of scenarios. Any number of scenarios can be accommodated, as long as

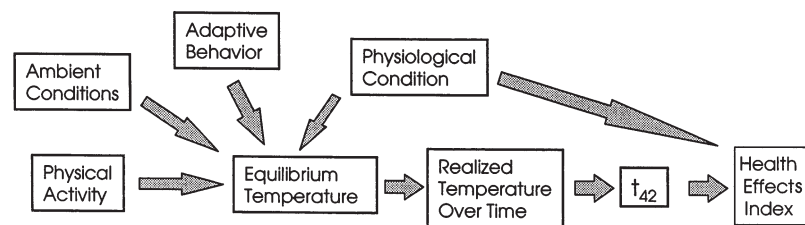


Fig. 1. Block diagram for heat strain framework and component models

they are well defined, mutually exclusive, collectively exhaustive, and can be assigned a probability of occurrence or a demographic fraction. These input modules drive a core temperature physiological module that predicts changes in body temperature over time. As heat stress and strain increase, different subpopulations may have differing abilities or options to adapt to or modify their exposure to ambient conditions (e.g., reducing activity or going to cooling centers). These adaptive behaviors change the exposure conditions and thus feed back to modify body temperature over time. Core temperature is a primary measure of heat effects, but by itself is insufficient to describe heat strain, because time is an important determinant of deleterious effects. A short time at a high core temperature has less serious potential consequences than an extended time at the same temperature. Therefore, an 'equivalent time at temperature' module converts the time-varying trajectory of core temperature to a unidimensional quantity (designated as t_{42}) representing potential heat strain. Then, the final module takes this time-at-temperature and converts it to an adverse HEI, which ranges from 0 to 100.

The subsequent sections describe the relationships used in each module. The models and relationships shown are a first effort to integrate information from various disciplines into an overall framework. The models should be refined as additional information becomes available to validate assumptions and reduce uncertainties.

2.1. Ambient conditions

Exposure of an individual to ambient environmental conditions is the starting point for the framework. Dry-bulb temperature and relative humidity on an hourly basis are used to describe the ambient exposure conditions. The ambient conditions are not necessarily the same as outdoor conditions, because people may be indoors in conditions which can either alleviate or exacerbate heat exposure. For the Chicago analyses, we defined 4 different environments: indoor unventilated; indoor ventilated (windows open); outdoor; and

