

Heat Waves and Hot Environments

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Hot weather is an important determinant of human mortality. In the United States, a major heat wave can cause literally thousands of excess deaths in a given summer. Yet, for purposes of public health, the term "heat wave" is not easily defined. "Hot" is a relative term, and periods of hot weather (heat waves) vary greatly in intensity and duration. Ambient temperatures may change rapidly, and they have marked diurnal fluctuations. Their effect on human beings is not the same in different geographic areas. Moreover, the microclimates, behaviors, and preexisting medical conditions of individual human beings dramatically affect the biological consequences of macroenvironmental heat. Thus, at the present time, a precise description of conditions constituting a heat wave of public health importance eludes description. Determining the precise hot weather conditions leading to an adverse impact on public health remains an important area for scientific investigation.

Physics and Physiology of Heat

Although there may be considerable fluctuation of the temperature of the extremities and outer body surfaces of human beings, thermal homeostatic mechanisms attempt to maintain a relatively constant inner body or "core" temperature. For practical purposes, there are four physical processes involved in thermal homeostasis: (1) heat gain from metabolism, (2) heat loss from evaporation, (3) heat gain or loss from conduction and convection, and (4) gain or loss of radiant heat energy. The body gains metabolic heat from the myriad biochemical reactions that are essential to life. The body loses heat,

however, when perspiration evaporates from the skin or secretions evaporate from the respiratory epithelium. If the temperature of the body surface is different from that of substances with which it is in contact, then the body gains or loses heat by means of conduction. If the substance with which it is in contact is a fluid medium such as air or water, then conduction is hastened by the flow of fluid over body surfaces (convection). Finally, regardless of the ambient temperature, if a person is in the presence of objects or surfaces hotter or colder than the body, then body heat is gained from or lost to those objects by means of radiation (1).

Four meteorological variables significantly impact the physical processes mediating thermal homeostasis. These are (1) air temperature (measured by shaded dry-bulb thermometer), (2) humidity (measured either as the dewpoint temperature or by comparison of dry-bulb and wet-bulb temperatures), (3) air motion (wind speed), and (4) solar radiant heat energy (measured in a variety of ways). When the dry-bulb air temperature is low, metabolically generated heat is more easily lost from the body to the air via conduction/convection. As air temperature increases, convective heat loss occurs less readily until, at temperatures above body temperature, convective heat loss is no longer possible and heat may be gained from the air. High humidity limits the cooling effect of the evaporation of perspiration and secretions, and, therefore, leads to increased heat stress. Increased air speed facilitates convective heat transfer and the evaporation of sweat. Radiant heat energy adds to heat stress, independent of other variables. For example, radiant heat causes one to feel hotter in direct sunlight than in the shade, even under identical air temperature, humidity, and air speed (2-4).

Thus, the stress that hot weather places on thermal homeostasis is not a simple function of temperature alone. Other meteorological variables come into play. A number of indices have been developed for the purpose of yielding a single number expressing the combined effects of the environmental variables relevant to heat stress. One such index is the *Effective Temperature* (ET) (4). An empirical index developed during the early part of the twentieth century, it was based on actual observations of human subjects under a variety of conditions, temperature, humidity, and air movement. The effective temperature of any given combination of temperature, humidity, and air movement is the temperature of still, saturated air that would yield the same subjective thermal sensation. Because of concern that the original scale was too sensitive to the effect of humidity at low temperatures and not sensitive enough to humidity at high temperatures, a reformulated version of ET has been published (5, 6).

Another index, currently favored by U.S. meteorologists, is the *Apparent Temperature* scale of Professor R. G. Steadman. Apparent Temperature was derived mathematically and is based on physical and physiological principles (7). The *Heat Index* currently reported by the U.S. National Weather Service is based on the Apparent Temperature index of Steadman.

The *Wet-Bulb Globe Temperature* (WBGT) is frequently used to assess heat exposure in occupational situations. This index is calculated as a weighted average of dry-bulb,

wet-bulb, and globe thermometer temperatures. (The globe thermometer is a dry-bulb thermometer with the bulb located at the center of a 6-inch-diameter thin copper sphere painted matte black on the outside. Among the factors influencing the globe thermometer reading is radiant heat). The formula weighting was chosen so that WBGT values would approximate those of ET (8).

Because WBGT requires the measurements of three separate instruments, its use in the field is problematic. The *Botsball* or wet globe thermometer (basically a thermal probe located within a wet, black sphere) is a single instrument designed to take into account the combined effects of dry-bulb temperature, humidity, air movement, and radiant heat energy. The Botsball temperature reading can be related to that of the WBGT by means of a simple linear function (9).

Heat stress indices have significant limitations. Most indices involve implicit assumptions about metabolic heat production, clothing, and body habitus. Since these parameters vary among people, the predicted value of heat stress for any single person is, at best, an approximation. Certain heat indices are difficult to use because the raw data required for their calculation (e.g., globe temperatures) are not easily available. Finally, it should be recognized that the value of an index calculated from the results of meteorologic observations at a weather station may differ greatly from the values obtained if one could measure the microclimates to which individuals are exposed.

Health Effects of the Heat

Spectrum of Illness Recognized as Heat-Related

Illnesses recognizable as the direct result of exposure to prolonged periods of high environmental temperature are heatstroke, heat exhaustion, heat syncope, and heat cramps. Heat waves may also increase morbidity and mortality due to other illnesses that occur even in the absence of heat stress (e.g., myocardial infarction; see below). Burns, which result from the local application of intense heat, are not considered here.

Heatstroke occurs when perspiration and the vasomotor, hemodynamic, and adaptive behavioral responses to a heat stress are insufficient to prevent a substantial rise in core body temperature. Some authors distinguish between "classical" and "exertional" heatstroke. Classical heatstroke is said to occur largely in sedentary elderly people who are exposed to a prolonged (days to weeks) period of heat stress. Exertional heatstroke affects younger, relatively fit persons who exert themselves in a hot environment (as in a summer road race) beyond their capacity to maintain thermal equilibrium. Anhidrosis (absent or greatly diminished perspiration) is reportedly more common in the presentation of classical heatstroke (10).

Classical and exertional heatstroke are not distinct clinical entities. Rather, they represent two ends of a spectrum of circumstances under which heatstroke occurs. Anyone,

young or old, can develop heatstroke if subjected to a sufficiently prolonged and intense heat stress. Exercise increases the production of metabolic heat, predisposing persons of all ages to the development of heatstroke.

