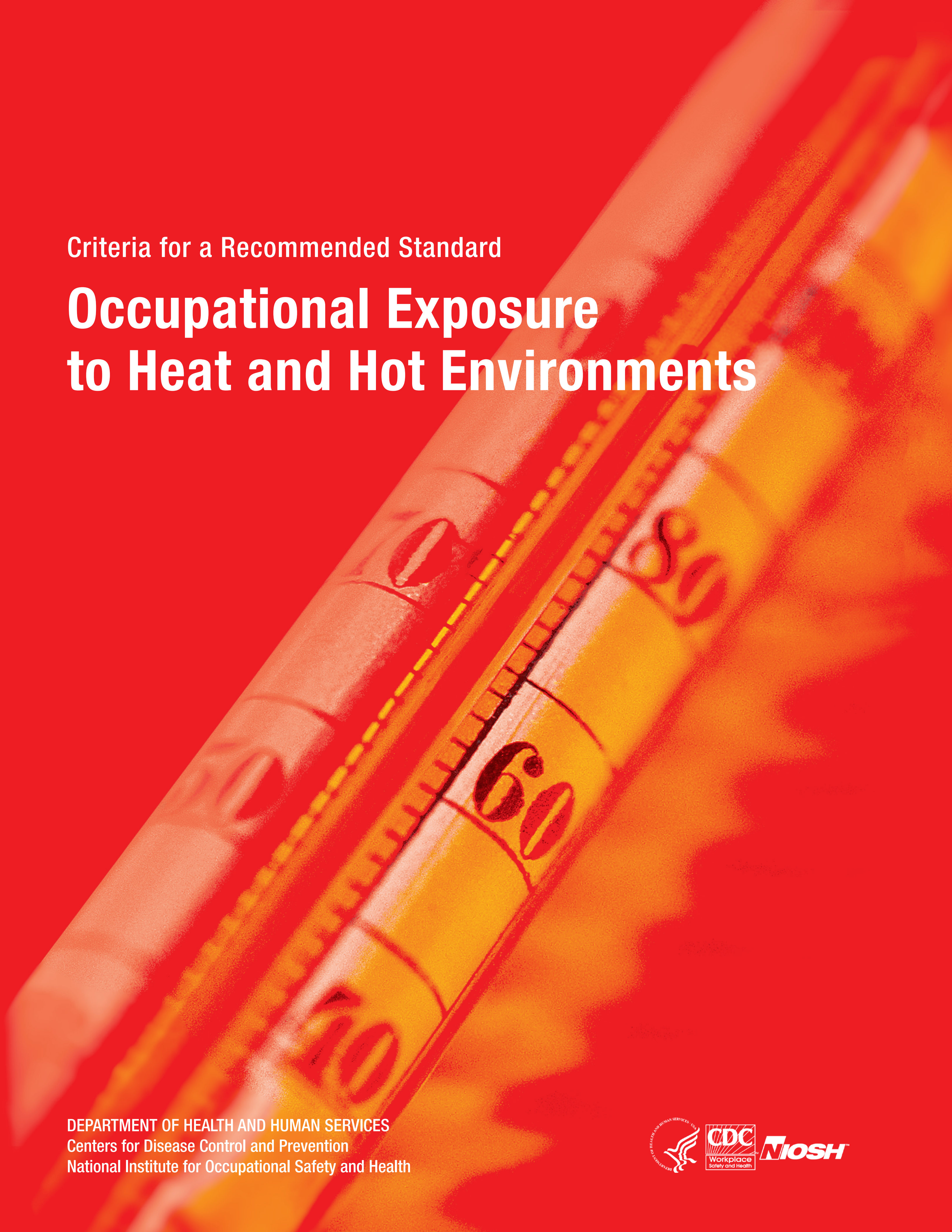

Criteria for a Recommended Standard

Occupational Exposure to Heat and Hot Environments

DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention
National Institute for Occupational Safety and Health

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Occupational Exposure to Heat and Hot Environments

Revised Criteria 2016

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Foreword

When the U.S. Congress passed the Occupational Safety and Health Act of 1970 (Public Law 91-596), it established the National Institute for Occupational Safety and Health (NIOSH). Through the Act, Congress charged NIOSH with recommending occupational safety and health standards and describing exposure levels that are safe for various periods of employment, including but not limited to the exposures at which no worker will suffer diminished health, functional capacity, or life expectancy because of his or her work experience.

Criteria documents contain a critical review of the scientific and technical information about the prevalence of hazards, the existence of safety and health risks, and the adequacy of control methods. By means of criteria documents, NIOSH communicates these recommended standards to regulatory agencies, including the Occupational Safety and Health Administration (OSHA) and the Mine Safety and Health Administration (MSHA), health professionals in academic institutions, industry, organized labor, public interest groups, and others in the occupational safety and health community.

A criteria document, *Criteria for a Recommended Standard: Occupational Exposure to Hot Environments*, was prepared in 1972 and first revised in 1986. The revision presented here takes into account the large amount of new scientific information on working in heat and hot environments. This revision includes updated information on heat-related illnesses, risk factors affecting heat-related illness, physiological responses to heat, effects of clothing on heat exchange, and recommendations for control and prevention.

Occupational exposure to heat can result in injuries, disease, death, and reduced productivity. Workers may be at risk for heat stress when exposed to hot environments. Exposure to hot environments and extreme heat can result in illnesses, including heat stroke, heat exhaustion, heat syncope, heat cramps, and heat rashes, or death. Heat also increases the risk of workplace injuries, such as those caused by sweaty palms, fogged-up safety glasses, and dizziness.

NIOSH urges employers to use and disseminate this information to workers. NIOSH also requests that professional associations and labor organizations inform their members about the hazards of occupational exposure to heat and hot environments.

NIOSH appreciates the time and effort taken by the expert peer, stakeholder, and public reviewers, whose comments strengthened this document.

J Howard

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Executive Summary

Occupational exposure to heat can result in injuries, disease, reduced productivity, and death. To address this hazard, the National Institute for Occupational Safety and Health (NIOSH) has evaluated the scientific data on heat stress and hot environments and has updated the *Criteria for a Recommended Standard: Occupational Exposure to Hot Environments* [NIOSH 1986a]. This document was last updated in 1986, and in recent years, including during the Deepwater Horizon oil spill response of 2010, questions were raised regarding the need for revision to reflect recent research and findings. In addition, there is evidence that heat stress is an increasing problem for many workers, particularly those located in densely populated areas closer to the equator where temperatures are expected to rise in relation to the changing climate [Lucas et al. 2014]. This revision includes additional information about the physiological changes that result from heat stress; updated information from relevant studies, such as those on caffeine use; evidence to redefine heat stroke and associated symptoms; and updated information on physiological monitoring and personal protective equipment and clothing that can be used to control heat stress.

Workers who are exposed to extreme heat or work in hot environments indoors or outdoors, or even those engaged in strenuous physical activities may be at risk for heat stress. Exposure to extreme heat can result in occupational illnesses caused by heat stress, including heat stroke, heat exhaustion, heat syncope, heat cramps, heat rashes, or death. Heat can also increase workers' risk of injuries, as it may result in sweaty palms, fogged-up safety glasses, dizziness, and may reduce brain function responsible for reasoning ability, creating additional hazards. Other heat injuries, such as burns, may occur as a result of contact with hot surfaces, steam, or fire. Those at risk of heat stress include outdoor workers and workers in hot environments, such as fire fighters, bakery workers, farmers, construction workers, miners (particularly surface miners), boiler room workers, and factory workers.

In 2011, NIOSH published with the Occupational Safety and Health Administration (OSHA) a co-branded infosheet on heat illness. Through this combined effort, many recommendations were updated, including those on water consumption. In addition, factors that increase risk and symptoms of heat-related illnesses were more thoroughly defined. In 2013, NIOSH published "Preventing Heat-related Illness or Death of Outdoor Workers". Outdoor workers are exposed to a great deal of exertional and environmental heat stress.

Chapters on basic knowledge of heat balance and heat exchange largely remain unchanged, although clothing insulation factors have been updated to reflect current International Organization for Standardization (ISO) recommendations. Additional information on the biological effects of heat has become available in recent studies, specifically increasing the understanding of the central nervous system, circulatory regulation, the sweating mechanism, water and electrolyte balance, and dietary factors. New knowledge has been established about risk factors that can increase a worker's risk of heat-related illness. Those over the age of 60 are at additional risk for suffering from heat disorders [Kenny et al. 2010]. Additional studies have examined sex-related differences regarding sweat-induced electrolyte loss and whole-body sweat response, as well as how pregnancy affects heat stress tolerance [Meyer et al. 1992; Navy Environmental Health Center 2007; Gagnon and Kenny 2011]. As obesity and the increasingly overweight percentage of the population in the United States continue to increase, this is now a major health concern in workers. Heat disorders among the obese and overweight occur more frequently than in lean individuals [Henschel 1967; Chung and Pin 1996; Kenny et al. 2010]. Another factor affecting heat-related illness is use of drugs, including cocaine, alcohol, prescription drugs, and caffeine. Caffeine use has long been argued against, as it has a diuretic effect and may reduce fluid volume, leading to cardiovascular strain during heat exposure [Serafin 1996]. However, more recent studies have found that the effect of caffeine on heat tolerance may be much less than previously suspected [Roti et al. 2006; Armstrong et al. 2007a; Ely et al. 2011].

The definition of heat stroke has also changed in recent years. Heat stroke is now classified as either classic heat stroke or exertional heat stroke which is more common in workplace settings. Characteristics of the individual (e.g., age and health status), type of activity (e.g., sedentary versus strenuous exertion), and symptoms (e.g., sweating versus dry skin) vary between these two classifications [DOD 2003]. Re-education is needed in the workplace especially about symptoms. Many workers have incorrectly been taught that as long as they were still sweating they were not in danger of heat stroke.

Measurements of heat stress are largely unchanged since the last revision, although additional information has been added about bimetallic thermometers and the psychrometric chart. The latter is a useful graphic representation of the relationships among dry bulb temperature, wet bulb temperature, relative humidity, vapor pressure, and dew point temperature. Such charts are especially valuable for assessing the indoor thermal environment. In addition, many modern computers and mathematical models can be used to calculate heat stress indices, based on weather station data.

Heat stress can be reduced by modifying metabolic heat production or heat exchange by convection, radiation, or evaporation. In a controlled environment, these last three can be modified through engineering controls, including increasing ventilation, bringing in cooler outside air, reducing the hot temperature of a radiant heat source, shielding the worker, and using air conditioning equipment. Heat stress can also be administratively controlled through limiting the exposure time or temperature (e.g., work/rest schedules), reducing metabolic heat load, and enhancing heat tolerance (e.g., acclimatization). Although most healthy workers will be able to acclimatize over a period of time, some workers may be heat intolerant. Heat intolerance may be related to many factors; however, a heat tolerance test can be used to evaluate an individual's tolerance, especially after an episode of heat exhaustion or exertional heat stroke [Moran et al. 2007]. Additional preventive strategies against heat stress include establishing a heat alert program and providing auxiliary body cooling and protective clothing (e.g., water-cooled garments, air-cooled garments, cooling vests, and wetted overgarments).

Employers should establish a medical monitoring program to prevent adverse outcomes and for early identification of signs that may be related to heat-related illness. This program should include preplacement and periodic medical evaluations, as well as a plan for monitoring workers on the job.

Health and safety training is important for employers to provide to workers and their supervisors before they begin working in a hot environment. This training should include information about recognizing symptoms of heat-related illness; proper hydration (e.g., drinking 1 cup [8 oz.] of water or other fluids every 15–20 minutes); care and use of heat-protective clothing and equipment; effects of various factors (e.g., drugs, alcohol, obesity, etc.) on heat tolerance; and importance of acclimatization, reporting symptoms, and giving or receiving appropriate first aid. Supervisors also should be provided with appropriate training about how to monitor weather reports and weather advisories.

The NIOSH Recommended Alert Limits (RALs) and Recommended Exposure Limits (RELs) were evaluated. It was determined that the current RALs for unacclimatized workers and RELs for acclimatized workers are still protective for most workers. No new data were identified to use as the basis for updated RALs and RELs. Most healthy workers exposed to environmental and metabolic heat below the appropriate NIOSH RALs or RELs will be protected from developing adverse health effects. The Wet Bulb Globe Temperature–based limits for acclimatized workers are similar to those of OSHA, the American Conference of Governmental Industrial Hygienists, the American Industrial Hygiene Association, and the ISO. In addition, the Universal Thermal Climate Index (UTCI), originally developed in 2009, is gaining acceptance as a means of determining environmental heat stress on workers [Blazejczyk et al. 2013].

During the 2014 peer review of the draft criteria document, concerns were expressed about the sufficiency of the scientific data to support the NIOSH ceiling limits for acclimatized and unacclimatized workers. In fact, many acclimatized workers live and work in temperatures above the ceiling limits without adverse health effects. Further consideration of the scientific data led to the decision to remove the ceiling limit recommendations from the document.

Although research has produced substantial new information since the previous revision of this document, the need for additional research continues. Two newer areas of research that will likely continue to grow are the effects of climate change on workers and how heat stress affects the toxic response to chemicals. It is likely but unclear to what extent global climate change will impact known heat-exposure hazards for workers, especially with regard to severity, prevalence, and distribution [Schulte and Chun 2009; Schulte et al. 2016]. Toxicological research has shown that heat exposure can affect the absorption of chemicals into the body. Most of what is known on this subject comes from animal studies, so a better understanding of the mechanisms and role of ambient environment with regard to human health is still needed [Gordon 2003; Gordon and Leon 2005]. With changes in the climate, the need for a better understanding will become increasingly important [Leon 2008].

In addition to the updated research, this criteria document includes more resources for worker and employer training. Information about the use of urine color charts, including a chart and additional information, is in Appendix B. The National Weather Service Heat Index is in Appendix C, along with the OSHA-modified corresponding worksite protective measures and associated risk levels.

NIOSH recommends that employers implement measures to protect the health of workers exposed to heat and hot environments. Employers need to ensure that unacclimatized and acclimatized workers are not exposed to combinations of metabolic and environmental heat greater than the applicable RALs/RELs (see Figures 8-1 and 8-2). Employers need to monitor environmental heat and determine the metabolic heat produced by workers (e.g., light, moderate, or heavy work). Additional modifications (e.g., worker health interventions, clothing, and personal protective equipment) may be necessary to protect workers from heat stress, on the basis of increases in risk. In hot conditions, medical screening and physiological monitoring are recommended. Employers, supervisors, and workers need to be trained on recognizing symptoms of heat-related illness; proper hydration; care and use of heat-protective clothing and equipment; effects of various risk factors affecting heat tolerance (e.g., drugs, alcohol, obesity, etc.); importance of acclimatization; importance of reporting symptoms; and appropriate first aid.

Employers should have an acclimatization plan for new and returning workers, because lack of acclimatization has been shown to be a major factor associated with worker heat-related illness and death. NIOSH recommends that employers provide the means for appropriate hydration and encourage their workers to hydrate themselves with potable water <15°C (59°F) made accessible near the work area. Workers in heat <2 hours and involved in moderate work activities should drink 1 cup (8 oz.) of water every 15–20 minutes, but during prolonged sweating lasting several hours, they should drink sports drinks containing balanced electrolytes. In addition, employers should implement a work/rest schedule and provide a cool area (e.g., air-conditioned or shaded) for workers to rest and recover. These elements are intended to protect the health of workers from heat stress in a variety of hot environments.