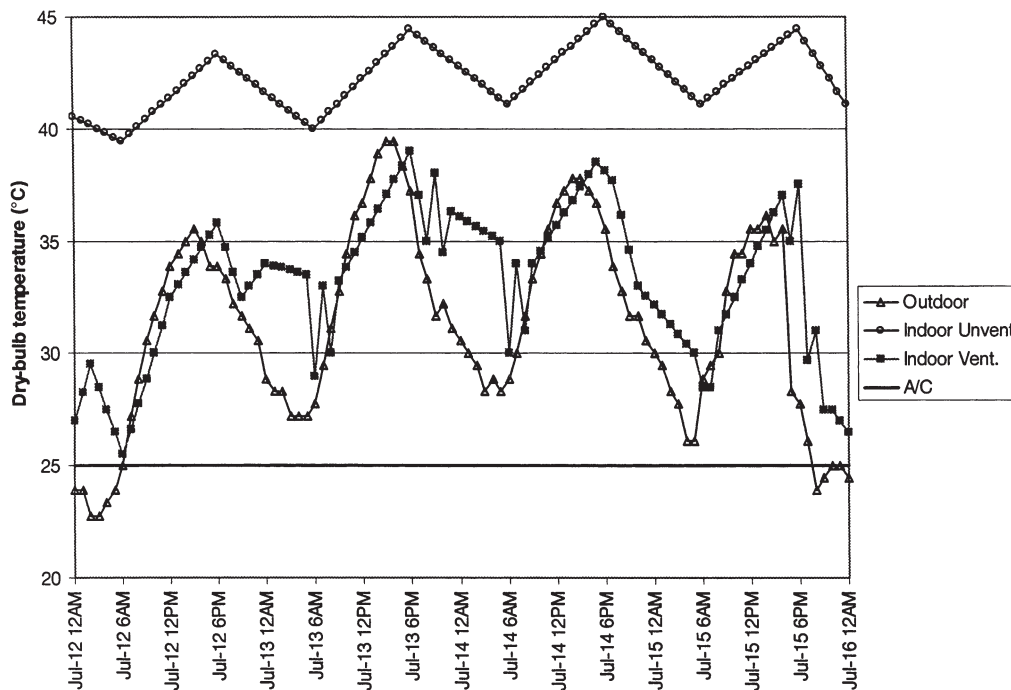


Fig. 2. Temperature profiles for different exposure levels for 1995 Chicago heat event. (Sources: Midwestern Climate Center [1996] for outdoor conditions; Huang [1996] for indoor conditions)

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:

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

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 $M_{\text{net}} = 150 \text{ W}$.

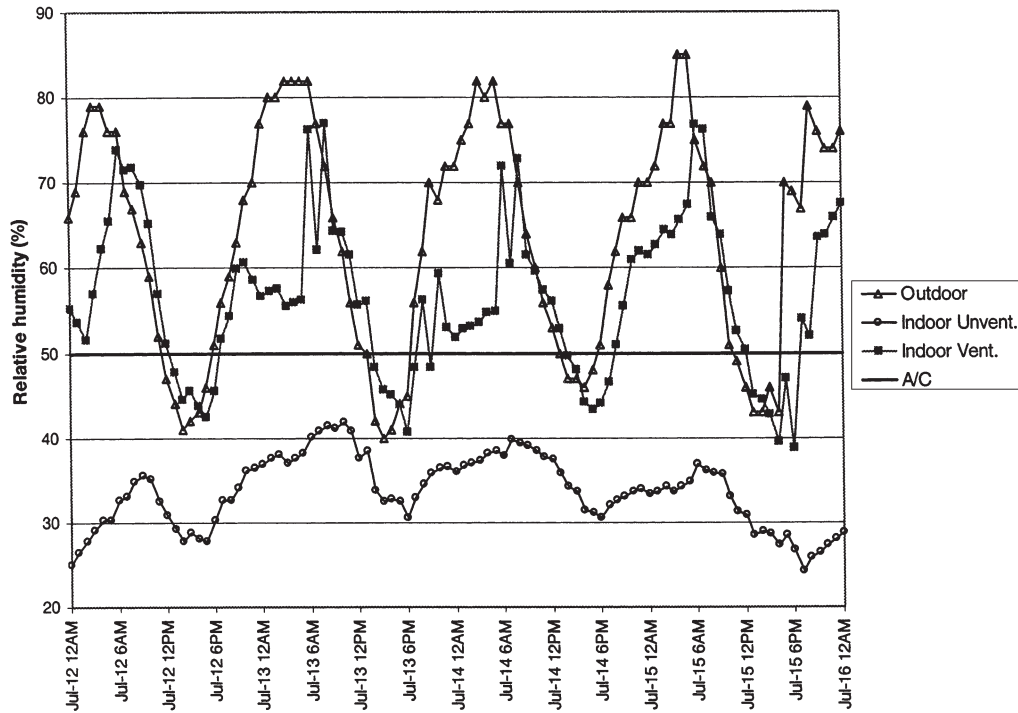


Fig. 3. Humidity profiles for different exposure levels for 1995 Chicago heat event. Outdoor conditions from Midwestern Climate Center (1996). Indoor conditions were calculated from outdoor assuming the same absolute humidity but temperature as shown in Fig. 2

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:

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

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:

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

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.