Although standardized diagnostic criteria do not exist, a patient's condition is usually designated as heatstroke when rectal temperature rises to $\geq 105^{\circ}\text{F}$ (40.6°C) as a result of high environmental temperatures. Mental status is affected, and the patient may be delirious, stuporous, or comatose. Anhidrosis may or may not be present.

Heatstroke is a medical emergency. Rapid cooling—usually by means of ice massage, ice-water bath, or special facilities for evaporative cooling—is essential to prevent permanent neurological damage or death. Further treatment is supportive, and admission to an intensive-care unit is often required. The outcome is often fatal, even with expert care. The death-to-case ratio in reported case series generally varies from 0% to 40%, averaging about 15% (11–19).

Heat exhaustion is a much less severe disease than heatstroke. Patients may complain of dizziness, weakness, or fatigue. Body temperature may be normal or slightly to moderately elevated. The cause of heat exhaustion seems to be fluid and electrolyte imbalance due to increased perspiration in response to intense heat. Therefore, treatment is directed toward the normalization of fluid and electrolyte status, and the prognosis is generally good (10).

Heat syncope refers to the sudden loss of consciousness by persons who are not acclimatized to hot weather. Consciousness returns promptly with assumption of a recumbent posture. The cause is thought to be circulatory instability due to superficial vasodilation in response to the heat, and the disorder is benign (20).

Heat cramps occur as the result of fluid and electrolyte imbalances following strenuous exercise done in the heat. Cramps tend to occur in the muscles that have been exercised most. They are common in athletes who must perform in the heat or in workers in “hot” industries. Such persons may be highly acclimatized to hot weather and therefore able to lose great quantities of fluid and electrolytes in their perspiration. Disproportionate repletion of fluid and salt leads to the imbalances (10).

Public Health Impacts: Historical Perspective

Heat-Wave Associated Mortality

Currently in the United States, in years during which no major heat wave occurs, approximately 270 deaths are recorded on death certificates as having been caused by the heat (21). A few such deaths occur during winter and the cooler months of the year, indicating that not all of them are caused directly by meteorologic conditions. However, the great majority occur during summer. In years in which prolonged periods of abnormally high temperatures (heat waves) affect large areas of the country, the number

of deaths attributed to heat may rise greatly. In 1980, when summer temperatures reached all-time high levels in much of the central and southern United States, some 1,700 deaths were diagnosed as heat-related, over six times the number expected if there had been no heat wave (21).

Such figures, however, do not reflect the full extent of the problem. In July 1980, some 5,000 deaths over the number expected occurred in the United States, far more than the 1,700 documented as having been caused by the heat (21). This finding is consistent with those of many other studies that show that only a portion of the increase in mortality during heat waves is documented on death certificates as having been caused by the heat (22). Totals of diagnoses of heat-related death have regularly underestimated heat-wave-associated excess mortality by from 22% to 100% (Table 12-1) (23, 29). (In this chapter, “excess mortality” and “excess deaths” during a heat wave refer to the difference between the number of deaths observed and the number expected based on the crude death rate in the same geographic area during some appropriate control period during which neither a heat wave nor any epidemic was present.) Mortality figures give no indication of the substantial number of nonfatal illnesses that occur as a result of the heat.

Causes of Death

Of the syndromes whose sole cause is environmental heat, heatstroke is the only one with a substantial death-to-case ratio. Thus, one might suppose that the great majority of deaths diagnosed as caused by heat represent mortality due to heatstroke. Some studies support this supposition. Henschel and others reviewed the hospital charts of 120 persons whose deaths had been certified as heat-related during the 1966 heat wave in St. Louis. They found that virtually all had a temperature of $\geq 103^{\circ}\text{F}$ (39.4°C) upon hospital admission, a fact that they interpreted as showing that most of these deaths were due to heatstroke (29). However, in another series only 60% of 57 persons hospitalized for physician-diagnosed heat-related illness and who later died met the authors' strict definition of heatstroke (26). During the heat waves in Detroit in May 1962 and June–July 1963, as well as during the June–July 1976 heat wave in Birmingham, England, no deaths were classified as having been caused by the heat, despite substantial “excess” mortality of from 17% to 32% (Table 12-1) (23, 24).

Current death-certificate coding practices at the national level in the United States make it difficult to evaluate the precise clinical diagnoses leading to heat-related death. Deaths are coded by their external cause, environmental heat (*International Classification of Diseases*, 9th Revision, Code E900), rather than by the specific illness that the heat produces.

Mortality associated with a heat wave is often so great that it appears as a sudden and substantial increase in the total number of deaths occurring in a given area (Fig. 12-1). Increases of over 50% in the crude mortality rate are not uncommon. Moreover,

Table 12-1 Numbers of Total Deaths Observed, Total Deaths Expected, Excess Deaths, and Deaths Attributed to the Heat in Specific Locations During Selected Heat Waves, 1872-1980

Location/Period	Reference	Observed Deaths	"Expected" Deaths	Excess Deaths	Excess deaths (as a percentage of expected)	Deaths Classified as Related to Heat	Deaths Related to Heat (as a percentage of excess deaths)
Birmingham, England June 24-July 8, 1976	24	491	384*	107	27.9	0	0.0
Detroit, Michigan May 12-May 18, 1962	25	429	325†	104	32.0	0	0.0
June 23-July 6, 1963	25	783	669‡	114	17.0	0	0.0
Illinois, State of July 1-July 31, 1966	26	9,617	8,469‡	1,148	13.6	80	7.0
July 1-July 31, 1936	26	9,423	6,727‡	2,696	40.1	1,193	44.3
Kansas City, Missouri July 1-July 31, 1980	27	598	362‡	236	65.2	157	66.5
Memphis, Tennessee July 1-July 31, 1980	28	817	711‡	106	14.9	83	78.3
New York, New York July 22-July 28, 1972	29	2,319	1,592‡	727	45.7	10	1.4
August 31-Sept 7, 1973	29	2,242	1,808‡	434	24.0	22	5.1
June 30-July 6, 1892	29	1,569	769‡	800	104.0	212	26.5
July 24-July 30, 1892	29	1,434	1,081‡	353	32.7	231	65.4
Aug 9-Aug 15, 1986	29	1,810	809‡	1,001	123.7	671	67.01
St. Louis, Missouri July 1-July 31, 1980	27	850	542‡	308	56.8	122	39.6
July 9-July 14, 1966	30	543	240*	303	126.2	182	60.1

*Deaths during previous 2 weeks.

†Deaths during same period the following year.

‡Deaths during same period the previous year.

§Eight times the daily average of September 1973 deaths.