Contents

Abbreviations

A_b	Area, Body Surface
A_{Du}	Area, DuBois
A_r	Area, Effective Radiating
A_s	Area, Solar Radiation
A_w	Area, Wetted
$A_w/SWA_{Du} \times 100$	Wettedness, Percent of Skin
ACGIH	American Conference of Governmental Industrial Hygienists
ACSM	American College of Sports Medicine
ADH	Antidiuretic Hormone
AIHA	American Industrial Hygiene Association
ATP	Adenosine Triphosphate
BLS	Bureau of Labor Statistics
BMI	Body Mass Index
bpm	Beats Per Minute
BUN	Blood Urea Nitrogen
°C	Degrees Celsius
C	Convection
Cal/OSHA	California OSHA
CDC	Centers for Disease Control and Prevention
CET	Corrected Effective Temperature
CK	Creatine Kinase
CNS	Central Nervous System
CPK	Creatine Phosphokinase
CO	Cardiac Output
CSF	Cerebrospinal Fluid
DIC	Disseminated Intravascular Coagulation
DOD	Department of Defense
-E	Evaporative Heat Loss
E	Evaporation
ECG	Electrocardiogram

E_{max}	Maximum Water Uptake
E_{req}	Amount of Sweat Evaporated to Maintain Body Heat Balance
EHE	Extreme Heat Event
EMS	Emergency Medical Services
ET	Effective Temperature
°F	Degrees Fahrenheit
F_{cl}	Reduction Factor for Heat Exchange due to Clothing
g	Gram
GI	Gastrointestinal
h	Hour
H	Body Heat Content
h_c	Convective Heat Transfer Coefficient
h_e	Evaporative Heat Transfer Coefficient
h_r	Radiative Heat Transfer Coefficient
h_{r+c}	Radiative + Convective Heat Transfer Coefficient
HAP	Heat Alert Program
HHE	Health Hazard Evaluation
HR	Heart Rate
HSI	Heat Stress Index
HTT	Heat Tolerance Test
I_a	Thermal Insulation, Still Air
I_{cl}	Thermal Insulation, Clothing
$I_{cl}+I_a$	Thermal Insulation, Effective
i_m	Moisture Permeability Index of Clothing
i_m/clo	Permeability Index-Insulation Ratio
ISO	International Organization for Standardization
K	Conduction
kcal	Kilocalories
kg	Kilogram
kJ	Kilojoule
L	Liter
M	Metabolism
Met	Unit of metabolism
min	Minute

mL	milliliter
mmHg	Millimeters of Mercury
MR	Metabolic Rate
MRT	Mean Radiant Temperature
ms^{-1}	Meters per Second
MSHA	Mine Safety and Health Administration
Na	Sodium
NIOSH	National Institute for Occupational Safety and Health
NFPA	National Fire Protection Association
NOAA	National Oceanic and Atmospheric Administration
NSAIDs	Nonsteroidal Anti-inflammatory Drugs
OEL	Occupational Exposure Limit
OSHA	Occupational Safety and Health Administration
P_a	Pressure, atmospheric
P_{sk}	Pressure, Wetted Skin
$P_{sk,s}$	Pressure, Skin Temperature
PCr	Creatine Phosphate
PHEL	Physiological Heat Exposure Limit
PPE	Personal Protective Equipment
R	Radiation
RAL	Recommended Alert Limit
RAAS	Renin-Angiotensin-Aldosterone System
REL	Recommended Exposure Limit
RER	Respiratory Exchange Ratio
RH	Relative Humidity
S	Body Heat Storage
SACHS	Standards Advisory Committee on Heat Stress
SCBA	Self-contained Breathing Apparatus
SR	Sweat Produced Per Unit Time
SV	Stroke Volume
SWA	Area of Skin Wet with Sweat
$\bar{t}_a$	Temperature, Ambient
t_{adb}	Temperature, Adjusted Dry Bulb
$\bar{t}_b$	Temperature, Mean Body

t_{cr}	Temperature, Core body
t_{dp}	Temperature, Dew-point
t_g	Temperature, Globe
t_{nwb}	Temperature, Natural Wet Bulb
t_o	Temperature, Operative
t_{or}	Temperature, Oral
t_r	Temperature, Radiant
$\bar{t}_r$	Temperature, Mean Radiant
t_{re}	Temperature, Rectal
t_{sk}	Temperature, Skin
$\bar{t}_{sk}$	Temperature, Mean Skin
t_{ty}	Temperature, Tympanic
t_{wb}	Temperature, Psychrometric Wet Bulb
TLV®	Threshold Limit Value
TWA	Time Weighted Average
ULPZ	Upper Limit of the Prescriptive Zone
UTCI	Universal Thermal Climate Index
V_a	Air Velocity
$\dot{V}_E$	Minute Ventilation
$\dot{V}O_2$ max	Maximum Oxygen Consumption
$\underline{w}$	Wettedness, Skin
W	Work
WBGT	Wet Bulb Globe Temperature
WGT	Wet Globe Temperature
WHO	World Health Organization

Glossary

Acclimatization: The physiological changes that occur in response to a succession of days of exposure to environmental heat stress and reduce the strain caused by the heat stress of the environment; and enable a person to work with greater effectiveness and with less chance of heat injury.

Area, DuBois (A_{Du}): Total nude body surface area in square meters (m^2), calculated from the DuBois formula, and based on body weight (kg) and height (m).

Area, Effective Radiating (A_r): Surface area of the body in square meters (m^2) that exchanges radiant energy with a radiant source.

Area, Solar Radiation (A_s): Surface area of the body in square meters (m^2) that is projected normal to the sun.

Area, Wetted (A_w): Square meters (m^2) of skin area covered by sweat.

Body Heat Balance: Steady-state equilibrium between body heat production and heat loss to the environment.

Body Heat Balance Equation: Mathematical expression of relation between heat gain and heat loss, expressed as $S = (M - W) \pm C \pm R \pm K - E$

Body Heat Storage (S): The change in heat content (either + or –) of the body.

Circadian Rhythm: Synchronized, rhythmic biological phenomena that occur on approximately a 24-hour cycle.

clo: A unit expression of the insulation value of clothing, $1 \text{ clo} = 5.55 \text{ kcal}\cdot\text{m}^2\cdot\text{h}^{-1}\cdot{}^\circ\text{C}^{-1}$. A clo of 1 is equal to the insulation required to keep a sedentary person comfortable at 21°C (~70°F). It is also sometimes expressed as $1 \text{ clo} = 0.155 \text{ m}^2\cdot{}^\circ\text{C}\cdot\text{W}^{-1}$.

Conductive Heat Transfer (K): The net heat exchange involving the direct transfer of heat via direct contact between two mediums (solid, liquid, or gas) that have a temperature differential.

Conductive Heat Transfer Coefficient (h_k): The rate of heat transfer between two mediums (solid, liquid, or gas) that have a temperature differential, expressed as $\text{W}\cdot\text{m}^{-2}\cdot{}^\circ\text{C}^{-1}$.

Convective Heat Transfer (C): The net heat exchange by convection between an individual and the environment.

Convective Heat Transfer Coefficient (h_c): The rate of heat transfer between the body surface and the ambient air per square meters (m^2) of skin surface, expressed as $\text{W}\cdot\text{m}^{-2}\cdot{}^\circ\text{C}^{-1}$.

Evaporative Heat Loss (-E): Body heat loss by evaporation of water (sweat) from the skin, expressed as kcal or W.

Evaporative Heat Transfer (E): Rate of heat loss by evaporation of water from the skin or gain from condensation of water on the skin, expressed as $\text{kcal}\cdot\text{h}^{-1}$, $\text{W}\cdot\text{m}^{-2}$, or W.

Evaporative Heat Transfer Coefficient (h_e): The rate of heat exchange by evaporation between the body surface and the ambient air, as a function of the vapor pressure difference between the two and air velocity.

Heat Capacity: Mass multiplied by specific heat of a body.

Heat Content of Body: The product of mean body temperature (t_b) and body heat capacity (body mass × tissue specific heat), with the latter being constant for any given body composition.

Heat Cramp: A heat-related illness characterized by spastic contractions of the voluntary muscles (mainly arms, hands, legs, and feet), usually associated with restricted salt intake and profuse sweating without significant body dehydration.

Heat Exhaustion: A heat-related illness characterized by elevation of core body temperature above 38°C (100.4°F) and abnormal performance of one or more organ systems, without injury to the central nervous system. Heat exhaustion may signal impending heat stroke.

Heat Strain: The physiological response to the heat load (external or internal) experienced by a person, in which the body attempts to increase heat loss to the environment in order to maintain a stable body temperature.

Heat Stress: The net heat load to which a worker is exposed from the combined contributions of metabolic heat, environmental factors, and clothing worn which results in an increase in heat storage in the body.

Heat Stroke: An acute medical emergency caused by exposure to heat from an excessive rise in body temperature [above 41.1°C (106°F] and failure of the temperature-regulating mechanism. Injury occurs to the central nervous system characterized by a sudden and sustained loss of consciousness preceded by vertigo, nausea, headache, cerebral dysfunction, bizarre behavior, and excessive body temperature.

Heat Syncope: Collapse and/or loss of consciousness during heat exposure without an increase in body temperature or cessation of sweating, similar to vasovagal fainting except that it is heat induced.

Heat Tolerance: The physiological ability to endure heat and regulate body temperature at an average or better rate than others, often affected by the individual's level of acclimatization and physical conditioning.

Humidity, Relative (RH): The ratio of the water vapor present in the ambient air to the water vapor present in saturated air at the same temperature and pressure.

Hyperpyrexia: A body core temperature exceeding 40°C (104°F).

Hyperthermia: A condition where the core temperature of an individual is higher than 37.2°C (99°F). Hyperthermia can be classified as mild (37.2–38.5°C; 99–101.3°F), moderate (i.e., heat exhaustion [38.5–39.5°C; 101.3–103.1°F]), profound (>39.5°C; 103.1°F), or profound clinical hyperthermia (i.e., heat stroke [>40.5°C; 104.9°F]), and death can occur without treatment (>45°C; 113°F).

Maximum Oxygen Consumption ($\dot{V}O_2$ max): The maximum amount of oxygen that can be used by the body.

Metabolic Rate (MR): Amount of chemical energy transferred into free energy per unit time.

Metabolism (M): Transformation of chemical energy into free energy that is used to perform work and produce heat.

Prescriptive Zone: The range of environmental temperatures where exercise at a given intensity results in thermal equilibrium, i.e., no change in core body temperature.

Pressure, Atmospheric (P_a): Pressure exerted by the weight of the air, which averages 760 mmHg at sea level and decreases with altitude.

Pressure, Water Vapor (P_a): The pressure exerted by the water vapor in the air.

Qualified Health Care Professional: An individual qualified by education, training, and licensure/regulation and/or facility privileges (when applicable) who performs a professional service within his or her scope of practice in an allied health care discipline, and independently reports that professional service.

Radiant Heat Exchange (R): The net rate of heat exchange by radiation between two radiant surfaces of different temperatures.

Radiative Heat Transfer Coefficient (h_r): Rate of heat transfer between two black surfaces per unit temperature difference, expressed as $W \cdot m^{-2} \cdot {}^{\circ}C^{-1}$.

Recommended Alert Limit (RAL): The NIOSH-recommended heat stress alert limits for unacclimatized workers.

Recommended Exposure Limit (REL): The NIOSH-recommended heat stress exposure limits for acclimatized workers.

Rhabdomyolysis: A medical condition associated with heat stress and prolonged physical exertion, resulting in the rapid breakdown of muscle and the rupture and necrosis of the affected muscles.

Standard Man: A representative human with a body weight of 70 kg (154 lb) and a body surface area of 1.8 m^2 (19.4 ft^2).

Sweating, Thermal: Response of the sweat glands to thermal stimuli.

Temperature, Adjusted Dry Bulb (t_{adb}): The dry bulb temperature is the temperature of the air measured by a thermometer that is shielded from direct radiation and convection.

Temperature, Ambient (t_a): The temperature of the air surrounding a body. Also called *air temperature* or *dry bulb temperature.*

Temperature, Ambient, Mean ($\bar{t}_a$): The mean value of several dry bulb temperature readings taken at various locations or at various times.

Temperature, Core Body (t_{cr}): Temperature of the tissues and organs of the body. Also called *Core Temperature.*

Temperature, Dew-point (t_{dp}): The temperature at which the water vapor in the air first starts to condense.

Temperature, Effective (ET): Index for estimating the effect of temperature, humidity, and air movement on the subjective sensation of warmth.

Temperature, Globe (t_g): The temperature inside a blackened, hollow, thin copper globe measured by a thermometer whose sensing element is in the center of the sphere.

Temperature, Mean Body ($\bar{t}_b$): The mean value of temperature at several sites within the body and on the skin surface. It can be approximated from skin and core temperatures.

Temperature, Mean Radiant ($\bar{t}_r$): The mean surface temperature of the material and objects surrounding the individual.

Temperature, Mean Skin ($\bar{t}_{sk}$): The mean of temperatures taken at several locations on the skin, weighted for skin area.

Temperature, Natural Wet Bulb (t_{nwb}): The wet bulb temperature under conditions of the prevailing air movement.

Temperature, Operative (t_o): The temperature of a uniform black enclosure within which an individual would exchange heat by convection and radiation at the same rate as in a nonuniform environment being evaluated.

Temperature, Oral (t_{or}): Temperature measured by placing the sensing element under the tongue for 3 to 5 minutes.

Temperature, Psychrometric Wet Bulb (t_{wb}): The lowest temperature to which the ambient air can be cooled by evaporation of water from the wet temperature-sensing element with forced air movement.

Temperature, Radiant (t_r): The point temperature of the surface of a material or object, calculated from the following: $MRT = T_g + (1.8\ V_a^{0.5})(T_g - T_a)$, where MRT = Mean Radiant Temperature (°C), T_g = black globe temperature (°C), T_a = air temperature (°C), and V_a = air velocity ($m{\cdot}s^{-1}$).

Temperature, Rectal (t_{re}): Temperature measured 10 centimeters (cm) into the rectal canal.

Temperature, Skin (t_{sk}): Temperature measured by placing the sensing element on the skin.

Temperature, Tympanic (t_{ty}): True tympanic temperature is measured by placing the sensing element directly onto the tympanic membrane and recording the temperature. Estimates of tympanic temperature are usually obtained by placing a device into the ear canal close to the tympanic membrane.

Temperature Regulation: The maintenance of body temperature within a restricted range under conditions of positive heat loads (environmental and metabolic) by physiologic and behavioral mechanisms.

Thermal Insulation, Clothing: The insulation value of a clothing ensemble.

Thermal Insulation, Effective: The insulation value of the clothing plus the still air layer.

Thermal Strain: The sum of physiologic responses of the individual to thermal stress.

Thermal Stress: The sum of the environmental and metabolic heat load imposed on the individual.

Total Heat Load: The total heat exposure of environmental plus metabolic heat.

Universal Thermal Climate Index (UTCI): This index takes into account the human thermo-physiological significance across the entire range of heat exchange and the applicability of whole-body calculations including local skin cooling; it is valid in all climates and seasons.

Wet Bulb Globe Temperature (WBGT): This is an environmental temperature arrived at by measuring dry air temperature, humidity, and radiant energy (i.e., usually direct sunlight being absorbed by clothing), used to calculate a thermal load on the person.

Wettedness, Skin ($\underline{w}$ ***):*** The amount of skin that is wet with sweat.

Wettedness, Percent of Skin: The percentage of the total body skin surface that is covered with sweat.

Work: Physical efforts performed using energy from the metabolic rate of the body.

Symbols

Symbol	Term	Units
A_b	Body surface area	m^2
A_{Du}	Body surface area, DuBois	m^2
A_r	Skin area exposed to radiation	m^2
A_w	Wetted area of skin	m^2
C	Heat exchange by convection	W, $W \cdot m^{-2}$
CO	Cardiac output of blood per minute	$L \cdot min^{-1}$
E_{max}	Maximum water vapor uptake by the air at prevailing meteorological conditions	$kg \cdot h^{-1}$
E_{req}	Amount of sweat that must be evaporated to maintain body heat balance	$kg \cdot h^{-1}$
F_{cl}	Reduction factor for loss of convective heat exchange due to clothing	dimensionless
H	Body heat content	W
h_c	Convection heat transfer coefficient	$W \cdot m^{-2} \cdot °C^{-1}$; $kcal \cdot h^{-1} \cdot m^{-2} \cdot °C^{-1}$
h_e	Evaporative heat transfer coefficient	$W \cdot m^{-2} \cdot kPa^{-1}$
HR	Heart rate	bpm
h_r	Radiative heat transfer coefficient	$W \cdot m^{-2} \cdot °C^{-1}$; $kcal \cdot h^{-1} \cdot m^{-2} \cdot °C^{-1}$
h_{r+c}	Radiative + convective heat transfer coefficient	$W \cdot m^{-2} \cdot °C^{-1}$; $kcal \cdot h^{-1} \cdot m^{-2} \cdot °C^{-1}$
I_a	Thermal insulation of still air layer	clo
I_{cl}	Thermal insulation of clothing layer	clo
i_m	Moisture permeability index of clothing	dimensionless
i_m/clo	Permeability index–insulation ratio	dimensionless
K	Heat exchanged by conduction	W, $W \cdot m^{-2}$
kcal	Kilocalories	$kcal \cdot h^{-1}$

Symbol	Term	Units
M	Metabolism	met
Met	Unit of metabolism; 1 met = 50 $kcal \cdot m^{-2} \cdot h^{-1}$	met
mmHg	Pressure in millimeters of mercury	mmHg
$m \cdot s^{-1}$	Meters per second	$m \cdot sec^{-1}$
P_a	Water vapor pressure of ambient air	mmHg, kP_a
P_{sk}	Water vapor pressure of wetted skin	mmHg, kP_a
$p_{sk,s}$	Water vapor pressure at skin temperature	mmHg, kP_a
RH	Relative humidity	percent
R	Radiant Heat exchange	W, $W \cdot m^{-2}$
S	Sweat produced	L
SR	Sweat produced per unit time	$g \cdot min^{-1}$, $g \cdot h^{-1}$, $kg \cdot min^{-1}$, $kg \cdot h^{-1}$
SV	Stroke volume, or amount of blood pumped by the heart per beat	mL
SWA	Area of skin wet with sweat	m^2
%SWA	$SWA/A_{Du} \times 100$ = % of body surface wet with sweat	percent
T	Absolute temperature (t + 273)	°K
T_a	Ambient air dry-bulb temperature	°C, °F
t_{adb}	Ambient dry-bulb temperature, adjusted for solar radiation	°C, °F
t_{cr}	Body core temperature	°C, °F
T_{dp}	Dew point temperature	°C, °F
T_g	Black globe temperature	°C, °F
T_{nwb}	Natural wet-bulb temperature	°C, °F
t_o	Operative temperature	°C, °F
t_r	Radiant temperature	°C, °F
$\bar{t}_r$	Mean radiant temperature	°C, °F
t_{re}	Rectal temperature	°C, °F

Symbol	Term	Units
t_{sk}	Skin temperature	°C, °F
$\bar{t}_{sk}$	Mean skin temperature	°C, °F
T_{pwb}	Psychrometric wet-bulb temperature	°C, °F
$\bar{t}_w$	Mean radiant temperature of the surroundings	°C,°F
t_{wg}	Wet globe temperature	°C, °F
V_a	Air velocity	$m{\cdot}s^{-1}$, fpm
$\dot{V}O_2$ max	Maximum oxygen consumption	$mL{\cdot}kg^{-1}{\cdot}min^{-1}$ (a measure of aerobic fitness) , or $L{\cdot}h^{-1}$ (a measure of total O_2 consumed at peak or maximal effort)
W	Work	$kcal{\cdot}h^{-1}$
μ	Mechanical efficiency of work	%, percent
$\underline{w}$	Skin wettedness	dimensionless
σ	Stefan-Bolzmann constant	$W{\cdot}m^{-2}{\cdot}K^{-4}$
ε	Emittance coefficient	dimensionless

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1

Recommendations for an Occupational Standard for Workers Exposed to Heat and Hot Environments

The National Institute for Occupational Safety and Health (NIOSH) recommends that worker exposure to heat stress in the workplace be controlled by complying with all sections of the recommended standard found in this document. Compliance with this recommended standard should prevent or greatly reduce the risk of adverse health effects to exposed workers. Heat-related occupational illnesses, injuries, and reduced productivity occur in situations in which the total heat load (environmental plus metabolic heat) exceeds the capacities of the body to maintain normal body functions. The reduction of adverse health effects can be accomplished by the proper application of engineering and work practice controls, worker training and acclimatization, measurements and assessment of heat stress, medical monitoring, and proper use of heat-protective clothing and personal protective equipment (PPE).