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

where $(i_m/clo) = 0.94 v_{\text{air}}^{0.28} [\text{W (mm Hg)}^{-1}]$ for shorts and short-sleeved shirt, $v_{\text{air}} = 0.5 \text{ m s}^{-1}$ assumed, ϕ_a = relative humidity, and $P_a = 0.057279 T_a^2 - 1.54435 T_a + 26.05975 \text{ mm Hg}$, 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:

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

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:

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

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

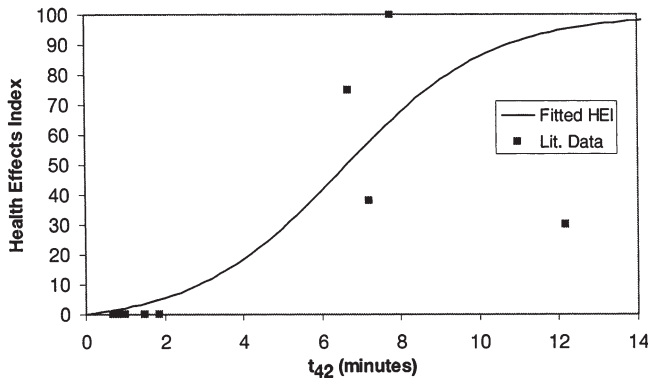


Fig. 4. Health effects index (HEI) as a function of t_{42} for healthy individuals. (■) Data points derived from Wyndham et al. (1965) and Schwartz et al. (1977). Solid curve represents fitted logistic function

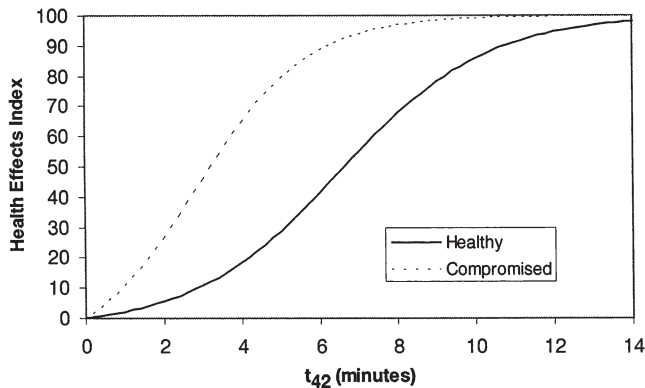


Fig. 5. Comparison of HEI as a function of t_{42} for healthy and physiologically compromised individuals

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 ($21.2 \div 5.6$) 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 ($9.6 \div 21.2$) 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 ($77.3 \div 13.8$) 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	0.61
	Go to air-conditioning	11.20	1.3	
	Open windows	4.80	9.6	
	Do not open windows	0.00	21.2	
Compromised	Air-conditioning	88.00	0.0	3.39
	Go to air-conditioning	6.60	13.8	
	Open windows	5.26	45.1	
	Do not open windows	0.14	77.3	
				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 $80 \div 5 = 16$, while the HEI ratio would be $400 \div 100 = 4$.

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

less 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-

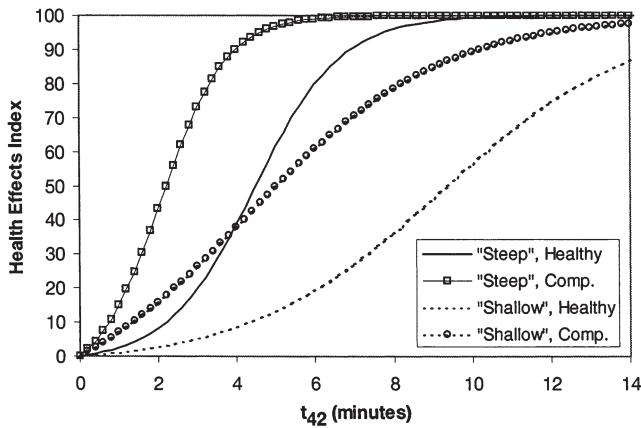


Fig. 6. Sensitivity scenarios for HEI S-curve

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			
			Indoor unventilated	Indoor ventilated	Outdoors	Air-conditioned
Healthy	HEI S-curve	Shallow	9.9	4.8	2.9	0.0
		Nominal	21.2	9.6	5.6	0.0
		Steep	45.6	17.3	8.6	0.0
Compromised	HEI S-curve	Shallow	47.8	25.8	17.8	0.0
		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.

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