¶Deaths during previous week.

**Based on deaths during previous 8 days.

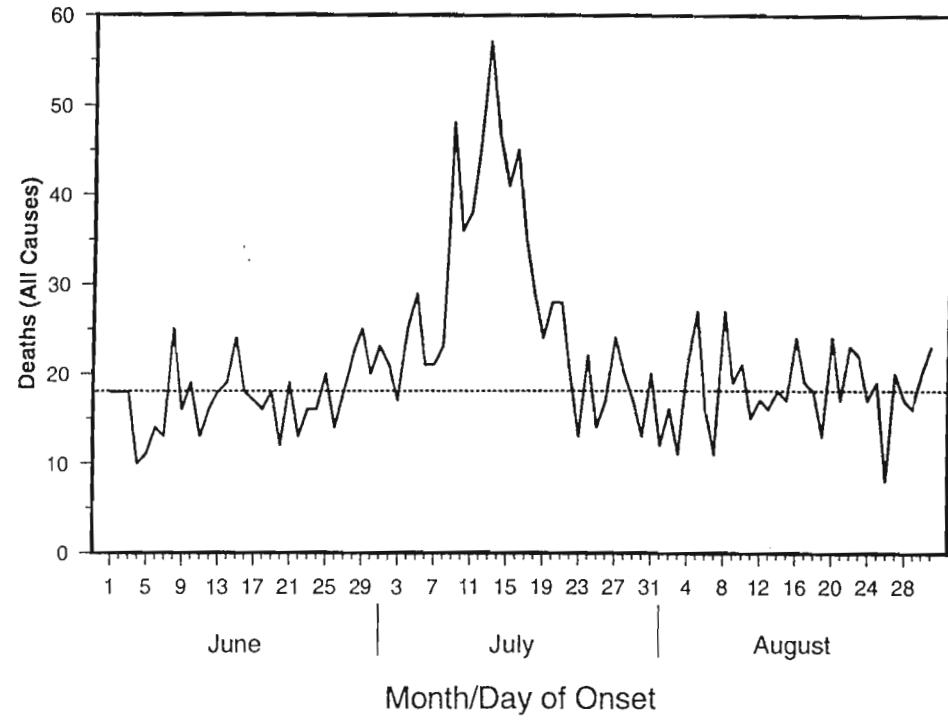


Figure 12-1. Trends in daily deaths showing the increase in numbers of deaths associated with the July 1980 heat wave, St. Louis, June 1-August 31, 1980.

despite the increased use of air-conditioning, there has been no clear and substantial decrease in the death toll taken by heat waves in recent years (Table 12-1).

Schuman and others found stroke (brain infarction or hemorrhage) to be an important cause of heat-wave-associated death; they found that deaths due to "cerebrovascular accident" rose 82% and accounted for 52% of the excess mortality caused by a heat wave in Detroit, Michigan, in 1963. During another Detroit heat wave in May of the previous year, they observed a 104% rise in deaths from stroke (26% of all heat-wave-associated deaths) (24). When Schuman studied the July 1966 heat wave in New York City, he found a far less dramatic increase of 27%, accounting for a little over 6% of an estimated 1,181 excess deaths caused by the heat. He felt, however, that the coding of stroke deaths in New York was different from that of other cities and tended to underestimate the problem (30).

Other investigators have observed that certified stroke deaths increase during severe heat (Table 12-2). However, the magnitude of the increase has been less than that noted in the Detroit studies. Increases have ranged from 25% to 55%, accounting for about 5% to 20% of heat-wave-associated mortality (25, 31). An increase in hospital admissions for persons with nonfatal strokes has also been reported (32).

Table 12-2 Percentage Increase in Selected Causes of Death and Percentage of Excess Deaths Attributable to these Causes During Heat Waves, Selected Heat Waves, United States, 1934-1983

Heat Wave Location & Period	Reference	Condition (Author's words)	Percentage Increase over Control Period	Percentage of Heat Wave Deaths Attributable to This Cause
Cerebrovascular				
Kansas State, July 1-31, 1934	32	Cerebral hemorrhage and softening	54.2	11.1
Illinois State, July 1-31, 1936	32	Cerebral hemorrhage and softening	39.2	6.0
Detroit, Michigan, May 12-18, 1962	25	Cerebrovascular accident	103.8	26.0
Detroit, Michigan, June 23-July 6, 1963	25	Cerebrovascular accident	81.9	51.8
Illinois State, July 1-31, 1966	26	Vascular lesions of central nervous system	26.3	20.1
New York, New York, July 2-15, 1966	31	Cerebrovascular accident	27.2	6.4
St. Louis, Missouri, July 9-14, 1966	30	Cerebral accident	53.3	7.0
Cardiac				
Kansas State, July 1-31, 1934	32	Diseases of the heart	22.5	12.5
Illinois State, July 1-31, 1936	32	Diseases of the heart	40.8	25.2
Detroit, Michigan, May 12-18, 1962	25	Heart disease	14.0	18.3
Detroit, Michigan, June 23-July 6, 1963	25	Heart disease	6.9	15.8
Illinois State, July 1-31, 1966	26	Arteriosclerotic heart disease	13.3	36.1
New York, New York, July 2-15, 1966	31	Arteriosclerotic heart disease	40.8	41.5
St. Louis, Missouri, July 9-14, 1966	30	Cardiovascular disease	55.4	20.0
Memphis, Tennessee, July 1-31, 1980	28	Cardiovascular*	40.0	84.9
Latum, Italy, July 1-31, 1983	41	Cardiovascular disease†	58.7	90.4
Respiratory				
Kansas State, July 1-31, 1934	32	Pneumonia, all forms	74.6	2.5
Illinois State, July 1-31, 1936	32	Pneumonia, all forms	21.9	2.0
Detroit, Michigan, May 12-18, 1962	25	Respiratory	0.0	0.0
Detroit, Michigan, June 23-July 6, 1963	25	Respiratory	42.9	5.3

Table 12-2 (Continued)

Heat Wave Location & Period	Reference	Condition (Author's words)	Percentage Increase over Control Period	Percentage of Heat Wave Deaths Attributable to This Cause
New York, New York, July 2-15, 1966	31	Respiratory	84.2	13.5
St. Louis, Missouri, July 9-14, 1966	30	Pulmonary disorders	27.8	3.3

*May include cerebrovascular deaths.

†Includes cerebrovascular deaths.