In this criteria document, total heat stress is considered to be the sum of the heat generated in the body (metabolic heat), plus the heat gained from the environment (environmental heat), minus the heat lost from the body to the environment. Environmental and/or metabolic heat stress results in physiological responses (heat strain) to promote the transfer of heat from the body back to the environment to maintain core body temperature [Parsons 2003]. Many of the bodily responses to heat exposure are desirable and beneficial. However, at some level of heat stress, a worker's compensatory mechanisms are no longer capable of maintaining body temperature at a level required for normal body functions. As a result, the risk of heat-related illnesses, disorders, and other hazards increases. The level of heat stress at which excessive heat strain will result depends on the heat tolerance capabilities of the worker. However, even though there is a wide range of heat tolerance between workers, each worker has an upper limit for heat stress, beyond which the resulting heat strain can cause the worker to become a heat casualty. In most workers, appropriate repeated exposure to elevated heat stress causes a series of physiologic adaptations called acclimatization, whereby the body becomes more efficient in coping with the heat stress. Such an acclimatized worker can tolerate a greater heat stress before a harmful level of heat strain occurs.

The occurrence of heat-related illnesses among a group of workers in a hot environment, or the recurrence of such illnesses in individual workers, represents "sentinel health events", which indicate that heat control measures, medical screening, or environmental monitoring measures may not be adequate [Rutstein et al. 1983]. One occurrence of heat-related illness in a particular worker indicates the need for medical inquiry about appropriate workplace protections. The recommendations in this document are intended to provide limits of heat stress so that workers' risks of incurring heat-related illnesses and disorders are reduced.

Almost all healthy workers who are not acclimatized to working in hot environments and who are exposed to combinations of environmental and metabolic heat less than the applicable NIOSH Recommended Alert Limits (RALs;

Figure 8-1) should be able to tolerate the heat stress (i.e., the sum of metabolic heat plus environmental heat, minus the heat lost from the body to the environment) without a substantial increase in their risk of incurring acute adverse health effects. Almost all healthy workers who are heat-acclimatized to working in hot environments and who are exposed to combinations of environmental and metabolic heat less than the applicable NIOSH Recommended Exposure Limits (RELs; Figure 8-2) should be able to tolerate the heat stress without incurring adverse effects. The estimates of both environmental and metabolic heat are expressed as 1-hour time-weighted averages (TWAs), as described by the American Conference of Governmental Industrial Hygienists (ACGIH) [ACGIH 2014]. In this criteria document, when not otherwise qualified, the term "healthy workers" refers to those who are physically and medically fit and do not require additional protection, modifications in acclimatization procedures, or additional physiological monitoring beyond the normal recommendations for the amount of heat exposure.

The medical monitoring program should be designed and implemented to minimize the risk to workers' health and safety from any heat hazards in the workplace (see Chapters 4, 5, and 6). The medical monitoring program should provide both preplacement medical evaluations for those persons who are candidates for hot jobs and periodic medical evaluations for those workers who are currently working in hot jobs.

1.1 Workplace Limits and Surveillance

1.1.1 Recommended Limits

Unacclimatized workers

Total heat exposure to workers should be controlled so that unprotected (i.e., those not wearing PPE that would provide protection against heat) healthy workers who are not acclimatized to working in hot environments are not exposed to combinations of metabolic and environmental heat greater than the applicable RALs, given in Figure 8-1.

Acclimatized workers

Total heat exposure to workers should be controlled so that unprotected healthy workers who are acclimatized to working in hot environments are not exposed to combinations of metabolic and environmental heat greater than the applicable RELs, given in Figure 8-2. For additional information on acclimatization, see 4.1.5 Acclimatization to Heat.

Effect of Clothing

The recommended limits given in Figures 8-1 and 8-2 are for healthy workers who are physically and medically fit for the level of activity required by their jobs and who are wearing the conventional one-layer work clothing ensemble consisting of not more than long-sleeved work shirts and trousers (or equivalent). The RAL and REL values given in Figures 8-1 and 8-2 may not provide adequate protection if workers wear clothing with lower air and vapor permeability or insulation values greater than those for the conventional one-layer work clothing ensemble. In addition, some workers at increased risk may need additional modifications to be protected from heat stress. A discussion of these modifications to the RALs and RELs is given in 3.3 Effects of Clothing on Heat Exchange.

1.1.2 Determination of Environmental Heat

Measurement methods

In most situations environmental heat exposures should be assessed by the Wet Bulb Globe Thermometer (WBGT) method or equivalent techniques, such as Effective Temperature

(ET), Corrected Effective Temperature (CET), or Wet Globe Temperature (WGT), which are then converted to estimated WBGT values. When air- and vapor-impermeable protective clothing is worn, the dry bulb temperature (t_a) or the adjusted dry bulb temperature (t_{adb}) is a more appropriate measurement than the WBGT, because impermeable clothing does not transfer humid heat loss but only dry heat loss (e.g., radiation, convection, and conduction) [Åstrand et al. 2003]. These temperature readings may be used to determine the degree of heat stress the worker is experiencing in the work environment and allow a qualified safety and health professional to determine how to mitigate that heat stress to prevent heat injury.

Measurement requirements

Environmental heat measurements should be made at or as close as feasible to the work area where the worker is exposed and represents the environmental heat conditions at the worker's position. When a worker is not continuously exposed in a single hot area but moves between two or more areas with differing levels of environmental heat or when the environmental heat substantially varies at the single hot area, the environmental heat exposures should be measured at each area and during each period of constant heat levels where employees are exposed. Hourly TWA WBGTs should be calculated for the combination of jobs (tasks), including all scheduled and unscheduled rest periods.

Modifications of work conditions

Environmental heat measurements should be made at least hourly, during the hottest portion of each work shift, during the hottest months of the year, and when a heat wave occurs or is predicted. If two such sequential measurements exceed the applicable RAL or REL, then work conditions should be modified by use of appropriate engineering controls, work practices, or other measures until two sequential measures are in compliance with the exposure limits of this recommended standard.

Initiation of measurements

A WBGT or individual environmental factors profile (e.g., air- and vapor-impermeable protective clothing, etc.) should be established for each hot work area, as a guide for determining when engineering controls and/or work practices or other control methods should be instituted. After the environmental profiles have been established, measurements should be made as described in this section, during the time of year and days when the profile indicates that total heat exposures above the applicable RALs or RELs may be reasonably anticipated or when a heat wave has been forecast by the nearest National Weather Service station or other competent weather forecasting service.

1.1.3 Determination of Metabolic Heat

The metabolic contribution to the heat load on the worker must be estimated or measured to ensure a safe working environment. A screening to estimate metabolic heat load should be calculated for each worker who is performing light, moderate, or heavy work. The metabolic heat rate should be determined in order to determine whether the total heat exposure exceeds the applicable RAL or REL.

Whenever the combination of measured environmental heat (WBGT) and screening estimated metabolic heat exceeds the applicable RAL or REL (Figures 8-1 and 8-2), the metabolic heat production should be measured using indirect calorimetry (see Chapter 5) or an equivalent method. Although performing indirect calorimetry in the field or on-site may not be feasible, indirect calorimetry can be performed on subjects performing at similar work levels in a laboratory setting. This information could provide an estimate of the metabolic heat production at that workload and thus support decisions regarding RAL or REL. Alternatively, the responsible individual (i.e., qualified safety and health professional) may refer to the *Compendium of Physical Activities* for information on metabolic responses to various types of work in order to determine RALs and RELs [Ainsworth et al. 2011]. For a short list of activities and the associated metabolic heat rate, see Table 1-1.

Metabolic heat rates should be expressed as kilocalories per hour ($\text{kcal}\cdot\text{h}^{-1}$) or as watts (W) for a 1-hour TWA task basis that includes all activities engaged in during each period of analysis and all scheduled and nonscheduled rest periods ($1\ \text{kcal}\cdot\text{h}^{-1} = 1.16$ W).

EXAMPLE

If a moderate-workload task was performed by an acclimatized 70-kg (154-lb) worker for the entire 60 minutes of each hour, then the screening estimate for the 1-hour TWA metabolic heat would be about 300 $\text{kcal}\cdot\text{h}^{-1}$ (348.9 W). In Figure 8-2, a vertical line at 300 $\text{kcal}\cdot\text{h}^{-1}$ (348.9 W) intersects the 60 $\text{min}\cdot\text{h}^{-1}$ REL curve at a WBGT of 27.8°C (82°F). Then, if the measured WBGT exceeds 27.8°C (82°F), the worker's metabolic heat could be measured by the indirect open-circuit method or an equivalent procedure.

If the 70-kg worker was unacclimatized, Figure 8-1 indicates that metabolic heat measurement of the worker would be required above a WBGT of 25°C (77°F).

1.1.4 Physiologic Monitoring

Physiologic monitoring may be used as an alternative to determining the required estimates and measurements described in the preceding parts of this section. The total heat stress shall be considered to exceed the applicable RAL or REL when the physiological functions exceed the values given in 9.4 Physiological Monitoring. Heart rate, core body temperature, and body water loss can be assessed as measures of physiologic response to heat. More advanced methods and new tools are also available for physiologic monitoring (see 8.4 Physiologic Monitoring of Heat Strain and 9.4 Physiologic Monitoring).

1.2 Medical Monitoring

1.2.1 General

(1) The employer should institute a medical monitoring program for all workers who are or may be exposed to heat stress above the RAL, whether they are acclimatized or not. A medical monitoring program is essential to assess and monitor workers' health and physical well-being both prior to and while working in hot environments; to provide emergency medical care or other treatment as needed and gather medical information (e.g., identify changes in health status, identify training needs for prevention efforts). More information is available in Chapter 7 Medical Monitoring.

(2) The employer should ensure that all medical evaluations and procedures are performed by or under the direction of the responsible healthcare provider (e.g., licensed physician or other licensed and/or credentialed healthcare professional).

(3) The employer should provide the required medical monitoring without cost to the workers, without loss of pay, and at a reasonable time and place.

1.2.2 Preplacement Medical Evaluations

For the purposes of the preplacement medical evaluation, all workers should be considered to be unacclimatized to hot environments. At a minimum, the preplacement medical evaluation of each prospective worker for a hot job should include the following elements:

(1) A comprehensive work and medical history. The medical history should include a comprehensive review of all body systems as would be standard for a preplacement physical examination, along with specific questions regarding previous episodes of diagnosed heat-related illness, rhabdomyolysis, and questions aimed at determining acclimatization to the new employment environment.

(2) A comprehensive physical examination should be conducted. At the discretion of the responsible healthcare provider, candidates who anticipate increased stress of physical activity of the job in a hot environment, those over 50 years of age or those younger than 50 years of age with underlying cardiac risk factors may need to have additional testing (e.g., electrocardiogram (ECG) with interpretation by a cardiologist).

(3) An assessment of the use of therapeutic drugs, over-the-counter medications, supplements, alcohol, or caffeine that may increase the risk of heat injury or illness (see Chapter 7).

(4) An assessment of obesity, defined as a body mass index (BMI) ≥ 30. Measure height and weight to calculate body mass index according to the following formula:

$$\text{BMI} = \text{weight (in pounds)} \times 703 / [\text{height (in inches)}]^2$$

(5) An assessment of the worker's ability to wear and use any protective clothing and equipment, especially respirators, that is or may be required to be worn or used.

(6) Other factors and examination details included in 7.3.1.1 Preplacement Physical Examination.

1.2.3 Periodic Medical Evaluations

Periodic medical evaluations should be made available at least annually to all workers who may be exposed at the worksite to heat stress exceeding the RAL. At minimum, the employer should provide the evaluations specified above. If circumstances warrant (e.g., an increase in job-related heat stress or changes in health status), the medical evaluation should be offered at more frequent intervals at the discretion of the responsible healthcare provider.

1.2.4 Emergency Medical Care

If the worker develops signs or symptoms of heat stroke or heat exhaustion, the employer should provide immediate emergency medical treatment (e.g., call 911 and cool down the worker). Other non-life-threatening heat-related illnesses may be treated with appropriate first aid procedures (see Table 4-3).

1.2.5 Information to Be Provided to the Responsible Healthcare Provider

The employer should provide the following information to the responsible healthcare provider performing or responsible for the medical monitoring program:

(1) A copy of this recommended standard.

(2) A description of the affected worker's duties and activities (e.g., shift schedules, work locations) as they relate to the worker's environmental and metabolic heat exposure.

(3) An estimate of the worker's potential exposure to workplace heat (both environmental

and metabolic), including any available workplace measurements or estimates.

(4) A description of any protective equipment or clothing the worker uses or may be required to use.

(5) Relevant information from previous medical evaluations of the affected worker that is not readily available to the responsible healthcare provider.

1.2.6 Responsible Healthcare Provider's Written Report of Medical Findings

The employer should obtain a written opinion from the responsible healthcare provider, which should include the following elements:

(1) Occupationally pertinent results of the medical evaluation.

(2) A medical opinion as to whether the worker has any medical conditions that would increase the health risk of exposure to heat in the work environment.

(3) An estimate of the individual's tolerance to withstand hot working conditions (see 6.2.3 Enhancing Tolerance to Heat and 6.2.5 Screening for Heat Intolerance).

(4) An opinion as to whether the worker can perform the work required by the job (i.e., physical fitness for the job).

(5) Recommendations for reducing the worker's risk for heat-related illness, which may include use of cooling measures, accommodations or limitations related to work/rest schedules and/or workload, or reassignment to another job, as warranted.

(6) A statement that the worker has been informed by the responsible healthcare provider of the results of the medical evaluation and any medical conditions that require further explanation or treatment. The worker is cleared to work in the hot environment so long as no adverse health effects occur. Specific findings, test results, or diagnoses that have no bearing on the worker's ability to work in heat or a hot environment should not be included in the report to the employer. Safeguards to protect the confidentiality of the worker's medical records should be enforced in accordance with all applicable federal and state privacy regulations and guidelines.

1.3 Surveillance of Heat-related Sentinel Health Events

1.3.1 Definition

Surveillance of heat-related sentinel health events is defined as the systematic collection and analysis of data concerning the occurrence and distribution of adverse health effects in defined populations at risk for heat injury or illness.

1.3.2 Requirements

In order to evaluate and improve prevention and control measures for heat-related effects (including the need for exposure assessment), the following should be obtained and analyzed for each workplace: (a) workplace modifications, (b) identification of highly susceptible workers, (c) data on the occurrence or recurrence in the same worker, (d) distribution in time, place, and person of heat-related adverse effects, and (e) environmental or physiologic measurements related to heat.

1.4 Posting of Hazardous Areas

1.4.1 Dangerous Heat Stress Areas

In work areas and at entrances to work areas or building enclosures where there is a reasonable likelihood of the combination(s) of environmental and metabolic heat exceeding the RAL/

REL, readily visible warning signs should be posted. These signs should contain information on the required protective clothing or equipment, hazardous effects of heat stress on human health, and information on emergency measures for heat injury or illness. This information should be arranged as follows:

DANGEROUS
HEAT STRESS AREA

HEAT STRESS–PROTECTIVE
CLOTHING OR EQUIPMENT REQUIRED

HEAT STROKE OR OTHER
HEAT-RELATED ILLNESS MAY OCCUR

1.4.2 Emergency Situations

In any area where there is a likelihood of heat stress emergency situations occurring, the warning signs required in this section should be supplemented with signs giving emergency and first aid instructions, as well as emergency contact information.

1.4.3 Additional Requirements for Warning Signs

All hazard warning signs should be printed in English and, where appropriate, in the predominant language of workers unable to read English. Workers unable to read the signs should be informed of the warning printed on the signs and the extent of the hazardous area(s). All warning signs should be kept clean and legible at all times.

1.5 Protective Clothing and Equipment

Engineering controls and safe work practices should be used to ensure that workers' exposure to heat stress is maintained at or below the applicable RAL or REL specified. In addition, protective clothing and equipment (e.g., water-cooled garments, air-cooled garments, ice-packet vests, wetted overgarments, and heat-reflective aprons or suits) should be provided by the employer to the workers when the total heat stress exceeds the RAL or REL (see 6.3 Personal Protective Clothing and Auxiliary Body Clothing).

1.6 Worker Information and Training

1.6.1 Information Requirements

All new and current workers who work in areas where there is reasonable likelihood of heat injury or illness, and their supervisors, should be kept informed, through continuing education programs, of the following:

(1) Heat stress hazards.
(2) Predisposing factors.
(3) Relevant signs and symptoms of heat injury and illness.
(4) Potential health effects of excessive heat stress.
(5) General first aid as well as worksite-specific first aid procedures.
(6) Proper precautions for work in heat stress areas.
(7) Workers' responsibilities for following proper work practices and control procedures to help protect the health and provide for the safety of themselves and their fellow workers, including instructions to immediately report to the supervisor the development of signs or symptoms of heat-related illnesses.
(8) The effects of therapeutic drugs, over-the-counter medications, alcohol, or caffeine that may increase the risk of heat injury or illness by reducing heat tolerance (see Chapter 7).

(9) The purposes for and descriptions of the environmental and medical monitoring programs and the advantages to the worker of participating in these surveillance programs.

(10) If necessary, proper use of protective clothing and equipment.

(11) Cultural attitude toward heat stress. A misperception may exist that someone can be "hardened" against the requirement for fluids when exposed to heat by deliberately becoming dehydrated before work on a regular basis. This misperception is dangerous and must be counteracted through educational efforts.

1.6.2 Training Programs

(1) The employer should institute a training program, conducted by persons qualified by experience or training in occupational safety and health, to ensure that all workers potentially exposed to heat stress and their supervisors have current knowledge of at least the information specified in this section. For each affected worker, the instructional program should include adequate verbal and/or written communication of the specified information. The employer should develop a written plan of the training program that includes a record of all instructional materials.

(2) The employer should inform all affected workers of the location of written training materials and should make these materials readily available, without cost to the affected workers.

1.6.3 Heat Stress Safety Data Sheet

(1) The information specified in this section should be recorded on a heat stress safety data sheet or on a form specified by the Occupational Safety and Health Administration (OSHA).

(2) In addition, the safety data sheet should contain:

(a) Emergency and first aid procedures, including site-specific contact information.

(b) Notes to the responsible healthcare provider regarding classification, medical aspects, and prevention of heat injury and illness. These notes should include information on the category and clinical features of each injury and illness, predisposing factors, underlying physiologic disturbance, treatment, and prevention procedures.

1.7 Control of Heat Stress

1.7.1 General Requirements

(1) The employer should establish and implement a written program to reduce exposures to or below the applicable RAL or REL by means of engineering and work practice controls.

(2) Where engineering and work practice controls are not sufficient to reduce exposures to or below the applicable RAL or REL, they should be used to reduce exposures to the lowest level achievable by these controls and should be supplemented by the use of heat-protective clothing or equipment. In addition, a heat alert program should be implemented as specified in this section.

1.7.2 Engineering Controls

(1) The type and extent of engineering controls required to bring the environmental heat below the applicable RAL or REL can be calculated with the basic heat exchange formulae (see Chapters 4 and 5). When the environmental heat exceeds the applicable RAL or REL, the following control requirements should be used.

(a) When the air temperature exceeds the skin temperature, convective heat gain should be reduced by decreasing air temperature and/or decreasing the air velocity if it exceeds 1.5 meters per second ($m \cdot sec^{-1}$) ($300\ ft \cdot min^{-1}$). When air temperature is lower than skin temperature, convective heat loss should be increased by increasing air velocity. The type, amount, and characteristics of clothing will influence heat exchange between the body and the environment.

(b) When the temperature of the surrounding solid objects exceeds skin temperature, radiative heat gain should be reduced by placing shielding or barriers that are radiant-reflecting or heat-absorbing between the heat source and the worker; by isolating the source of radiant heat; by increasing the distance to the heat source; or by modifying the hot process or operation.

(c) When necessary, evaporative heat loss should be increased by increasing air movement over the worker, by reducing the influx of moisture from steam leaks or from water on the workplace floors, or by reducing the water vapor content (humidity) of the air. The air and water vapor permeability of the clothing worn by the worker will influence the rate of heat exchange by evaporation.

1.7.3 Work and Hygienic Practices

(1) Work modifications and hygienic practices should be introduced to reduce both environmental and metabolic heat when engineering controls are not adequate or are not feasible. The most effective preventive work and hygienic practices for reducing heat stress include, but are not limited to the following:

(a) Limiting the time the worker spends each day in the hot environment by decreasing exposure time in the hot environment and/or increasing recovery time spent in a cool environment.

(b) Reducing the metabolic demands of the job by such procedures as mechanization, the use of special tools, or an increase in the number of workers per task.

(c) Increasing heat tolerance by instituting a heat acclimatization plan (see Table 4-1 Acclimatization in workers) and by increasing physical fitness.

(d) Training supervisors and workers to recognize early signs and symptoms of heat illnesses and to administer relevant first aid procedures.

(e) Implementing a buddy system in which workers are responsible for observing fellow workers for early signs and symptoms of heat intolerance, such as weakness, unsteady gait, irritability, disorientation, changes in skin color, or general malaise.

(f) Some situations may require workers to conduct self-monitoring, and a workgroup (i.e., workers, responsible healthcare profider, and safety manager) should be developed to make decisions on self-monitoring options and standard operating procedures.

(g) Providing adequate amounts of cool (i.e., less than 15°C [59°F]), potable water near the work area and encouraging all workers that have been in the heat for up to 2 hours and involved in moderate work activities to drink a cup of water (about 8 oz.) every 15 to 20 minutes. Individual, not communal, drinking cups should be provided. During prolonged sweating lasting more than 2 hours, workers should be provided with sports drinks that contain balanced electrolytes to replace those lost during sweating, as long as the concentration of electrolytes/carbohydrates does not exceed 8% by volume.

1.7.4 Heat Alert Program

A written Heat Alert Program should be developed and implemented whenever the National Weather Service or other competent weather service forecasts that a heat wave is likely to occur the following day or days. A heat wave is indicated when the daily maximum temperature exceeds 35°C (95°F) or when the daily maximum temperature exceeds 32°C (90°F) and is 5°C (9°F) or more above the maximum reached on the preceding days. More details are described in 6.2.6 Heat Alert Program.

1.8 Recordkeeping

1.8.1 Environmental and Metabolic Heat Surveillance

(1) The employer should establish and maintain an accurate record of all measurements made to determine environmental and metabolic heat exposures to workers, as required in this recommended standard (see 1.1.2 Determination of Environmental Heat).

(2) Where the employer has determined that no metabolic heat measurements are required as specified in this recommended standard, the employer should maintain a record of the screening estimates relied upon to reach the determination (see 1.1.3 Determination of Metabolic Heat).

1.8.2 Medical Surveillance

The employer should establish and maintain an accurate record for each worker subject to medical monitoring, as specified in this recommended standard (see 1.2 Medical Monitoring).

1.8.3 Surveillance of Heat-related Sentinel Health Events

The employer should establish and maintain an accurate record of the data and analyses specified in this recommended standard (see 1.3 Surveillance of Heat-related Sentinel Health Events).

1.8.4 Heat-related Illness Surveillance

The employer should establish and maintain an accurate record of any heat illness or injury and the environmental and work conditions at the time of the illness or injury (see 7.4 Medical Surveillance—Periodic Evaluation of Data).

1.8.5 Heat Stress Tolerance Augmentation

The employer should establish and maintain an accurate record of all heat stress tolerance augmentation for workers by heat acclimatization procedures (see 4.1.5 Acclimatization to Heat) and/or physical fitness enhancement.

2 Introduction

Criteria documents are developed by the National Institute for Occupational Safety and Health (NIOSH) under the authority of section 20(a) (3) of the Occupational Safety and Health Act of 1970. Through the Act, Congress charged NIOSH with recommending occupational safety and health standards and describing exposure limits that are safe for various periods of employment. These limits include, but are not limited to, the exposures at which no worker will suffer diminished health, functional capacity, or life expectancy as a result of his or her work experience. By means of criteria documents, NIOSH communicates these recommended standards to regulatory agencies (including the Occupational Safety and Health Administration [OSHA] and the Mine Safety and Health Administration [MSHA]), health professionals in academic institutions, industry, organized labor, public interest groups, and others in the occupational safety and health community. Criteria documents contain a critical review of the scientific and technical information about the prevalence of hazards, the existence of safety and health risks, and the adequacy of control methods.

In 1972 NIOSH published the *Criteria for a Recommended Standard: Occupational Exposure to Hot Environments* [NIOSH 1972], and in 1986 it published a revised criteria document [NIOSH 1986a] and a companion pamphlet, "Working in Hot Environments, Revised 1986" [NIOSH 1986b]. These publications presented the NIOSH assessment of the potential safety and health hazards encountered in hot environments, regardless of the workplace, and recommended a standard to protect workers from those hazards.

Heat-related occupational illnesses and injuries occur in situations where the total heat load (environmental and metabolic) exceeds the capacities of the body to maintain homeostasis. In the 1986 documents, NIOSH recommended sliding scale limits based on environmental and metabolic heat loads. These recommendations were based on the relevant scientific data and industry experience at that time. This criteria document reflects the most recent NIOSH evaluation of the scientific literature and supersedes the previous NIOSH criteria documents. This document presents the updated criteria and methods for recognition, evaluation, and control of occupational heat stress by engineering and preventive work practices. It also addresses the recognition, treatment, and prevention of heat-related illnesses by providing guidance for medical supervision, hygienic practices, and training programs.

The recommended criteria were developed to ensure that adherence to them will (1) protect against the risk of heat-related illnesses and heat-related reduction in safety performance, (2) be achievable by techniques that are valid and reproducible and (3) be attainable by means of existing techniques. This recommended standard is also designed to prevent harmful effects from interactions between heat and toxic chemical and physical agents. The recommended environmental limits for various intensities of physical work, as indicated in Figures 8-1 and 8-2, are not upper tolerance limits for heat exposure for all workers but, rather, levels at which engineering controls, preventive work and hygienic practices, and administrative or other control procedures should be implemented in order to reduce the risk of heat-related illnesses, even in the least heat tolerant workers.

Despite efforts to prevent heat-related deaths and illnesses, they continue. A 2008 Centers for Disease Control and Prevention (CDC) report identified 423 worker deaths among U.S. agricultural industries (16% were crop workers) and nonagricultural industries during 1992–2006. The heat-related average annual death rate for the crop workers was 0.39 per 100,000 workers, compared with 0.02 for all U.S. civilian workers [Luginbuhl et al. 2008]. Even with heat-specific workplace regulations in place in California, heat-related illnesses and deaths still occur particularily in agricultural workers who are at additional risk (e.g., extreme conditions, lack of knowledge, poverty, seasonality, low level of education, and other vulnerabilities related to migratory status) [Stoecklin-Marois et al. 2013].

In 2010, 4,190 injury or illness cases arising from exposure to environmental heat among private industry and state and local government workers resulted in one or more days of lost work [Bureau of Labor Statistics 2011]. Eighty-six percent of the heat-affected workers were aged 16–54 years. In that same year, 40 workers died from exposure to environmental heat. The largest number of workers (18) died in the construction industry, followed by 6 deaths in natural resources (including agriculture) and mining, 6 deaths in professional and business services (including waste management and remediation), and 3 deaths in manufacturing. Eighty percent of the deaths occurred among workers 25–54 years of age. Because heat-related illnesses are often not recognized, and only illnesses involving days away from work are reported, the actual number of occupational heat-related illnesses and deaths is not known. Additionally, estimates of the number of workers exposed to heat are not available.

A study of OSHA citations issued between 2012 and 2013 revealed 20 cases of heat-related illness or death of workers [Arbury et al. 2014]. In most of these cases, employers had no program to prevent heat illness, or programs were deficient; and acclimatization was the program element most commonly missing and most clearly associated with worker death.

3 Heat Balance and Heat Exchange

An essential requirement for continued normal body function is that the deep body core temperature be maintained within the range of about 37°C (98.6°F) ± 1°C (1.8°F). Achieving this body temperature equilibrium requires a constant exchange of heat between the body and the environment. The rate and amount of the heat exchange are governed by the fundamental laws of thermodynamics of heat exchange between objects. The amount of heat that must be exchanged is a function of (1) the total heat produced by the body (metabolic heat), which typically range from about 1 kcal per kilogram (kg) of body weight per hour (1.16 W) at rest to 5 $\text{kcal}\cdot\text{kg}^{-1}$ body $\text{weight}\cdot\text{h}^{-1}$ (7 W) for moderately hard industrial work, and (2) the heat gained, if any, from the environment. The rate of heat exchange with the environment is a function of air temperature and humidity, skin temperature, air velocity, evaporation of sweat, radiant temperature, and type, amount, and characteristics of the clothing worn. Respiratory heat loss is generally of minor consequence except during hard work in very dry environments. The following is a simple version of the heat balance equation.

3.1 Heat Balance Equation

The basic heat balance equation is

$$S = (M-W) \pm C \pm R \pm K - E$$

where

S = change in body heat content
(M−W) = total metabolism minus external work performed
C = convective heat exchange
R = radiative heat exchange
K = conductive heat exchange
E = evaporative heat loss

To solve the equation, measurement of metabolic heat production, air temperature, water-vapor pressure, wind velocity, and mean radiant temperature are required [Belding 1971; Ramsey 1975; Lind 1977; Grayson and Kuehn 1979; Goldman 1981; Nishi 1981; ISO 1982b; ACGIH 1985; DiBenedetto and Worobec 1985; Goldman 1985a,b; Horvath 1985; Havenith 1999; Malchaire et al. 2001].

3.2 Modes of Heat Exchange

The major modes of heat exchange between humans and the environment are convection, radiation, and evaporation. Conduction usually plays a minor role in workplace heat stress, other than for brief periods of body contact with hot tools, equipment, floors, or other items in the work environment, or for people working in water or in supine positions [Havenith 1999]. The equations for calculating heat exchange by convection, radiation, and evaporation are available in Standard International (SI) units, metric units, and English units. In SI units, heat exchange is in watts per square meter of body surface ($\text{W}\cdot\text{m}^{-2}$). The heat-exchange equations are available in metric and English units for both the seminude individual and the worker wearing a conventional long-sleeved work shirt and trousers. The values are in $\text{kcal}\cdot\text{h}^{-1}$ for the "standard man," defined as one who weighs

70 kg (154 lb) and has a body surface area of 1.8 m^2 (19.4 ft^2). For the purpose of this discussion, only SI or metric units will be used. For workers who are smaller or larger than the standard man, appropriate correction factors must be applied [Belding 1971]. The equations utilizing the SI units for heat exchange by C, R, and E are presented in Appendix A.

3.2.1 Convection (C)

The rate of convective heat exchange between the skin of a person and the ambient air immediately surrounding the skin is a function of the difference in temperature between the ambient air (t_a) and the mean weighted skin temperature ($\bar{t}_{sk}$) and the rate of air movement over the skin (V_a). This relationship is stated algebraically for the "standard man" wearing the conventional one-layer work clothing ensemble as [Belding 1971]:

$$C = 7.0\, V_a^{\,0.6}(t_a - \bar{t}_{sk})$$

where

C = convective heat exchange, $kcal \cdot h^{-1}$
V_a = air velocity in meters per second ($m \cdot sec^{-1}$)
t_a = ambient air temperature °C
$\bar{t}_{sk}$ = mean weighted skin temperature, usually assumed to be 35°C

When $t_a > 35°C$, there will be a gain in body heat from the ambient air by convection;

when $t_a < 35°C$, heat will be lost from the body to the ambient air by convection.