The variability in the magnitude of heat-wave-related increases in stroke mortality relative to other causes suggests that some deaths attributed to stroke are misclassified. However, there is evidence for increased coagulability of blood in heat-stressed persons, and such increased coagulability may be the biological basis for an increase in thrombotic and embolic stroke in hot weather (33, 34). Moreover, the relative consistency of the finding of excess stroke mortality during heat waves in different years and in different locations argues that the association is a real one.

The frequency of deaths attributed to heart disease also increases during heat waves (Table 12-2), mainly due to an increase in deaths attributed to ischemic heart disease. The cause-specific death rate has increased in different heat waves by amounts ranging from about 7% to 55%, accounting for approximately 10%-40% of heat-wave-associated deaths (24, 25, 27, 29-31).

A recent investigation of heat-wave-associated mortality during the July 1993 heat wave in Philadelphia showed that cardiovascular deaths increased more than 100% over baseline. Excess cardiovascular deaths outnumbered hyperthermia (heatstroke) deaths by about 5 to 1. Interestingly, the investigators found no increase in cerebrovascular (stroke) deaths. This investigation underlines the importance of heat as an exacerbating factor for persons with preexisting heart disease and reinforces the point that heatstroke is not always the principal cause of excess death during a heat wave (Centers for Disease Control & Prevention, unpublished data).

The evidence mentioned above regarding increased coagulability of blood in heat-stressed persons lends plausibility to the idea that hot weather causes an increase in deaths from ischemic heart disease, since thrombosis or embolism may exacerbate cardiac ischemia (33, 34). Moreover, the increase in cardiac deaths occurs consistently during heat waves. Thus, the link between heat and death from ischemic heart disease is strong.

It is nevertheless possible that some of the heat-wave-associated deaths attributed to stroke or ischemic heart disease are actually misclassified heatstroke deaths. This situation could arise because of problems in postmortem diagnosis. The recognition of heatstroke in a living patient who has characteristic neurologic findings and a very high body temperature presents few difficulties for the average clinician, especially if anhidrosis (greatly diminished sweating) is present. However, heatstroke can progress rapidly to death, often within few hours of the onset of symptoms. In one study of 90 fatal heatstrokes, duration of illness was less than 24 hours for 70% of patients (35). Thus, many persons who develop heatstroke die before they can be found and brought to medical attention. In the United States such relatively sudden out-of-hospital deaths are usually referred to the local coroner or medical examiner for a determination of cause of death. Frequently, however, no detailed postmortem examination of the body is done, and the determination of the cause of death is based principally on a description of the circumstances under which the body was found. Thus, the possibility exists that some heatstroke victims examined because of relatively sudden, unattended death are diagnosed as having died from other, more common causes (e.g., stroke, myocardial infarction) that can appear to be similar (36).

Postmortem temperature measurement can be useful in the diagnosis of heatstroke. During hot weather in some jurisdictions, the temperature of each body referred to the medical examiner is routinely measured, either by an investigator in the field or by the morgue attendant. A postmortem temperature of $\geq 106^{\circ}\text{F}$ measured soon after death is a useful indicator of heatstroke, because core temperature changes relatively little during the first 1 to 3 hours after death, especially if the ambient temperature is not particularly low. The possibility of false-positive and false-negative results must be considered, however, since the core temperature of a cadaver eventually approaches that of its surroundings. In time, the body of a person who died of heatstroke will cool if the ambient temperature is lower than that of the body core and can rise if ambient temperature exceeds core temperature (37–39).

Stroke and other types of cardiovascular disease taken together may account for as much as 90% of the excess mortality noted during heat waves (27, 40). Nevertheless, numbers of deaths from other causes have also been reported to rise. A clearly defined period of excess death due to respiratory causes corresponding to the July 1966 heat wave in the United States is apparent from national mortality statistics (41). In New York City, respiratory deaths rose 84% and accounted for 14% of the excess mortality attributed to the heat wave (30). However, in other heat waves respiratory deaths have not contributed substantially to excess mortality, generally accounting for 5% or less of such deaths (24, 31). Currently, there is no clear pathophysiologic explanation for how an increase in respiratory deaths could occur from a heat wave.

During a 2-week period of hot weather in New York City in 1966, there was a striking increase of 139% in the number of homicides committed. However, increases of similar magnitude have not been demonstrated subsequently, and, in any case, increased num-

bers of homicides accounted for less than 2% of the mortality excess during the 1966 heat wave (30).

Many heat-wave-associated deaths are not a clear and direct result of an overwhelming heat stress (heatstroke), nor do they fall into any of the other categories of disease mentioned above. They are seen in the form of apparently excess deaths from a broad variety of underlying causes (e.g., nephritis, diabetes) that do not have any obvious relationship to the heat. Mortality excesses in each of these categories do not occur consistently during heat waves. Moreover, each specific diagnosis tends to account for a relatively small proportion of the excess death (24, 30, 31). It has been suggested that heat-wave-related mortality in this broad group of categories may reflect an ability of heat stress to precipitate death for debilitated persons who are ill from a wide variety of chronic diseases and would die in the near future anyway.

As evidence of this assumption, Lyster presented weekly totals of deaths occurring in Greater London and the rest of England's southeast region before, during, and after the summer heat waves in 1975 and 1976. Mortality increased during both periods of severe heat in both geographic areas, but the increases were followed by several weeks of seemingly lower-than-normal mortality (42). In heat waves before and since, however, such a phenomenon has been sought but not observed. Henschel presented data showing that the average daily death rate in St. Louis was about the same before and after the 1966 heat wave, and Ellis *et al.* reported the absence of a deficit of deaths following a heat wave in New York in 1972 (28, 29). Similarly, there was no substantial fall in mortality following the excess deaths resulting from the 1980 heat wave in St. Louis and Kansas City, Missouri (26). Thus, a depression in the crude mortality rate following a heat-wave-induced elevation is not always found.

Heat-Wave-Associated Morbidity

Nonfatal illness resulting from heat waves has been less well quantified than has heat-wave-related mortality. During the July 1980 heat wave, hospitals in St. Louis and Kansas City, Missouri, admitted 229 and 276 patients, respectively, with nonfatal illnesses thought by the attending physicians to be related to the heat (26). In Memphis during the same period, there were 483 visits to emergency rooms for heat-related illness. Loss of consciousness was a frequent complaint, affecting almost half of the patients seen at City Hospital in Memphis. Dizziness, nausea, and cramps were other common symptoms. The proportions of the illnesses diagnosed for the 471 patients for whom diagnosis was known were as follows: heatstroke, 17%; heat exhaustion, 58%; heat syncope, 4%; heat cramps, 6%; and other heat-related illness, 15% (27).