This basic convective heat-exchange equation in English units has been revised for the "standard man" wearing the conventional one-layer work clothing ensemble:

$$C = 0.65\, V_a^{\,0.6}(t_a - \bar{t}_{sk})$$

where

C = convective heat exchange in $Btu \cdot h^{-1}$
V_a = air velocity in feet per minute (fpm)
t_a = ambient air temperature °C (°F)
$\bar{t}_{sk}$ = mean weighted skin temperature, usually assumed to be 35°C (95°F)

3.2.2 Radiation (R)

The radiative heat exchange is primarily a function of the temperature gradient between the mean radiant temperature of the surroundings ($\bar{t}_w$) and the mean weighted skin temperature ($\bar{t}_{sk}$). Radiant heat exchange is a function of the fourth power of the absolute temperature of the solid surroundings, less the skin temperature $(T_w - T_{sk})^4$, but an acceptable approximation for the conventional one-layer clothed individual is this [Belding 1971]:

$$R = 6.6\,(\bar{t}_w - \bar{t}_{sk})$$

R = radiant heat exchange, $kcal \cdot h^{-1}$ or $W \cdot m^{-2}$
$\bar{t}_w$ = mean radiant temperature of the solid surrounding surface, °C (°F)
$\bar{t}_{sk}$ = mean weighted skin temperature

For the conventional one-layer clothed individual and English units, the equation becomes

$$R = 15.0\,(\bar{t}_w - \bar{t}_{sk})$$

R = radiant heat exchange, $Btu \cdot h^{-1}$
$\bar{t}_w$ = mean radiant temperature, °C (°F)
$\bar{t}_{sk}$ = mean weighted skin temperature

3.2.3 Evaporation (E)

The evaporation of water (sweat) from the skin surface results in a heat loss from the body. The maximum evaporative capacity (and heat loss) is a function of air motion (V_a) and the water vapor pressure difference between the ambient air (P_a) and the wetted skin at skin temperature (P_{sk}). The equation for this relationship is for the conventional one-layer clothed worker [Belding 1971]:

$$E = 14V_a^{0.6} (P_{sk} - P_a)$$

E = Evaporative heat loss, kcal·h^{-1}
V_a = air speed, m·s^{-1}
P_a = water vapor pressure of ambient air, mmHg
P_{sk} = vapor pressure of water on skin, assumed to be 42 mmHg (5.6 kP$_a$) at a 35°C (95°F) skin temperature

This translates in English units for the conventional one-layer clothed worker to:

$$E = 2.4V_a^{0.6} (P_{sk} - P_a)$$

E = Evaporative heat loss, Btu·h^{-1}
V_a = air velocity, f·min^{-1}
P_a = water vapor pressure air, mmHg
P_{sk} = water vapor pressure on the skin, assumed to be 42 mmHg (5.6 kP$_a$) at a 95°F (35°C) skin temperature

3.2.4 Conduction (K)

This type of heat exchange involves the direct transfer of heat via direct contact between two mediums (solid, liquid, or gas) that have a temperature differential. Therefore, for the purposes of this document, rate of heat transfer depends on the temperature gradient between the skin surface and the surrounding surfaces (hot metal surface, ice vest against the skin) and the thermal qualities of the surfaces (e.g., water absorbs heat at a much greater rate than air or stone) [McArdle et al. 2010a]. Under most circumstances, conduction is small in comparison with radiation, convection, and evaporative losses. However, under special circumstances such as wearing ice vests or liquid circulating personal cooling systems, conduction becomes more important. The equation for this theoretical relationship is as follows:

$$h_k = KA (T_1 - T_2) / l$$

h_k = Heat exchange via conduction
K = thermal "conductivity" determined by the physical properties of the objects
A = area for conduction of heat, m^2
l = distance between points at T_1 and T_2 for the conduction for heat, m
T_1 = temperature of the warmer object, °C
T_2 = temperature of the cooler object, °C

This equation could be rewritten to represent thermal conduction to or from skin, as follows:

$$h_k = KA (T_{skin} - T_{object}) / l$$

where

K = conductive heat exchange in kcal·h^{-1}

3.3 Effects of Clothing on Heat Exchange

Clothing serves as a barrier between the skin and the environment to protect against normal environmental elements of heat, cold, moisture, and abrasion. Special clothing has been developed to add further protection against hazardous chemical, physical, and biologic agents. A clothing ensemble will, of necessity, alter the rate and amount of heat exchange between the skin and the ambient air by convection, conduction, radiation, and sweat evaporation. Therefore, to calculate heat exchange by each of these modes, it is necessary to apply correction factors that reflect the type, amount, and characteristics of the clothing being worn when the clothing differs substantially from the conventional one-layer work clothing (i.e., more than one layer and/or greater air and vapor impermeability). This clothing efficiency factor (F_{cl}) for dry heat exchange is nondimensional [Goldman 1978; McCullough et al. 1982; Vogt et al. 1982]. In general, the thicker and greater the air and vapor impermeability of the clothing barrier layer or layers, the more it interferes with convective, radiative, and evaporative heat exchange.

Corrections of the RAL and REL have been suggested to reflect the F_{cl} based on heat transfer calculation for a variety of environmental and metabolic heat loads and three clothing ensembles [*Heat Stress Management Program for the Nuclear Power Industry: Interim Report* 1986]. The conventional one-layer clothing ensemble was used as the basis for comparisons with the other clothing ensembles. When a two-layer clothing ensemble is worn, the RAL and REL should be lowered by 2°C (3.8°F). When a partially air and/or vapor impermeable ensemble or heat reflective or protective aprons, leggings, gauntlets, etc., are worn, the RAL and REL should be lowered 4°C (7.2°F). These suggested corrections of the RAL or REL are scientific judgments that have not been substantiated by controlled laboratory studies or long-term workplace experience.

In those workplaces where a vapor and air impermeable encapsulating ensemble must be worn, the WBGT is not the appropriate measurement of environmental heat stress. In these instances, the adjusted dry bulb air temperature (t_{adb}) must be measured and used instead of the WBGT. Where the t_{adb} exceeds approximately 20°C (68°F), physiologic monitoring (core body temperature and/or pulse rate) is required. The method used for measuring core body temperature and pulse rate will be determined by the circumstances, and proper advance planning (attaching the physiological monitoring system) must take place prior to donning of the impermeable PPE. This physiologic monitoring must be conducted on a time schedule based upon metabolic heat production and t_{adb}. The suggested frequency of physiologic monitoring for moderate work varies from once every two hours at t_{adb} of 24°C (75°F) to every 15 minutes for moderate work at t_{adb} of 32°C (90°F) [NIOSH 1985].

3.3.1 Clothing Insulation and Non-evaporative Heat loss

Even without any clothing, a thin layer of still air (the boundary layer) is trapped next to the skin. This external still air film acts as a layer of insulation against heat exchange between the skin and the ambient environment. Typically, without body or air motion, this air layer (l_a) provides about 0.8 clo units of insulation. One clo unit of clothing insulation is defined as allowing 5.55 $kcal\cdot m^2\cdot h^{-1}$ of heat exchange by radiation and convection (H_{R+C}) for each °C of temperature difference between the skin (at a mean skin temperature $\bar{t}_{sk}$) and adjusted dry bulb temperature $t_{adb} = (t_a+\bar{t}_r)/2$. The mean skin temperature is the average of temperatures taken at several locations on the skin, weighted for skin area. The "weighting" system takes into consideration the amount of heat transfer over these various areas of skin. The areas of skin are typically chest, bicep, forearm, thigh, calf, and subscapular region. The weighting system used by Ramanathan [1964] is generally considered an accurate manner of determining mean skin temperature from these data. For the standard man with 1.8 m^2 of surface area, the hourly heat exchange by radiation and convection (H_{R+C}) can be estimated as

$$H_{R+C} = (10/clo)\ (\bar{t}_{sk} - t_{adb})$$

Thus, the 0.8 clo still air layer limits the heat exchange by radiation and convection for the nude standard individual to about 12.5 $kcal\cdot h^{-1}$ (14.5 W) for each °C of difference between skin temperature and air temperature. A resting individual in still air producing 90 kcal·h^{-1} (104.7 W) of metabolic heat will lose about 11 kcal·h^{-1} (12.8 W or 12%) by respiration and about the same by evaporation of the body water diffusing through the skin. The worker will then have to sweat and lose heat by evaporation to eliminate some of the remaining 68 kcal·h^{-1} (79.1 W) of metabolic heat if the t_{adb} is less than 5.5°C below t_{sk} [Goldman 1981].

The still air layer is reduced by increasing air motion, reaching a minimal value of approximately 0.2 clo at air speeds above 4.5 m·sec^{-1} (890 f·min^{-1} or 10 mph). At this wind speed, 68 kcal·h^{-1} (79.1 W) can be eliminated from the skin without sweating at an air temperature only 1.4°C below skin temperature, that is, 68/(10/0.2) = 1.4°C.

Studies of clothing materials have led to the conclusion that the insulation provided by clothing is generally a linear function of its thickness. Differences in fibers or fabric weave have only very minor effects on insulation, unless these directly affect the thickness or the vapor or air permeability of the fabric. The function of the fibers is to maintain a given thickness of still air in the fabric and block heat exchange. The fibers are more conductive than insulating; increasing fiber density (as when trying to fit two socks into a boot that has been sized to fit properly with one sock) can actually reduce the insulation provided [Goldman 1981].

The typical value for clothing insulation is 1.57 clo·cm^{-1} of thickness (4 clo·inch^{-1}). It is difficult to extend this generalization to very thin fabric layers or to garments like underwear, which may simply occupy an existing still air layer of not more than 0.5 cm thickness. These thin layers show little contribution to the intrinsic insulation of the clothing, unless there is (a) “pumping action” of the clothing layers by body motion (circulation of air through and between layers of clothing due to body movement); (b) compression of the clothing by pressure from other clothing, by objects in contact with the body, or by external wind; or (c) penetration of some of the wind (as a function of the air permeability of the outer covering fabric) into the trapped air layer [ASHRAE 1981b; Goldman 1981; McCullough et al. 1982]. Table 3-1 lists the intrinsic insulation contributed by adding each of the listed items of typical clothing ensembles. The total intrinsic insulation is not the sum of the individual items, but 80% of their total insulation value; this allows for an average loss of 20% of the sum of the individual items to account for the compression of one layer on the next. This average 20% reduction is a rough approximation, which is highly dependent on such factors as the nature of the fiber, the weave, the weight of the fabric, the use of foam or other nonfibrous layers, and the clothing fit and cut.

In summary, insulation is generally a function of the thickness of the clothing ensemble, and this, in turn, is usually a function of the number of clothing layers. Thus, each added layer of clothing, if not compressed, will increase the total insulation. That is why most two-layer protective clothing ensembles exhibit quite similar insulation characteristics and most three-layer systems are comparable, regardless of some rather major differences in fiber or fabric type [Goldman 1981].

Because clothing can significantly insulate the wearer from the external environment and trap body heat because of the insulation or low permeability, the thermal influence of the clothing has led to the development of clothing adjustment factors that can be used to determine the overall thermal stress of the wearer. An example of adjustments for the thermal properties of clothing appears in Table 3-2.

As can be seen from Table 3-2, adjustments to the total thermal stress can be made for a variety of clothing and PPE. This table is useful for determining the total heat stress

Table 3-1. Clo insulation values for typical clothing ensembles

Clothing ensemble	I_{cl}	
	Clo	M^2 °C W^{-1}
Underpants, coveralls, socks, shoes	0.70	0.11
Underpants, shirt, pants, socks, shoes	0.75	0.115
Underpants, shirt, coveralls, socks, shoes	0.80	0.125
Underpants, shirt, pants, light jacket, socks, shoes	0.85	0.135
Underpants, shirts, pants, smock, socks, shoes	0.90	0.14
Underpants, short-sleeve undershirt, shirt, pants, light jacket, socks, shoes	1.0	0.155
Underpants, short-sleeve undershirt, shirt, pants, coveralls, socks, shoes	1.1	0.17
Long underwear shirt and bottoms, heavy jacket, pants, socks, shoes	1.2	0.185
Underpants, short-sleeve undershirt, shirt, pants, light jacket, heavy jacket, socks, shoes	1.25	0.19
Underpants, short-sleeve undershirt, coveralls, heavy jacket and pants, socks, shoes	1.40	0.22
Underpants, short-sleeve undershirt, pants, light jacket, heavy jacket and pants, socks, shoes	1.55	0.225
Underpants, short-sleeve undershirt, pants, light jacket, heavy quilted outer jacket and overalls, socks, shoes	1.85	0.285
Underpants, short-sleeve undershirt, pants, jacket, heavy quilted outer jacket and overalls, socks, shoes, cap, gloves	2.0	0.31
Long underwear shirt and bottoms, heavy jacket and pants, parka with heavy quilting, overalls with heavy quilting, socks, shoes, cap, gloves	2.55	0.395

Adapted from the International Organization for Standardization (ISO) [2007].

that is imposed on the worker wearing these or similar types of clothing. More comprehensive lists are available in the literature or in ACGIH guidelines.

3.3.2 Clothing Permeability and Evaporative Heat Loss

Evaporative heat transfer through clothing tends to be affected linearly by the thickness of the ensemble. The moisture permeability index (i_m) is a dimensionless unit with a theoretical lower limit value of 0 for a vapor- and air-impermeable layer and an upper value of 1 if all the moisture that the ambient environment can take up (as a function of the ambient air vapor pressure and fabric permeability) can pass through the fabric. Since moisture vapor transfer is a diffusion process limited by the characteristic value for diffusion of moisture through still air, values of i_m approaching 1 should be found only with high wind and thin clothing. A typical i_m value for most clothing

Table 3-2. Clothing adjustment factors for types of clothing

Clothing	Clothing adjustment factors (°C–WBGT) Previous	2006
Work clothing (baseline)	0	0
Cloth coveralls	3.5	0
Double-layer cloth clothing	5	3
Spunbound melt-blown synthetic (SMS) coveralls	–	0.5
Polyolefin coveralls	–	1
Limited-use vapor-barrier coveralls	–	11

Adapted from Bernard TE, Threshold Limit Values for Physical Agents Committee, ACGIH [2014].

materials in still air is less than 0.5 (e.g., i_m will range from 0.45 to 0.48). Water repellent treatment, very tight weaves, and chemical protective impregnations can reduce the i_m value significantly. However, even impermeable layers seldom reduce the i_m value to zero, since an internal evaporation-condensation cycle is set up between the skin surface and the inner surface of the impermeable layer, which effectively transfers some heat from the skin to the vapor barrier. This shunting, by passing heat across the intervening insulation layers, can be reflected as an i_m value of about 0.08, even for a totally impermeable overgarment.

Very few fiber treatments have been found to improve the i_m index value of fabric layers; surfactants, which increase the number of free hydroxyl (OH) radicals on the fiber surface or which somehow improve wicking, appear to increase the i_m value of a fabric. However, the ultimate evaporative heat transferred from the skin through the clothing and external air layers to the environment is not simply a function of the i_m but a function of the permeability index–insulation ratio (i_m/clo). The maximum evaporative heat exchange with the environment can be estimated for the H_{R+C} of a "standard man" with 1.8 m^2 of surface area, as:

$$HE_{max}=10i_m/clo \times 2.2(P_{sk}-P_a)$$

The constant 2.2 is the Lewis number; P_{sk} is the water vapor pressure of sweat (water) at skin temperature (t_{sk}); and P_a is the water vapor pressure of the ambient air at air temperature, t_a. Thus, the maximum evaporative transfer tends to be a linear, inverse function of insulation, if not further degraded by various protective treatments, which range from total impermeability to water repellent treatments [Goldman 1973, 1981, 1985a]. The Lewis number is derived from fraction of skin wettedness, latent heat, and evaporative heat transfer (h_e).

3.3.3 Physiologic Problems of Clothing

The percent of sweat-wetted skin surface area ($\underline{w}$) needed to eliminate the required amount of heat from the body by evaporation can be estimated simply as the ratio of the required evaporative cooling (E_{req}) and the maximum water vapor uptake capacity of the ambient air (E_{max}). A totally wetted skin = 100%.

$$\underline{w} = E_{req} / E_{max}$$

Having some sweat-wetted skin is not uncomfortable; in fact, some sweating during exercise in heat increases comfort. As the extent of skin wetted with sweat approaches 20%, the sensation of discomfort begins to be noted. Discomfort is marked and performance decrements can appear with between 20% and 40% wetting of the body surface; performance decrements become increasingly noted as $\underline{w}$ approaches 60%. Sweat begins to be wasted, dripping rather than evaporating at 70%; physiologic strain becomes marked between 60% and 80% $\underline{w}$. Increases of $\underline{w}$ above 80% result in limited tolerance, even for physically fit, heat-acclimatized young workers. The above arguments indicate that any protective work clothing will pose some limitations on tolerance, since with I_a plus I_{clo} rarely below 2.5 clo, their i_m/clo ratios are rarely above 0.20 [Goldman 1985b].

The physiologic strain that arises with wearing clothing, heat transfer, and work can be estimated from equations that describe the competition for the blood pumped by the heart. The cardiac output (CO) is the stroke volume (SV) (or volume of blood pumped per beat) multiplied by heart rate (HR) in beats per minute (CO = SV × HR). The cardiac output increases essentially linearly with increasing work; the rate-limiting process for metabolism is the maximum rate of delivery of oxygen to the working muscle via the blood supply. It is expressed in liters per minute ($L \cdot min^{-1}$). In heat stress, this total blood supply must be divided between the working muscles and the skin where the heat exchange occurs. In this consideration, it is important to realize that not all of the CO is diverted to muscle and skin. Other organ systems must receive oxygenated blood during exercise as well, especially the brain, heart, and viscera [McArdle et al. 2010a].

SV rapidly reaches a constant value for a given intensity of work. Thus, the work intensity (i.e., the rate of oxygen delivered to the working muscles) is essentially indicated by HR; the individual worker's maximum HR limits the ability to continue work. Conditions that impair the return of blood from the peripheral circulation to fill the heart between beats will affect work capacity. The maximum achievable HR is a function of age and can be roughly estimated by this relationship: 220 beats per minute (bpm) minus age in years [Hellon and Lind 1958; Drinkwater and Horvath 1979]. Given equivalent HR at rest (e.g., 60 bpm), a 20-year-old worker's HR has the capacity to increase by 140 bpm, i.e., (220–20)–60, while a 60-year-old worker can increase his HR only 100 bpm, i.e., (220–60)–60. Because the demands of a specific task will be roughly the same for 20- and 60-year-old individuals who weigh the same and do the same amount of physical work, the decline in maximum achievable HR with age increases both the perceived and the actual relative physiologic strain of work on the older worker. Given the above, it is preferable to use a percent of the person's HR_{max} rather than refer to a specific absolute HR as a target for work.

The ability to transfer the heat produced by muscle activity from the core of the body to the skin is also a function of the CO. Blood passing through core body tissues is warmed by heat from metabolism during rest and work. The basic requirement is that skin temperature (t_{sk}) must be maintained at least 1°C (1.8°F) below deep body temperature (t_{re}) if blood that reaches the skin is to be cooled before returning to the body core. The heat transferred to the skin is limited, ultimately, by the CO and by the extent to which t_{sk} can be maintained below t_{re}.

A worker's t_{re} is a function of metabolic heat production (M) ($t_{re} = 36.7 + 0.004M$), as long as there are no restrictions on evaporative and convective heat loss by clothing, high ambient vapor pressures, or very low air motion; for example, at rest, if M = 105W, t_{re} is about 37.1°C (98.8°F). Normally, under the same conditions of unlimited evaporation, skin temperatures are below t_{re} by about 3.3°C + (0.006M); thus, at rest, when t_{re} is 37°C, the corresponding t_{sk} is about 33°C, that is, 37–(3.3 + 0.6). This 3° C–4°C difference between t_{re} and t_{sk} indicates that, at rest, each liter of blood flowing from the deep body to the skin can transfer approximately 4.6 watts or 4 kcal of heat to the skin. Since t_{re} increases and t_{sk} decreases from the evaporation of sweat with increasing M, it normally becomes easier to eliminate body heat with increasing work, since the difference between t_{re} and t_{sk} increases by about 1°C (1.8°F) per 100 watts (86 kcal) of increase in M (i.e., t_{re} up 0.4°C (0.7°F), and t_{sk} down 0.6°C (1.1°F) per 100 watts of M). Thus, at sustainable hard work (M = 500 watts or 430 $kcal \cdot h^{-1}$), each liter of blood flowing from core to skin can transfer 9 kcal (10.5 W) to the skin, which is 2.5 times that at rest [Goldman 1973, 1985a].

Work under a heat stress condition sets up a competition for CO, particularly as the blood vessels in the skin dilate to their maximum and less blood is returned to the central circulation. Gradually, less blood is available in the venous return to fully fill the heart between beats, causing the SV to decrease; therefore, HR must increase to maintain the same CO. For fit, young workers, the average work HR should be limited to about 110 bpm if an 8-hour work shift is to be completed; an average HR of 140 bpm should be maintained for no more than 4 hours, and an average HR of 160 bpm should be maintained for no more than 2 hours [Brouha 1960]. If the intensity of work results in a HR in excess of these values, then the intensity of work should be reduced. Thus, heat added to the demands of work rapidly results in problems, even in healthy, young workers. These problems are amplified if circulating blood volume is reduced as a result of inadequate water intake to replace sweat losses, which can average one liter an hour over an 8-hour work shift, or by vomiting, diarrhea, or diuresis.

The crisis point, heat exhaustion and collapse, is a manifestation of inadequate blood supply to the brain; this occurs when CO becomes inadequate because of insufficient return of blood from the periphery to fill the heart for each beat or because of inadequate time between beats to fill the heart as HR approaches its maximum.

Clothing interferes with heat loss from the skin, and skin temperature rises predictably with increased clothing. Because of the insulation-induced rise in t_{sk} and the resultant limited ability to dissipate heat that has been transferred from the core to the skin, core temperature (t_{re}) also rises when clothing is worn. Another type of interference with heat loss from the skin arises when sweat evaporation is required for body cooling (i.e., when M + HR + C > O) but is limited by high ambient water vapor pressure, low wind, or low clothing permeability index (i_m/clo).

As E_{req} approaches E_{max}, skin temperature increases dramatically and deep body temperature begins to increase rapidly. Deep body temperatures above 38.0°C (100.4°F) are considered undesirable for an average worker. The risk of heat-exhaustion collapse is about 25% at a deep body temperature of 39.2°C (102.6°F), associated with a skin temperature of 38°C (100.4°F) (i.e., t_{sk} converging toward t_{re} and approaching the 1°C [1.8°F] limiting difference where one liter of blood can transfer only 1 or 2 $kcal \cdot h^{-1}$ [1.16 W or 2.33 W] to the skin). At a similarly elevated t_{sk} where t_{re} is 39.5°C (103.1°F), there is an even greater risk of heat-exhaustion collapse, and as t_{re} approaches 40°C (104°F), with elevated skin temperatures, most individuals are in imminent danger of heat-related illness. Finally, t_{re} levels above 41°C (105.8°F) are associated with heat stroke, a life-threatening major medical emergency. The competition for CO is exacerbated by dehydration (limited SV), age (limited maximum HR), and reduced physical fitness (compromised CO). These work-limiting and potentially serious deep body temperatures are reached more rapidly when combinations of these three factors are involved.

As indicated in the above statements, maximum work output may be substantially degraded by almost any protective clothing worn during either heavy work in moderately cool environments or low work intensities in hot conditions, because of the clothing interfering with heat elimination. Heat stress is also likely to be increased with any two-layer protective ensembles or any effective single-layer vapor barrier system for protection against toxic products, unless some form of auxiliary cooling is provided [Goldman 1973, 1985a].

4 Biologic Effects of Heat

4.1 Physiologic Responses to Heat

4.1.1 The Central Nervous System

The central nervous system is responsible for the integrated organization of thermoregulation. The hypothalamus is thought to be the central nervous system's primary seat of control. Historically, in general terms, the anterior hypothalamus has been considered to function as an integrator and "thermostat," whereas the posterior hypothalamus provides a "set point" of the core or deep-body temperature and initiates the appropriate physiologic responses to keep the body temperature at that set point if the core temperature changes.

According to this model, the anterior hypothalamus receives the information from receptors sensitive to changes in temperature in the skin, muscle, stomach, other central nervous system tissues, and elsewhere. In addition, the anterior hypothalamus itself contains neurons that are responsive to changes in temperature of the arterial blood serving the region. The neurons responsible for the transmission of the temperature information use monoamines, among other neurotransmitters; this has been demonstrated in animals [Cooper et al. 1982]. These monoamine transmitters are important in the passage of appropriate information to the posterior hypothalamus. It is known that the set point in the posterior hypothalamus is regulated by ionic exchanges. However, the set-point hypothesis has generated considerable controversy [Greenleaf 1979]. The problem with the notion of a set point is that (1) a neuroanatomical region controlling the set point has never been identified and (2) the physiological responses to heat cannot be explained by the notion of a set point. At present, it appears that the hypothalamic region does integrate neural traffic from thermoreceptors and integrates a physiological response to an increase in temperature. However, the current data suggest that the hypothalamus controls temperature within a so-called inter-thermal threshold (range of temperatures around a mean with which no physiological response occurs). The physiological responses occur only when the temperature moves beyond the "thresholds" to elicit either sweating or thermogenesis and the appropriate vasomotor response (i.e., vasoconstriction or vasodilation) [Mekjavic and Eiken 2006]. The ratio of sodium to calcium ions is also important in thermoregulation. The sodium ion concentration in the blood and other tissues can be readily altered by exercise and by exposure to heat.

When a train of neural traffic is activated from the anterior to the posterior hypothalamus, it is reasonable to suppose that once a "hot" pathway is activated, it will inhibit the function of the "cold" pathway and vice versa. However, there is a multiplicity of neural inputs at all levels in the central nervous system, and many complicated neural "loops" undoubtedly exist.

Current research suggests that, instead of the historical notion of a set point, neural input into the hypothalamus is integrated into a response that can be described as "cross inhibitory." In other words, when neural inputs from warm thermoreceptors in the skin are dominant, the integrated response results in an increase in sweating and cutaneous vasodilation, while simultaneously inhibiting thermogenesis and vice versa [Mekjavic and Eiken 2006]. Paradoxically, when appropriate, the core body temperature will increase and be regulated at a higher level in order to maintain an increase in heat loss, by maintaining a thermal gradient between the core body temperature and the skin temperature for the transfer of heat to the environment [Taylor et al. 2008]. Although this discussion focuses on the role of the anterior hypothalamus in heat stress and injury, there are many other factors that influence the anterior hypothalamus and the control of heat balance. These factors include thyroid and reproductive hormones, cerebrospinal fluid (CSF) ions and osmolality, glucose, and input from other brain regions related to arousal, circadian variation, and menstrual function [Kandel and Schwartz 2013].

A question that must be addressed is the difference between a physiologically raised body temperature and a fever; it is considered that the set point is elevated, as determined by the posterior hypothalamus. At the onset of a fever, the body invokes heat-conservation mechanisms (such as shivering and cutaneous vasoconstriction) in order to raise the body temperature to its new, regulated stable temperature [Cooper et al. 1982]. In contrast, during exercise in heat, which may result in an increase in body temperature, body temperature rises to a new stable level where it is regulated by the hypothalamus, and only heat-dissipation mechanisms are invoked. Once a fever is induced, the elevated body temperature appears to be normally controlled by the usual physiologic processes around its new and higher regulated level [Taylor et al. 2008].

4.1.2 Muscular Activity and Work Capacity

The muscles are by far the largest single group of tissues in the body, representing some 45% of body weight. The bony skeleton, on which the muscles operate to generate their forces, represents a further 15% of body weight. The bony skeleton is relatively inert in terms of metabolic heat production. Even at rest, the muscles produce about 20% to 25% of the body's total heat production [Rowell 1993]. The amount of metabolic heat produced at rest is quite similar for all individuals when it is expressed per unit of surface area or of lean or fat-free body weight. On the other hand, the heat produced by the muscles during exercise can be much higher and must be dissipated if a heat balance is to be maintained. The heat load from metabolism is therefore widely variable, and working in hot environments (which imposes its own heat load or restricts heat dissipation) poses the greatest challenge to normal thermoregulation [Parsons 2003].

The proportion of maximal aerobic capacity ($\dot{V}O_2$ max) needed to do a specific job is important for several reasons. First, the cardiovascular system must respond with an increased CO, which, at levels of work up to about 40% VO_2 max, is brought about by an increase in both SV and HR. When maximum SV is reached, additional increases in CO can be achieved solely by increased HR until maximal HR is reached [McArdle et al. 1996b; Taylor et al. 2008]. These changes in the cardiovascular response to exercise are responsible for providing sufficient blood flow to muscle to allow for the increase in muscular work [McArdle et al. 1996b]. Further complexities arise when high work intensities are sustained for long periods, particularly when work is carried out in hot surroundings [Åstrand et al. 2003]. Second, muscular activity is associated with an increase in muscle temperature, which then is associated with an increase in core temperature, with attendant influences on the thermoregulatory controls. Third, at high levels of exercise, even in a temperate environment, the oxygen supply to the tissues may be insufficient to completely meet the oxygen needs of the working muscles [Taylor et al. 2008].

In warmer conditions, an adequate supply of oxygen to the tissues may become a problem even at moderate work intensities because of competition for blood distribution between the working muscle and the skin [Rowell 1993]. Because of the lack of oxygen, the working muscles must begin to draw on their anaerobic reserves, deriving energy from the oxidation of glycogen in the muscles [McArdle et al. 1996b]. That leads to the accumulation of lactic acid, which may be associated with the development of muscular fatigue. As the proportion of $\dot{V}O_2$ max used increases further, anaerobic metabolism assumes a relatively greater proportion of the total muscular metabolism. An oxygen "debt" occurs when oxygen is required to metabolize the lactic acid that accumulates in the muscles. This debt must be repaid during the rest period. In hot environments, the recovery period is prolonged as both the heat and the lactic acid stored in the body must be eliminated and water loss must be replenished. Generally, a euhydrated (normal hydration) state can be reached readily with normal ad libitum consumption of nonalcoholic beverages [Montain and Cheuvront 2008]. However, if a person consumes alcoholic beverages after a long day of hot work, the diuretic properties of alcohol may cause dehydration [Schuckit 2011]. This may delay the return to a euhydrated condition until the following day. There are also so-called alactate (absence of lactic acid accumulation) components to the oxygen debt and recovery process. Some of these alactate components involve blood returning to the lungs after distribution to the muscle, release of oxygen bound to myoglobin, residual effects of thermogenic hormones (epinephrine, norepinephrine, thyroxine, and glucocorticoids), and replenishment of adenosine triphosphate (ATP) and creatine phosphate (PCr) within the muscle cells [McArdle et al. 2010a].

It is well established that in a wide range of cool to warm environments, 5°C to 29°C (41°F–84.2°F), the deep body temperature rises during exercise to a similar equilibrium value in subjects exercising at the same proportion of $\dot{V}O_2$ max [Lind 1976, 1977]. However, two individuals doing the same job and working at the same absolute load level who have widely different $\dot{V}O_2$ max values will have quite different core temperatures. Current recommendations for an acceptable proportion of $\dot{V}O_2$ max for daily industrial work vary from 30% to 40% of the $\dot{V}O_2$ max, which, in comfortably cool surroundings [Åstrand et al. 2003], is associated with rectal temperatures of, respectively, 37.4°C to 37.7°C (99.3°F–99.9°F), whereas work at 50% $\dot{V}O_2$ max yields a rectal temperature of 38°C (100.4°F) in the absence of heat stress.

In addition to sex- and age-related variability, the interindividual variability of $\dot{V}O_2$ max is high; in a diverse worker population the range of $\dot{V}O_2$ max that includes 95 of every 100 individuals is ±20% of the mean $\dot{V}O_2$ max value. Differences in body weight (particularly the muscle mass) can account for about half that variability, but the source of the remaining variation has not been identified. Age is associated with a reduction in $\dot{V}O_2$ max after its peak at about 20 years of age; in healthy individuals, the $\dot{V}O_2$ max falls by nearly 10% each decade after age 30. The decrease with age is less in individuals who have maintained a higher degree of physical fitness. Women's $\dot{V}O_2$ max average about 70% of those for men in the same age group, because of lower absolute muscle mass, higher body fat content, and lower hemoglobin concentration [Åstrand and Rodahl 1977; Åstrand et al. 2003]. Many factors affect deep body temperature when men and women of varying body weights, ages, and work capacities do the same job.

Other sources of variability when individuals work in hot environments are differences in circulatory system capacity, sweat production, and ability to regulate electrolyte balance, each of which may be large.

Work capacity is reduced to a limited extent in hot surroundings if body temperature is elevated. That reduction becomes greater as body temperature increases. The $\dot{V}O_2$ max is not reduced by dehydration itself (except for severe dehydration), so its reduction in hot environments seems to be principally a function of body temperature. Core temperature must be above 38°C (100.4°F) before a reduction is noticeable; however, a rectal temperature of about 39°C (102.2°F) may result in some reduction of $\dot{V}O_2$ max.

The capacity for prolonged exercise of moderate intensity in hot environments is adversely affected by dehydration, which may be associated with a reduction of sweat production and a concomitant rise in rectal temperature and HR. If the total heat load and the sweat rate are high, it is increasingly more difficult to replace the water lost in the sweat (750–1,000 $mL \cdot h^{-1}$). The thirst mechanism is usually not strong enough to drive one to drink the large quantities of water needed to replace the water lost in the sweat [DOD 2003]. Evidence shows that as body temperature increases in a hot working environment, endurance decreases.