Indirect measures of morbidity also arise with the heat. In July 1980 in St. Louis and Kansas City, Missouri, emergency room visits rose 14% and 8%, respectively. The respective increases in overall hospital admissions were 5% and 2% (26).

Factors Influencing Morbidity and Mortality: Determinants of Risk

Variation in Heat-Related Health Effects over Time

The public health impact of heat at any given time depends not only on the weather conditions at that time but also on previously existing conditions. That this is true can be seen in the fact that there is a delay between the onset of the heat wave and the appearance of substantial adverse effects on public health. Unusually high temperatures on several days in succession are required to produce a noticeable increase in mortality, and heat waves lasting less than 1 week result in relatively few deaths. The importance of sustained hot conditions is also illustrated by the observation that heat waves in which relatively little nighttime cooling occurs (i.e., those in which daily minimum temperatures are especially elevated) are particularly lethal (28, 42, 43).

Over greater periods of time, however, hot weather seems to lose some of its virulence. Acclimatization of individuals to heat stress is a phenomenon that has been well documented by means of physiologic experimentation (44, 45). Populations, too, seem to acclimatize to the heat over the course of a summer (46). Thus, heat waves in the Northern Hemisphere occurring in August and September seem to be less lethal than those occurring in June and July (47). During a sustained heat wave, after an initial dramatic increase, the number of deaths tends to return toward baseline, even though the temperature may remain elevated (46). This fall in crude mortality may result not only from acclimatization, but also from earlier deaths of susceptible persons, decreasing their number in the population at risk (24).

Urbanization and Risk

Heat waves cause a disproportionately severe health impact in cities, to a large extent sparing more rural and suburban areas. In July 1980, deaths in St. Louis and Kansas City, Missouri, were 57% and 65% higher, respectively, than in July 1979. In contrast, there was an excess mortality of only 10% in the remainder of Missouri, which is largely suburban and rural (26). This trend is not a recent development. In a review of deaths caused by heat and registered in the United States from 1900 to 1928, Shattuck and Hilferty found that the rate of heat-related deaths was substantially higher in urban than in rural areas (48). In a later work, the same investigators found that the effect of heat on death rates increased markedly with increase in the size of a city, suggesting a sort of "dose-response" effect of urbanization (49).

One reason health effects of hot weather may be more extensive in cities is that temperatures there may actually be somewhat higher than in surrounding rural and suburban areas. During the 1980 heat wave, the daily maximum temperature averaged 2.5°C higher and the daily minimum temperature averaged 4.1°C higher at the Kansas City downtown airport than at the suburban Kansas City International Airport (26).

The concept of the urban "heat island" has also been invoked to explain the disproportionate severity of the health impact of heat in cities. The masses of stone, brick, concrete, asphalt, and cement that are typical of urban architecture absorb radiant heat energy from the sun during the day and radiate that heat during nights that would otherwise be cooler. In many cities there are relatively few trees to provide shade. Tall city buildings may effectively decrease wind velocity, thereby decreasing the contribution of moving air to evaporative and convective cooling (3, 50, 51).

The relative poverty of some urban areas is another factor that may contribute to the severity of urban heat-related health effects (26). Poor people are less able to afford cooling devices and the energy needed to run them.

One report from Italy suggests that an urban predominance of heat-related health effects may not be universal. During a heat wave in July 1983 in the Latium region, one of 20 regions into which Italy is divided, mortality recorded at various inpatient facilities (hospitals and clinics) increased 49% over the previous year in the area outside of Rome, but only 25% in Rome itself (40). The reasons for this anomalous finding are unclear.

There is considerable variation among different cities with regard to susceptibility to hot-weather-related health effects. For example, summer temperatures that would not be considered unseasonably high in Phoenix, Arizona, have occurred in St. Louis, Missouri, and caused a severe, adverse impact on public health. In July 1980 in St. Louis, 122 deaths, 229 hospitalizations, and an increase in total mortality of 57% over the previous year were attributed to the heat (26). During that period, however, the average daily maximum temperature in St. Louis was 95.4°F—12.2°F lower than the normal July daily maximum temperature in Phoenix—and the average daily minimum temperature in St. Louis was 74.5°F—3°F lower than Phoenix's normal daily minimum temperature. The highest temperature recorded in St. Louis during the heat wave was 107°F, only 2.2°F higher than the expected (normal) maximum temperature of 104.8°F on any given July day in Phoenix (52). Even after taking the higher humidity of St. Louis into account, its July 1980 temperatures were approximately those of an average July in Phoenix (7).

The reasons for the differences in heat sensitivity of various cities have not been studied extensively. Possible explanations include differences in age structure and acclimatization of the population, architectural style, building materials, and use of air conditioning.

High-Risk Groups

The overall mortality increases observed during heat waves disproportionately affect the elderly. During a heat wave in July 1983, deaths in Rome, Italy, increased 23% overall, but increased 35% among persons more than 64 years of age (40). The increase in mortality in Greater London resulting from a heat wave in 1975 occurred almost exclusively among persons 65 years of age or older (53). In New York City in the

summer of 1966, deaths among persons age 45–64 increased substantially, but the increase in deaths of persons more than 80 years of age was far greater. Investigators also judged that excess deaths among persons 80 years of age or older began earlier in the heat wave than those among persons ages 45–64 years, possibly indicating greater heat sensitivity of the older group (30).

Deaths specifically designated by physicians as having been caused by the heat also occur with a disproportionately high frequency among the elderly. This trend is easily seen in Figure 12-2, a graph of age-specific rates for heat-related mortality in the United States in the period 1979–91. Infancy and early childhood are periods of relative sensitivity to the heat. The rate of heat-related mortality is lowest in late childhood. It then increases monotonically throughout the teenage and adult years, with the slope of the curve increasing rapidly as old age is approached. This pattern is not a new finding; Shattuck and Hilferty observed essentially identical trends associated with heat-related deaths in Massachusetts in 1900–1930, in New York in 1900–1928, and in Pennsylvania in 1906–1928 (49).

The elderly are also at increased risk of acquiring nonfatal heatstroke. A 1980 study of heatstroke survivors in St. Louis and Kansas City revealed that 71% were more than 65 years of age, although this group made up only about 15% of the population at risk (26). Other studies have consistently confirmed the susceptibility of the elderly (27, 29, 47).