Cognitive function of individuals exposed to physical activity in a hot environment may increase, decrease, or change very little [O'Neal and Bishop 2010]. If cognitive function is impaired as the environmental heat stress increases, psychomotor, vigilance, and other experimental psychological tasks may show decrements in performance [Givoni and Rim 1962; Ramsey and Morrissey 1978; Hancock 1981, 1982; Marg 1983]. The decrement in performance may be at least partly related to increases in core temperature and dehydration. In some cases of heat exhaustion the rectal temperature is raised to the range of 38.5°C to 39.0°C (101.3°F–102.2°F), and disorganized central nervous system activity, as evidenced by poor motor function, confusion, increased irritability, blurring of vision, and changes in personality are present. These observations have prompted the unproven suggestion that reduced oxygen supply to the brain, called cerebral anoxia may be responsible [Macpherson 1960; Leithead and Lind 1964; Hancock 1982].

4.1.3 Circulatory Regulation

The autonomic nervous system and endocrine system control the allocation of blood flow among competing organ systems. The circulatory system delivers oxygen and nutrients to all tissues and for transports unwanted metabolites and heat from the tissues. However, as work in heat continues, the heart reaches a point where it cannot provide enough CO to meet both the peak needs of all of the body's organ systems and the need for dissipation of body heat.

During exercise, widespread, sympathetic circulatory vasoconstriction occurs initially throughout the body, even in the cutaneous bed. The increase in blood supply to the active muscles is ensured by the action of locally produced vasodilator substances, which also inhibit (in the blood vessels supplying the active muscles) the increased sympathetic vasoconstrictor activity. In inactive vascular beds, there is a progressive vasoconstriction with the severity of the exercise. This is particularly important in the large vascular bed in the digestive organs, where venoconstriction also permits the return of blood sequestered in its large venous bed, allowing up to one liter of blood to be added to the circulating volume [Rowell 1977, 1993].

If the need to dissipate heat arises, the autonomic nervous system reduces the vasoconstrictor tone of the cutaneous vascular bed, followed by "active" dilation by an unknown mechanism. The sweating mechanism and an unknown critical factor that causes the importantly large dilation of the peripheral blood vessels in the skin are mutually responsible for humans' large thermoregulatory capacity in the heat.

When individuals are exposed to continuous work at high proportions of $\dot{V}O_2$ max or to continuous work at lower intensities in hot surroundings, the cardiac filling pressure remains relatively constant, but the central venous blood volume decreases as the cutaneous vessels dilate. The SV falls gradually and the HR must increase to maintain the CO. The effective circulatory volume also decreases, partly because of dehydration from sweating, and partly as the thermoregulatory system tries to maintain adequate circulation to the exercising muscles and the skin [Rowell 1977].

One of the most important roles of the cardiovascular system in thermoregulation is the circulation of warm blood from the body core to the skin for heat transfer to the environment. When the body is at rest, in the absence of heat strain, the skin blood flow is approximately 200 to 500 $mL{\cdot}min^{-1}$, but it can increase up to 7–8 $L{\cdot}min^{-1}$ when under high heat strain. The skin blood flow responds to changes in body core temperature and is required to mobilize warm blood from the body core to the periphery to transfer heat to the environment [Taylor et al. 2008]. However, the redistribution of blood flow to the skin results in a corresponding decrease in splanchnic, renal, and muscle blood flow. During exercise in the heat, blood is simultaneously required to supply oxygen to working muscle and to carry heat from the body core to the periphery (skin). However, muscle blood flow decreases by about 25% under heat strain, from approximately 2.4 $mL{\cdot}g^{-1}{\cdot}min^{-1}$ down to 2.1 $mL{\cdot}g^{-1}{\cdot}min^{-1}$, because of increased cutaneous demands [Taylor et al. 2008]. This is readily accomplished in a well-hydrated individual under compensable heat stress [González-Alonzo et al. 2008; Taylor et al. 2008]. In the dehydrated individual working in the heat, increased core body temperature imposes stress on the cardiovascular system, as well as the thermoregulatory system in the hypothalamus [Taylor et al. 2008]. The mechanism of redistribution of blood to muscle and to the cutaneous circulation, under conditions of dehydration secondary to sweating, leads to an effective contraction of plasma volume [González-Alonzo et al. 2008]. A decrease in an effective plasma volume can result in an increase in heart rate and myocardial oxygen demand [Parsons 2003]. During heat stress at both rest and exercise, the HR, CO, and SV all increase to a greater extent at a given workload than would normally be observed under thermoneutral conditions [Rowell 1993]. However, this cannot be sustained indefinitely or when the body is dehydrated from sweating or substantial blood flow is redistributed to the cutaneous circulation because they effectively reduce pressure volume and, therefore, SV and CO [Taylor et al. 2008].

4.1.4 The Sweating Mechanism

In a hot environment, where heat transfer by radiation is not possible, the primary means for the transfer of heat to the environment is evaporative heat loss through the vaporization of sweat from the skin. The sweat glands are found in abundance in the outer layers of the skin. They are stimulated by cholinergic sympathetic nerves and secrete a hypotonic watery solution onto the surface of the skin. Several other mechanisms for heat transfer to the environment include convection, conduction, and behavioral (e.g., leave the area, put on or take off clothes, drink water, or modify environmental controls) [Taylor et al. 2008]. In a hot environment that has an ambient wet-bulb temperature of 35°C (95°F) the body at rest can sweat at a rate that results in a body fluid loss of 0.8 to 1.0 $L \cdot h^{-1}$. For every liter of water that evaporates, 2,436 kJ (580 kcal) are extracted from the body and transferred to the environment [McArdle et al. 1996a]. The enormous capacity for heat loss through evaporation is generally more than adequate to dissipate metabolic heat generated by a subject at rest (~315 $kJ \cdot h^{-1}$for a 75-kg man) and at high levels of activity. The mean sweat rate in endurance athletes ranges from 1.5 to 2.0 $L \cdot h^{-1}$, which provides an evaporative heat loss capacity of 3,654 to 4,872 kJ that is about 11.6–15.5 times the amount of heat produced at rest [Gisolfi 2000]. This is generally more than adequate to remove heat from the body, even at extreme levels of metabolic heat production.

However, in environments with high humidity, even though sweating continues (increasing the level of dehydration), the evaporation of sweat is inhibited, heat transfer from the body is reduced, and the internal body temperature increases. Thus, when the heat index is greater than 35°C (95°F), largely due to high relative humidity (RH) (or with a WBGT of 33°C to 25°C [91.4°F to 77°F], depending on the workload), the evaporative heat loss is virtually nonexistent. Consequently, even if the ambient dry temperature is within a comfortable range (e.g., 23°C [73.4°F]), the high humidity could result in an "apparent temperature" or heat index high enough to cause heat stress for the worker and possible heat injury [Taylor et al. 2008].

Sweating results in significant dehydration, which leads to thermal and cardiovascular strain. People who are acclimatized to the heat lose water at a peak rate of 3 $L \cdot h^{-1}$ through sweating and may lose up to 12 $L \cdot h^{-1}$ during intense exercise in hot environments [McArdle et al. 1996a]. Thus, a major issue resulting from high heat stress is the need for adequate rehydration in order to replace the water lost to the environment from sweating and to reduce the risk of hyperthermia.

A rule of thumb is that a 0.45-kg (1.0-lb) decrease in body weight represents a 450-mL (15.2-oz) decrease in body water in extracellular and intracellular compartments that needs to be replaced by consumption of water. Another source of body water loss is the respiratory tract [McArdle et al. 1996a]. Average water loss from the respiratory tract at rest is about 350 mL per day under mild conditions of heat and humidity. Respiratory water loss will also contribute to the dehydration of the worker, and fluid loss increases with activity.

An important constituent of sweat is salt, or sodium chloride. In most circumstances in the United States, a salt deficit does not readily occur because the normal American diet provides 4 grams per day (174 mEq per day) of sodium [Food and Nutrition Board, Institute of Medicine 2004]. However, the sodium content of sweat in unacclimatized individuals may range from 10 to 70 mEq per day sweat (0.23–1.62 $g \cdot L^{-1}$) [Montain and Cheuvront 2008], whereas for the acclimatized individual, sodium lost to sweat may be reduced to 23 $mEq \cdot L^{-1}$ (0.530 $g \cdot L^{-1}$), less than 50% of that of the unacclimatized individual. It is possible for a heat-unacclimatized individual who consumes a restricted salt diet to develop a negative salt balance. In theory, a prolonged negative salt balance with a large fluid intake may result in a need for moderate supplementation of dietary salt. If there is a continuing negative salt balance, acclimatization to heat is diminished. However, salt supplementation of the normal diet is rarely required, except possibly for heat-unacclimatized individuals during the first 2 or 3 days of heat exposure [Lind 1976; DOD 2003]. By the end of the third day of heat exposure, a significant amount of heat acclimatization will have occurred, and salt loss in the sweat and urine and the need for salt in the diet are decreased. In view of the high incidence of elevated blood pressure in the U.S. worker population and the relatively high salt content of the average U.S. diet, even for those who watch salt intake, recommending an increase in salt intake is probably not warranted. Salt tablets can irritate the stomach and should not be used [DOD 1980; 2003]. Heavier use of salt at meals has been suggested for heat-unacclimatized workers during the first 2 or 3 days of heat exposure if they are not on a restricted salt diet by order of the responsible healthcare provider [DOD 2003]. Sodium can also be replenished by drinking fluids containing approximately 20 $mEq \cdot L^{-1}$ sodium, which is an amount found in many sports drinks [Montain and Cheuvront 2008]. In general, simply adding salt to the diet will adequately restore electrolyte balance. Moreover, carefully induced heat acclimatization reduces or eliminates the need for salt supplementation of the normal diet.

Because potassium is lost in sweat, it can be substantially depleted when unacclimatized workers suddenly have to work hard in hot climates; marked depletion of potassium can lead to serious physiologic consequences, including the development of heat stroke [Leithead and Lind 1964]. High table salt intake may increase potassium loss. However, potassium loss is usually not a problem, except for individuals taking diuretics, because potassium is present in most foods, particularly meats and fruits [Greenleaf and Harrison 1986]. Since some diuretics cause potassium loss, workers taking such medication while working in a hot environment should seek medical advice, telling their doctor that they work in the heat and asking how medications may affect how their body responds to the hot environment. Salt and potassium supplements, if recommended by the responsible healthcare provider, may only be needed during the acclimatization period or under extraordinary circumstances.

4.1.4.1 Water and Electrolyte Balance and the Influence of Endocrines

It is imperative to replace the water lost in the sweat. It is not uncommon for workers to lose 6 to 8 L of sweat during a working shift in hot industries. If the lost water is not replaced, then body water levels will progressively decrease, which shrinks the extracellular space, interstitial and plasma volumes, and water in the cells. Evidence supports that the amount of sweat production depends on the state of hydration [Leithead and Lind 1964; Henschel 1971; Greenleaf and Harrison 1986], so that progressive dehydration results in a lower sweat production and a corresponding increase in body temperature, which can be a dangerous situation.

Water lost in large quantities of sweat is often difficult to replace completely as the day's work proceeds, and it is not uncommon for individuals to register a water deficit of 2% to 3% or greater of their body weight. During exercise in either cool or hot environments, a correlation has been reported between the elevation of rectal temperature and the percentage of water deficit in excess of 3% of body weight [Kerslake 1972 (p. 316)]. Because the normal thirst mechanism is not sensitive enough to ensure a sufficient water intake [Greenleaf and Harrison 1986; DOD 2003], every effort should be made to encourage individuals to drink water or other fluids (e.g., sports drinks). The fluid should be as palatable as possible, at less than 15°C (59°F). Small quantities taken at frequent intervals is a more effective regimen for practical fluid replacement than the intake of large amounts of fluids per hour [McArdle et al. 2010b]. Individual, not communal, drinking cups should be provided. Individuals are seldom aware of just how much sweat they produce or how much water is needed to replace that lost in the sweat; 1 $L \cdot h^{-1}$ is a common rate of water loss. With suitable instruction on how much to drink, most individuals will comply. A general rule of thumb for those exercising in the heat for 1 to 2 hours is to drink plain, cool water. Sweat is hypotonic to the plasma, and one does not lose a significant amount of sodium in the first hour or two of exercise [McArdle et al. 1996b]. Therefore, one does not require fluids containing electrolytes for this exposure. However, during prolonged sweating lasting several hours, it is advisable to consume a sports drink that contains balanced electrolytes to replace those lost during sweating, as long as the concentration of electrolytes/carbohydrates does not exceed 8% by volume. Exceeding the 8% limit will slow absorption of fluids from the gastrointestinal (GI) tract [Parsons 2003]. Since thirst is a poor indicator of hydration status, fluids should be consumed at regular intervals to replace water lost from sweating [McArdle et al. 1996b].

Plasma electrolytes (primarily sodium, potassium, and chlorine) are very strongly regulated by physiologic mechanisms because of their high importance in cellular volume and function. The control is accomplished by the kidney and is influenced by several hormonal pathways, including the renin-angiotensin-aldosterone system (RAAS) as well as antidiuretic hormone (ADH). Normal plasma sodium (Na^+) concentration falls within the range of 135 to 145 $mmol \cdot L^{-1}$. Thus, hyponatremia is defined as a Na^+ concentration <135 $mmol \cdot L^{-1}$ (mild) and severe hyponatremia as a plasma Na^+ concentration of 120 $mmol \cdot L^{-1}$. Hyponatremia (low plasma sodium) is regarded as the consequence of a poor fluid-replacement strategy before, during, and after prolonged exercise and may become a potentially life-threatening condition. However, consumption of large quantities of water alone is not enough to cause hyponatremia. The condition is accompanied by significant loss of Na^+ from sweating [Montain et al. 2006].

Hyponatremia usually occurs in high-endurance athletes participating in marathons, ultra-marathons, and so-called Iron Man triathlons, but it can also occur in people working for long periods of time in hot environments [Rosner and Kirven 2007]. The incidence of hyponatremia in high-endurance athletes ranges from 13% to 18%. Symptoms of hyponatremia can range from none to minimal (~70% of cases) to severe, including encephalopathy, respiratory distress, and death. During these events or work periods, hyponatremia develops when the athlete or worker consumes too much plain water in an attempt to rehydrate after copious sweating. The excess water results in a dilution of plasma Na^+, which, in turn, causes an osmotic disequilibrium that can lead to cerebral edema (brain swelling) and pulmonary edema. These conditions can be fatal in a small number of patients [Rosner and Kirven 2007].

Risk factors for the development of hyponatremia are an exercise duration of >4 hours, gender (females are more likely to develop hyponatremia), low body mass, excessive consumption of water (>1.5 $L \cdot h^{-1}$), pre-exercise hydration, consumption of nonsteroidal anti-inflammatory medications (although not all studies have shown this), and extreme environmental temperature [Rosner and Kirven 2007]. Consumption of large quantities of water is highly correlated with the development of hyponatremia [Almond et al. 2005], the majority of athletes or workers in hot environments proceed to develop hyponatremia; however, not all those with hyponatremia become symptomatic [Rosner and Kirven 2007]. Therefore, the presence of mild hyponatremia may not be harmful. Although hyponatremia can have devastating consequences, such as death, for the sufferer and the pathophysiology of the condition involves multiple factors including the environmental as well as individual predisposition, the condition can be treated effectively to reduce the level of morbidity and mortality [Rosner and Kirven 2007]. Prevention of this condition involves appropriate use of fluid-replacement strategies.

Two hormones are important in thermoregulation: ADH and aldosterone. A variety of stimuli encourage the synthesis and release of those hormones, such as changes in plasma volume and plasma concentration of sodium chloride. ADH is released by the pituitary gland, which has direct neural connections with the hypothalamus but may receive neural input from other sources. Its function is to reduce water loss by the kidney, but it has no effect on the water loss through sweat glands. Body water, including the plasma volume in the vascular compartment, is also controlled by the RAAS. Changes in fluid volume or electrolyte (sodium) concentration will activate the RAAS to conserve fluids and electrolytes at the level of the kidney and sweat glands through the action of aldosterone [Jackson 2006]. The control of fluid (especially plasma) volume is also important in the maintenance of blood pressure and organ perfusion. Another product of the RAAS is angiotensin II, a powerful vasoconstrictor that helps maintain blood pressure and overall cardiovascular function in the presence of significant fluid loss from the vascular compartment, as well as a significant stimulator of the release of aldosterone from the adrenal glands [Williams et al. 2003].