The predisposition of the elderly to health effects of the heat may partially reflect impaired physiologic responses to heat stress. Vasodilation in response to the heat requires increased cardiac output, but persons more than 65 years of age are less likely to have the capacity to increase cardiac output and decrease systemic vascular resistance during hot weather (54). Moreover, the body temperature at which sweating begins increases with increasing age (55). The elderly are more likely than younger persons to have chronic diseases or to be taking medications (e.g., major tranquilizers and anticholinergics) that can increase the risk of heatstroke (56, 57).

Finally, old people perceive differences in temperature less well than do younger persons (58). They may, therefore, less effectively regulate their thermal environments.

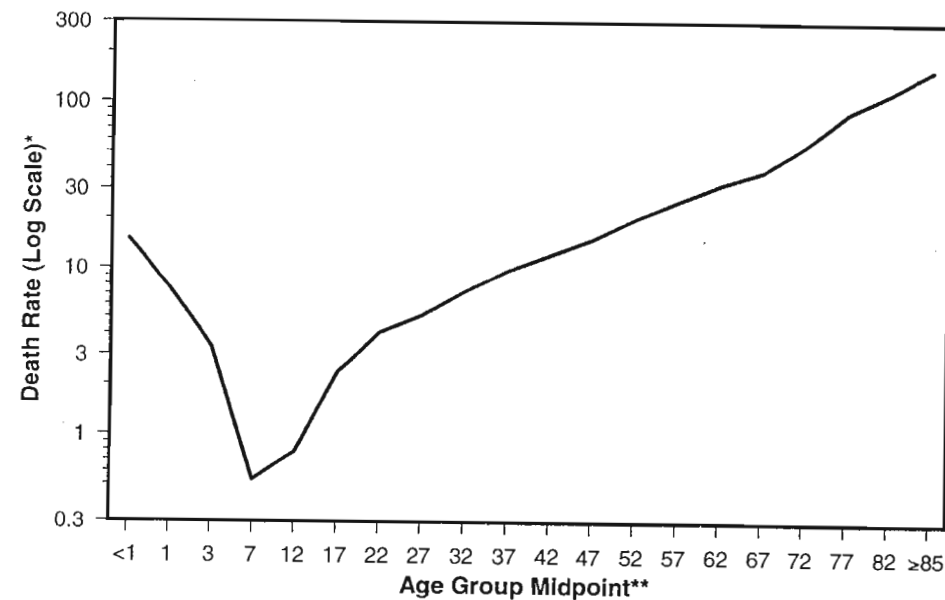
At the other extreme of age, the rate of physician-diagnosed heat-related death is higher for babies and young children, as shown in Figure 12-2. However, the magnitude of this increased risk is nowhere near as great as it is for elderly persons. There was no detectable increase in mortality for the age group 0–4 years in Greater London during the June–July 1975 heat wave (53). Only one of the 83 persons who died of heat-related causes in Memphis, Tennessee, in July 1980 was less than 20 years of age (a baby in the first year of life) (27). No cases of fatal or nonfatal heatstroke were found to have occurred among persons aged 0–18 years in St. Louis or Kansas City during the July 1980 heat wave, despite careful case-finding efforts in pediatric hospitals and medical examiners' offices (26). Nevertheless, Henschel and others found that four of 182 persons who died of heat-related illness during the July 1966 heat wave in St. Louis were babies less than 1 year of age (29). The small but definitely increased risk of death

from heat for babies and young children is most clearly seen in summaries of state and national data compiled over a number of years (as in Fig. 12-2) than in studies of specific heat waves in individual cities.

Other observations document the sensitivity to heat of the very young. Healthy babies kept in hot areas have been found to run temperatures as high as 103°F, and mild fever-causing illnesses of babies may be tipped over into frank heatstroke by heat stress. Children with congenital abnormalities of the central nervous system and with diarrheal illness appear to be particularly vulnerable (59, 60). Parents may contribute to risk by failing to give enough hypotonic fluid during the heatwave and by dressing the child too warmly (60, 61).

U.S. national figures show that in the teenage years and during early and middle adult life, males have an increased risk of heat-related death compared with females (Table 12-3). This difference may reflect a tendency toward greater heat exposure and exertional heat stress among males from occupational and leisure activities, but definitive epidemiologic data on this point are lacking. At the extremes of age, there is less difference between the sexes in rates of heat-related deaths.

Interestingly, many studies of heat waves have demonstrated greater numbers of heat casualties among women than among men. During the 1966 heat wave in New York City, deaths among women were 50% greater than expected, but deaths of men in-



* Per 10 million population.

** 5 year age groups except for first three groups; which are <1, 1, and 2-4 years old.

Figure 12-2. Rates of death attributed to heat (ICD E900), by age, United States, 1979–1991.

Table 12-3 Rates* of Death Attributed to the Heat (Underlying Cause Coded as E900, ICD-9) by Age Group and Sex with Age-Specific Male/Female Rate Ratios, United States, 1979-1991

Age Group	Male	Female	Rate Ratio
1	15.60	13.99	1.12
1	9.16	5.68	1.61
2-4	3.34	3.34	1.00
5-9	0.60	0.45	1.33
10-14	1.11	0.45	2.47
15-19	3.95	0.73	5.41
20-24	6.88	1.23	5.59
25-29	8.60	1.73	4.97
30-34	12.59	2.09	6.02
35-39	16.43	3.23	5.09
40-44	19.98	4.44	4.50
45-49	22.70	7.71	2.94
50-54	30.12	10.97	2.75
55-59	37.16	15.34	2.42
60-64	43.83	22.20	1.97
65-69	49.43	29.71	1.66
70-74	68.29	47.43	1.44
75-79	99.25	81.22	1.22
80-84	130.28	107.40	1.21
85+	202.90	142.01	1.43

*Per 10 million population/year.

creased by only 25% (30). In July of the same year in St. Louis, 59% of heat-related deaths occurred among women (29). In July 1980 in Memphis, 61% of 83 persons who were diagnosed by a physician as having died from the heat were women (28). In Latium Region, Italy, women accounted for 65% of fatal cases meeting specific diagnostic criteria for heatstroke (40).

The probable reason for an apparent excess of women among heat fatalities despite the generally higher age-specific rates of heat-related mortality among men is that age confounds the association of female sex with death due to heat. Elderly populations are the ones at greatest risk, and there are substantially more women than men among the elderly (62). The existence of such confounding was demonstrated in a heat-wave study in which age-adjusted rates of heatstroke were virtually identical for the two sexes despite a predominance of female study subjects (26).

Heat-related health effects are disproportionately severe in areas of low socioeco-

omic status. In 1966 in St. Louis, the death rate rose most dramatically in areas of low median family income in which there was substantial crowding (high numbers of persons/room) (30). In 1980 in St. Louis and Kansas City, Missouri, the heatstroke rate in census tracts in the highest socioeconomic quartile was about one-sixth that in tracts in the lowest quartile. The rates were intermediate in the tracts of intermediate socioeconomic status (26). Factors leading to the relatively low incidence of heat-related health effects in well-to-do areas may include availability of air-conditioning, abundance of trees and shrubs that provide shading, and access to health care.

In several studies, the rates of heat-related illness have been higher for blacks than for whites. In 1980 in Texas, the heat-related death rate was 21.1/million for blacks and 8.1/million for whites (47). Age-adjusted heatstroke rates were three to six times higher for minority races (principally blacks) in St. Louis and Kansas City in July 1980 (26). The association of black race and relatively low socioeconomic status may well account for the disproportionately high heatstroke rate for blacks in the United States. No biologically based vulnerability of any particular race has been shown.

Persons with a history of prior heatstroke have been shown to maintain thermal homeostasis in a hot environment less well than otherwise comparable volunteers (63). Whether a heatstroke damages the brain's thermoregulatory apparatus or thermoregulatory abnormalities antedate the first heatstroke is not known. However, persons with a history of heatstroke should be considered at risk of a recurrence.

Obesity is an important factor affecting heat tolerance. Obese subjects exercising in a hot environment show a greater increase in rectal temperature and heart rate than do lean subjects (64, 65). The insulating effect of subcutaneous fat impedes the transfer of metabolic heat from core to surface. Soldiers in the U.S. Army who died of heatstroke during basic training during World War II were much more obese than their peers (66). However, obesity may not importantly influence the rate of heatstroke for the largely sedentary elderly population that is at greatest risk during a heat wave (56).

Persons with other less common conditions may also tolerate the heat poorly. These conditions include congenital absence of sweat glands and scleroderma with diffuse cutaneous involvement. In both conditions, perspiration is markedly diminished, resulting in impaired thermoregulation in a hot environment (67, 68).

Some drugs predispose to heatstroke. Neuroleptic drugs (e.g., phenothiazines, butyrophenones, and thioxanthenes) have been particularly strongly implicated. Phenothiazine-treated animals survive in a hot environment for shorter periods than controls, and heatstroke occurs with increased frequency among patients taking these drugs (56, 69). Neuroleptics appear to sensitize both to cold and heat (69).

In laboratory tests of human volunteers, anticholinergic drugs decrease heat tolerance. Persons treated with anticholinergics while exposed to heat have been reported to have a decrease or cessation of sweating and a rise in rectal temperature (70). Many commonly used prescription drugs (e.g., tricyclic antidepressants, some antiparkinsonian agents) and nonprescription drugs (e.g., antihistamines, sleeping pills) have prominent

anticholinergic effects, and in one study the use of such drugs was more common among cases than controls (56). The likely mechanism of action appears to be inhibition of the ability to perspire.

Certain stimulant and antidepressant drugs taken in combination or in overdose situations may induce the syndrome of heatstroke. Severe hyperthermia has been reported to result from an amphetamine overdose, an amphetamine taken with a monoamine oxidase inhibitor, and a tricyclic taken in combination with a monoamine oxidase inhibitor (71–73).

Methodologic Problems of Epidemiologic Studies

The literature on heat-wave-related morbidity and mortality has been complicated by the fact that different researchers have studied different health outcomes. In some heat waves that cause substantial excess mortality, relatively few or none of these deaths are certified as having been caused by heat. Since physician-designated heat-related deaths are often so few in comparison to the magnitude of the total increase in mortality, some investigators have chosen to study the total increase in mortality itself as the health outcome of importance. In such studies the number of deaths has been studied in relation to the results of meteorologic measurements made at a local weather station. Since the administrative mechanisms for recording the mere occurrence of a death on a given date are fairly dependable in developed countries, the measure of the health outcome being studied (death on a particular day) is almost exact. Nevertheless, the weather station from which the data are taken may be at a site, such as an airport, miles away from the area in which most deaths occur. Even if readings are taken within the area inhabited by the population at risk, they are outdoor measurements that do not necessarily reflect the variable conditions within dwellings and other buildings in which most of the deaths occur. Since such studies also fail to take into account other host and environmental risk factors, only very limited conclusions can be drawn from their findings.

A number of studies of heat-wave-associated mortality have used information provided on death certificates, comparing deaths occurring during a heat wave with those during a control period. Apparent excess death attributed to a variety of diagnostic entities (e.g., stroke, ischemic heart disease) has been studied, not just those deaths corresponding to clear-cut heat-related illness (e.g., heatstroke). These studies have yielded interesting findings, but the well-known imprecision in certain of the data listed on death certificates leads to corresponding imprecision in study results. In particular, physicians' criteria for diagnosing various causes of death vary over time and in different locations.

In an attempt to deal specifically with morbidity and mortality that are clearly due to the heat—excluding cases of illness and death that could have occurred even in the

absence of heat—some investigators have limited their studies of disease to cases classified as “heat-related.” This term generally refers to a physician’s determination that an illness or death was in some way related to environmental heat (this is how the term is used in this chapter). But even defined in this way, the term is somewhat ambiguous. Heat can produce several distinct syndromes, all of which are “heat-related.” Moreover, heat-related death in some studies refers only to deaths in which environmental heat is judged to be the underlying cause of death, but in other reports, deaths for which heat was only a contributing factor are also included. Moreover, the use of this categorization in diagnosis and coding of the cause of death may vary greatly from region to region. Writing about the 1966 heat wave, which caused severe health consequences in New York City and St. Louis, Schuman observed that 130 deaths in St. Louis were attributed to “excessive heat and insulation” but that in New York City “only a handful of deaths were coded, preference being assigned . . . to underlying circulatory and degenerative conditions” (30). Variation among physicians regarding the determination of heat “relatedness” continues to complicate studies of heat-related health effects (74).

In an effort to limit parts of their investigation to the study of a clear-cut illness caused by heat, researchers investigating the effects of the 1980 heat wave in St. Louis and Kansas City, Missouri, defined the following people as having heatstroke:

Patients with a presenting temperature (measured anywhere on the body) greater than or equal to 41.1°C (106°F); patients with documented temperature greater than or equal to 40.6°C (105°F) if altered mental status or anhidrosis was also present; and those pronounced dead on arrival at the hospital or medical examiner’s office if the body temperature . . . was greater than or equal to 41.1°C (106°F). (56)

Other studies undertaken since that time have defined heatstroke similarly (14, 75). Strict definitions could also be developed for the study of other outcomes whose direct cause is the heat (i.e., heat exhaustion, heat syncope, heat cramps). Such definitions do not necessarily help the clinician attempting to diagnose the case of an individual patient. Their usefulness lies in their value as entry criteria for epidemiologic studies of groups of patients, enabling the investigator to explain precisely which clinical entities have been studied when “heat-related” illness is the subject of the study. In this manner, future investigators will be better able to clarify and quantify the health consequences of heat.

Prevention of Adverse Health Effects Caused by Heat

Timing of Preventive Measures

In most parts of the United States, heat waves severe enough to threaten health do not occur every year, and several relatively mild summers may intervene between major

heat waves. The erratic occurrence of heat waves hinders effective planning of prevention efforts. It may be administratively difficult for health departments to plan for adequate resources that will be available if needed but that will not be wasted if no heat wave occurs.

Although long-term weather forecasts (i.e., those done some months in advance of the event) cannot reliably predict periods of severe heat, near-term forecasts of hot weather several days in advance are becoming increasingly accurate. Could one also forecast the extent of mortality and morbidity expected to result from anticipated hot weather? Even 1 or 2 days of advance warning regarding the probable extent of heat-related adverse health effects would be of use in planning for their prevention.

Apparent Temperature, also known as *heat index* (one of the indices of human heat stress discussed above), has been proposed as a guide to classifying how hazardous to health the anticipated weather may be. However, the index was not developed for this specific use. The hazard posed by heat stress depends not only on its magnitude at a given moment but also on how it has varied over time. Moreover, this index in no way takes into account the variation in heat sensitivity of different regions. Thus, Apparent Temperature by itself, independent of geographical location and antecedent weather conditions, will probably not be found to be a very useful predictor of the extent of heat-related health effects to be expected in a population at risk (2, 3, 7).

Several authors have attempted to develop mathematical models to quantify the increase in numbers of deaths to be expected for a given degree of temperature increase. These formulae have taken into account such factors as the usual seasonal trends in mortality, acclimatization, and the age structure and previous hot weather exposure of the population at risk. Currently available mathematical models have been fitted retrospectively to past mortality and meteorologic data. They are reasonably in accord with the observations from which they were developed. However, none of these models has yet demonstrated its usefulness in the prospective prediction of heat-related adverse health effects (43, 46, 51). This is an important area for further research.

In the absence of reliable prediction, early detection of important adverse health consequences of heat could provide public health professionals with useful information, allowing them to mobilize resources for prevention relatively early in an epidemic of heat-related illness. A large increase in the caseload of the local medical examiner that is unexplained by any other disaster has been proposed as an early indicator of severe heat-related health effects in a community. This proposal was based on 1980 data from two midwestern cities showing that the number of cases reported to medical examiners increased to a proportionately greater extent than did other indirect measures of the heat's impact on public health, including total mortality, emergency room visits, and hospital admissions. Moreover, the total number of medical examiner cases is much more easily and rapidly available than these other statistics. Even the time required for postmortem diagnosis does not delay data collection (36). Although prospective evaluation has not yet established the degree of utility of this sort of surveillance, this fact

should not discourage state and local health departments from further evaluation of the method within their jurisdictions. There are as yet no firm criteria regarding just how much of an increase in caseload should trigger implementation of prevention programs.

Content of Prevention Programs

Programs to prevent heat-related illness should concentrate on measures whose efficacy is supported by empirical data. Many heat-illness prevention efforts have centered around the distribution of electric fans to persons at risk. However, study of the 1980 heat wave in Missouri did not show a significant protective effect of fans (56). This finding is consistent with theoretical predictions and empirical data showing that as air temperature rises toward about 99°F—the exact value depends on the humidity and other factors—increased air movement ceases to lessen heat stress. At even higher temperatures, increased movement of air may actually exacerbate heat stress (2–4, 7). Although further epidemiologic studies are required to evaluate the preventive efficacy of fans, fans probably should not be used in situations in which established indices of heat stress suggest they might be harmful.

Air-conditioning effectively prevents heatstroke and may decrease the incidence of other adverse health effects of heat waves. In one study, the presence of 24-hour air-conditioning in the home reduced heatstroke risk by 98%. In addition, just spending more time in air-conditioned places (regardless of whether there was a home air conditioner) was associated with a 4-fold reduction in heatstroke (56). These findings suggest that air-conditioned shelters are an effective means of preventing heatstroke. Persons at high risk who do not have home air-conditioning may benefit from spending a few hours each day in an air-conditioned environment.

The maintenance of adequate hydration is important in preventing heat-related illness. Increases in body temperature of heat-stressed volunteers were lessened when fluid losses were frequently replaced (76). Moreover, taking extra liquids has been associated with decreased risk of heatstroke (56). More fluid than the amount dictated by thirst may be required to fully offset the increased fluid losses that occur during hot weather (76, 77). Thus, unless there is a medical contraindication, persons at risk from the heat should be advised to make a special effort to increase the amount of liquid they consume.

Adequate intake of salt with meals is important. Although salt supplementation with tablets may be important in preventing electrolyte imbalances for carefully selected individuals who must tolerate intense heat for prolonged periods (20), it is of doubtful benefit in preventing heat-related illness in the general population (56). Furthermore, such supplementation may be harmful for persons with certain chronic illnesses in which a high sodium intake is undesirable (e.g., persons with hypertension, congestive heart failure). Therefore, salt tablets should not be recommended for consumption by the general population during a heat wave.

Persons at high risk should be advised to reduce activity in the heat, since such behavior appears to have protected against heatstroke in one study (56). Conversely, athletic exertion in the heat substantially increases risk, although risk does not increase as much for persons who have become acclimatized by training in a hot environment (66).

Target Groups

To be maximally effective, programs for the prevention of heat-related illness should be directed toward groups known to be at particularly high risk. Cities—especially low socioeconomic-status, inner-city areas—are particularly appropriate targets for prevention efforts. The elderly should receive special attention, since old age is one of the factors most strongly associated with increased risk of heatstroke or death from other causes during a heatwave. As much as possible, special living facilities for the elderly and institutions such as nursing homes and hospitals in which many elderly persons are to be found should be air conditioned during severely hot weather. The elderly living at home should not be forgotten, however, since they may be at even greater risk than those in institutions (78). Parents should be made aware of the increased heat sensitivity of babies and children less than 5 years of age. Patients taking neuroleptic or anticholinergic drugs should be counseled regarding their possible increased sensitivity to heat.

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