4.1.4.2 Dietary Factors

A well-balanced diet in temperate environments usually suffices for hot climates. A very high protein diet might increase the urine output needed for nitrogen removal and increase water intake requirements [Greenleaf 1979; Greenleaf and Harrison 1986]. The importance of water and salt balance has been emphasized in the previous discussion, and the possibility that it might be desirable to supplement the diet with potassium has also been discussed. In some countries where the normal diet is low or deficient in vitamin C, supplements may enhance heat acclimatization and thermoregulatory function [Strydom et al. 1976]. Sodium supplements can be abused in the interest of increasing fluid retention. However, an excess of dietary sodium may actually decrease plasma volume, even with a controlled fluid intake. Therefore, supplemental dietary sodium must be used judiciously to prevent further dehydration and electrolyte depletion in workers [McArdle et al. 1996a; Williams et al. 2003]. In addition, certain dietary supplements may impair or alter cellular and systemic adaptions associated with both thermotolerance and heat acclimation in exercising humans [Kuennen et al. 2011].

4.1.4.3 Gastrointestinal Factors

Exertional hyperthermia may result in changes in GI permeability (“leaky gut”) that can result in the release of paracellular endotoxins [Zuhl et al. 2014]. The release of these endotoxins can trigger the release of leukocytes, which can result in the further release of proinflammatory cytokines [Leon 2007]. This inflammatory cascade results in further damage to the organ systems. Certain factors increase the susceptibility to increased GI permeability and endotoxin damage, including age; dehydration; loss of electrolytes from sweating; cardiovascular disease; use of certain medications (e.g., diuretics, anticholinergics, and NSAIDs); and alcoholism. In addition, certain foods containing quercetin (e.g., capers, red onions, etc.) can increase hyperthermia-induced GI permeability and aggravate the effects of hyperthermia [Kuennen et al. 2011; Zuhl et al. 2014].

4.1.5 Acclimatization to Heat

When workers are exposed to hot work environments, they readily show signs of distress and discomfort, such as increased core temperatures and heart rates, headache or nausea, and other symptoms of heat exhaustion [Leithead and Lind 1964; WHO 1969; Kerslake 1972

(p. 316); Wyndham 1973; Knochel 1974; Hancock 1982; Spaul and Greenleaf 1984; DOD 2003]. On repeated exposure to a hot environment, there is a marked adaptation in which the principal physiologic benefit appears to result from an increased sweating efficiency evidenced by earlier onset of sweating, greater sweat production, and lower electrolyte concentration) and a concomitant stabilization of the circulation. As a result, after daily heat exposure for 7 to 14 days, most individuals perform the work with a much lower core temperature and HR and a higher sweat rate (i.e., a reduced thermoregulatory strain) and with none of the symptoms that were experienced initially [Moseley 1994; Armstrong and Stoppani 2002; DOD 2003; Navy Environmental Health Center 2007; Casa et al. 2009; ACGIH 2014]. During that period, the plasma volume rapidly expands, so that even though the blood is concentrated throughout the exposure to heat, the plasma volume at the end of the heat exposure in acclimatized individuals is often equal to or greater than before the first day of heat exposure.

Acclimatization to heat is an example of physiologic adaptation, which is well demonstrated in laboratory experiments and field experience [Lind and Bass 1963; WHO 1969]. However, acclimatization does not necessarily mean that individuals can work above the Prescriptive Zone (see Glossary) as effectively as below it [Lind 1977].

Full heat acclimatization occurs with relatively brief daily exposures to working in the heat. It does not require exposure to heat at work and rest for the entire 24 hours; in fact, such excessive exposures may be deleterious because it is difficult for individuals without heat acclimatization experience to replace all of the water lost in sweat. The minimum exposure time for achieving heat acclimatization is at least 2 hours per day, which may be broken into 1-hour exposures [DOD 2003]. Some daily period of relief from exposure to heat, in air-conditioned surroundings, is beneficial to the well-being of the individuals, if for no other reason than that they find it difficult to rest effectively in a hot environment [Kerslake 1972 (p. 316)]. The level of acclimatization depends on the initial level of individual physical fitness and the total heat stress experienced by the individual [DOD 2003]. Thus, a worker who does only light work indoors in a hot climate will not achieve the level of acclimatization needed to work outdoors with the additional heat load from the sun or to do harder physical work in the same hot environment indoors. Increased aerobic fitness confers at least partial acclimatization to the heat because of the increased metabolic heat production that occurs during exercise. Physically fit individuals have a reduced incidence of heat injury or illness during exposure to hot environments [Tipton et al. 2008].

Failure to replace the water lost in sweat will slow or even prevent the development of the physiologic adaptations described. It is important to understand that heat acclimatization increases the sweating rate; therefore, workers will have an increased water requirement [DOD 2003; Navy Environmental Health Center 2007]. In spite of the fact that acclimatization will be reasonably well maintained for a few days of no heat exposure, absence from work in the heat for a week or more results in a significant loss in beneficial adaptations. However, heat acclimatization can usually be regained in 2 to 3 days upon return to a hot job [Lind and Bass 1963; Wyndham 1973]. Heat acclimatization appears to be better maintained by individuals who are physically fit [Pandolf et al. 1977].

The total sweat production increases with acclimatization, and sweating begins at a lower core temperature [DOD 2003]. Cutaneous circulation and circulatory conductance decrease with acclimatization, reflecting the reduction in the proportion of CO that must be allocated for thermoregulation because of the more efficient sweating mechanism. Also during acclimatization, cardiovascular stability is improved, heart rate is lowered, stroke volume is increased, and myocardial compliance is improved [DOD 2003]. It is clear, however, that during exercise in heat, the production of aldosterone increases to conserve salt from both the kidney and the sweat glands, whereas an increase in ADH conserves the amount of water lost through the kidneys. The increase in levels of aldosterone results in a lower concentration of sodium in the sweat and thus serves to limit sodium and fluid loss from the plasma during exercise in the heat [Taylor et al. 2008].

It is clear from the foregoing descriptions that sudden seasonal shifts and sudden increases in environmental temperature may result in thermoregulatory difficulties for exposed workers. At such times, cases of heat disorder may occur, even for acclimatized workers.

Acclimatization to work in hot, humid environments provides adaptive benefits that also apply in desert environments, and vice versa; the qualifying factor appears to be the total heat load experienced by the individual [DOD 2003]. An acclimatization plan should be implemented at all workplaces where workers are exposed to heat. A recent study presented 20 cases of heat-related illness or death among workers [Arbury et al. 2014]. In most of these cases, employers had no program to prevent heat illness, or the program was deficient. Acclimatization was the program element most commonly missing and most clearly associated with worker death.

For a summary on acclimatization, see Table 4-1.

4.1.6 Other Related Factors

Many factors can increase a worker's risk of heat-related illness. Some of the factors are environmental, such as direct sun exposure, high temperatures, and humidity. Indoor radiant heat sources, like ovens and furnaces, also can increase the amount of heat in the environment. An indoor work environment can become a heat hazard if air conditioning is unavailable or ventilation is insufficient [Chen et al. 2003]. Other factors may be related to characteristics of each individual worker or an individual's current status of health at the time of exposure to heat stress in a hot environment. Heat-related illness factors are presented in Figure 4-1.

4.1.6.1 Age

The aging process results in a more sluggish response of the sweat glands, which leads to a less effective control of body temperature in the sedentary individual [Taylor et al. 2008]. Aging also results in a decreased level of skin blood flow associated with exposure to heat. The cause remains undetermined, but the decrease in skin blood flow implies an impaired thermoregulatory mechanism, possibly related to reduced efficiency of the sympathetic nervous system [Hellon and Lind 1958; Lind 1977; Drinkwater and Horvath 1979]. For women, it has been found that the skin temperature increases with age in moderate and high heat loads but not in low heat loads [Hellon and Lind 1958; Drinkwater and Horvath 1979]. When two groups of male coal miners of average age 47 and 27 years, respectively, worked in several comfortable or cool environments, they showed little difference in their responses to heat near the REL with light work, but in hotter environments the older men showed a substantially greater thermoregulatory strain than their younger counterparts; the older men also had lower aerobic work capacities [Lind et al. 1970]. In analyzing the distribution of 5 years' accumulation of data on heat stroke in South African gold mines, Strydom [1971] found a marked increase in heat stroke with increasing age of the workers. Thus, men over 40 years of age represented less than 10% of the mining

Table 4-1. Acclimatization in workers

Topics	Additional information
Disadvantages of being unacclimatized	▪ Readily show signs of heat stress when exposed to hot environments. ▪ Difficulty replacing all of the water lost in sweat. ▪ Failure to replace the water lost will slow or prevent acclimatization.
Benefits of acclimatization	▪ Increased sweating efficiency (earlier onset of sweating, greater sweat production, and reduced electrolyte loss in sweat). ▪ Stabilization of the circulation. ▪ Work is performed with lower core temperature and heart rate. ▪ Increased skin blood flow at a given core temperature.
Acclimatization plan	▪ Gradually increase exposure time in hot environmental conditions over a period of 7 to 14 days. ▪ For new workers, the schedule should be no more than 20% of the usual duration of work in the hot environment on day 1 and a no more than 20% increase on each additional day. ▪ For workers who have had previous experience with the job, the acclimatization regimen should be no more than 50% of the usual duration of work in the hot environment on day 1, 60% on day 2, 80% on day 3, and 100% on day 4. ▪ The time required for non–physically fit individuals to develop acclimatization is about 50% greater than for the physically fit.
Level of acclimatization	▪ Relative to the initial level of physical fitness and the total heat stress experienced by the individual.
Maintaining acclimatization	▪ Can be maintained for a few days of non-heat exposure. ▪ Absence from work in the heat for a week or more results in a significant loss in the beneficial adaptations leading to an increased likelihood of acute dehydration, illness, or fatigue. ▪ Can be regained in 2 to 3 days upon return to a hot job. ▪ Appears to be better maintained by those who are physically fit. ▪ Seasonal shifts in temperatures may result in difficulties. ▪ Working in hot, humid environments provides adaptive benefits that also apply in hot, desert environments, and vice versa. ▪ Air conditioning will not affect acclimatization.

Adapted from [Moseley 1994; Armstrong and Stoppani 2002; DOD 2003; Casa et al. 2009; ACGIH 2014; OSHA-NIOSH 2011].

Figure 4-1. Examples of heat-related illness risk factors

population, but they accounted for 50% of the fatal cases and 25% of the nonfatal cases of heat stroke. The incidence of cases per 100,000 workers was 10 or more times greater for men over 40 years than for men under 25 years of age. In all the experimental and epidemiologic studies described above, the workers had been medically examined and were considered free of disease. Chronic hypohydration increases with age, which may be a factor in the observed higher incidence of fatal and nonfatal heat stroke in the older group. The reasons for hypohydration in older adults appear to be related to a decreased thirst drive resulting in, among other things, a suboptimal plasma volume. A reduced plasma volume (and most likely total body water, given the normal transfer of fluids between compartments) can impair thermoregulatory dynamics [McArdle et al. 2010a]. Another study suggests that age-related impairments in heat loss may not be evident during durations of <15 minutes of exercise; therefore, older workers may be safe from thermal strain and dehydration if they work for shorter intervals [Wright et al. 2014].

Older people are more susceptible to the effects of heat, and a significant fraction of those who suffer from heat disorders are older than 60 [Kenny et al. 2010]. The age-related susceptibility to heat is multifactorial and may be related to decreases in sweating and cutaneous blood flow, changes in cardiovascular function, and decreases in overall fitness [Kenney et al. 1990; Minson et al. 1998; Inoue et al. 1999]. Decreased sweating may be due to reduced sweat production rather than a reduced number of sweat glands [Inbar et al. 2004]. Thus, while acclimatization to heat can occur in the elderly, the rate of acclimatization is reduced [Armstrong and Kenney 1993; Inoue et al. 1999].

4.1.6.2 Sex

Although not all aspects of heat tolerance in women have been fully examined, their thermoregulatory capacities have been. An average-sized woman has a lower aerobic capacity than an average-sized man. When they work at similar proportions of their O_2max, women perform either similarly or only slightly less well than men [Drinkwater et al. 1976; Avellini et al. 1980a; Avellini et al. 1980b; Frye and Kamon 1981].

A study examining sweat electrolyte loss during exercise in the heat found that sweat concentrations of Na^+ and Cl^- in men were higher than in women [Meyer et al. 1992]. The study was unable to explain why there appeared to be a difference between the sexes, although sweating rate and the effects of hormonal variations may play a part.

A recent study investigated whether there was an effect of sex on whole-body sudometer (a device to measure total sweat loss from the individual) activity during exercise in the heat [Gagnon and Kenny 2011]. Gagnon and Kenny found that females have a lower whole-body sweat response during exercise in the heat, which results in a greater increase in body temperature. The study concludes that the "results were not due to differences in physical characteristics, as both sexes were matched for body mass and surface area."

According to Nunneley [1978], there seemed to be little change in thermoregulatory capacities at different times during women's menstrual cycles.

Pregnancy is a consideration for women in coping with heat stress, and as it progresses tolerance of heat stress is reduced [Navy Environmental Health Center 2007]. Pregnancy naturally elevates the body's temperature, which subjects women to heat exhaustion more rapidly during periods of extreme temperatures. The fetus acts as a source of metabolic heat and also increases the weight of the mother. Also, because the pregnant woman is caring for more than one body, more fluids and energy are needed to cool her core temperature. Increased heat loads during early pregnancy increase the fetus' risk of teratogenic effects [Tillett 2011]. However, earlier studies suggested that the basal core temperature of pregnant women participating in submaximal exercise (i.e., bicycle test) studies over the course of their pregnancy decreased slightly postpartum [Lindqvist et al. 2003]. This result agrees with an even earlier study which reported that the magnitude of any first trimester exercise-associated thermal stress for the embryo or fetus is reduced by the maternal physiologic adaptions during pregnancy

[Clapp 1991]. Moderate thermal stress (21.1°C [70°F] for 20 minutes) did not induce regular uterine contractility, nor was it harmful to the fetus when it was exposed during late pregnancy [Vaha-Eskeli and Erkkola 1991].

4.1.6.3 Body Fat

It is well established that obesity predisposes individuals to heat disorders [Leithead and Lind 1964]. In fact, heat disorders occur 3.5 times more frequently in the obese than in lean individuals [Henschel 1967; Chung and Pin 1996; Kenny et al. 2010]. The acquisition of fat means that additional weight must be carried, thereby calling for a greater expenditure of energy to perform a given task and use of a greater proportion of the $\dot{V}O_2$ max on an overall per-weight basis. However, there is no difference in $\dot{V}O_2$ max between obese and lean subjects if measured on an oxygen consumption per lean body weight basis [Vroman et al. 1983]. In addition, the ratio of body surface area to body weight (m^2 to kg) in the obese individual becomes less favorable for heat dissipation. Probably more important is the lower physical fitness and decreased maximum work capacity and cardiovascular capacity frequently associated with obesity. The increased layer of subcutaneous fat provides an insulation barrier between the skin and the deep-lying tissues. The fat layer theoretically reduces the direct transfer of heat from the muscles to the skin [Wells and Buskirk 1971].

The limited number of studies in the area of heat stress and obesity has shown that obese individuals have a lower forearm blood flow during exercise in the heat, which is thought to reduce the cutaneous exchange of heat with the environment [Vroman et al. 1983; Kenny et al. 2010]. The reason for this occurrence is not completely clear, but it may be due to changes in sympathetic control over the vasculature or reduced stroke volume that regulate the relative blood flow to muscle (to perform work) and the blood flow to the cutaneous vasculature for the purpose of heat exchange. Some evidence exists that obese individuals suffer from asymptomatic small-fiber neuropathies, which lower thermal sensitivity [Herman et al. 2007].

It has also been proposed that the increase in thermal load in the obese is due simply to the reduced specific heat capacity of adipose tissue, which contains a lower amount of water per gram, in comparison with lean mass. Thus, for a given thermal load, the obese individual will store thermal energy at a greater rate than the lean individual, resulting in a greater average tissue temperature [Henschel 1967; Kenny et al. 2010].

Finally, the extra weight carried by the obese versus lean individual results in an increase in metabolic energy for any given task. The increase in metabolic energy produced in the form of muscular work results in an increase in body temperature that must be exchanged, in comparison with a lean individual performing the same task in the same environment [Bar-Or et al. 1969; Kenny et al. 2010]. Therefore, because it is apparent that obesity places the individual at a significantly higher risk of suffering a heat-related illness at any given workload or environmental temperature than the risk for a lean individual, additional accommodations may be necessary.

4.1.6.4 Drugs

(1) Therapeutic Drugs

Many drugs prescribed for therapeutic purposes can interfere with thermoregulation [Khagali and Hayes 1983]. Some of these drugs are anticholinergic in nature or involve inhibition of monoamine oxidative reactions, but almost any drug that affects central nervous system activity, cardiovascular reserve (e.g., beta blockers), or body hydration could potentially affect heat tolerance. Cardioselective beta blockers (e.g., atenolol, betaxolol, metoprolol, and acebutolol) do not allow the small blood vessels in the skin to dilate, thereby reducing blood flow, which impedes sweat production and causes body temperature to rise. Thus, beta blockers predispose those working in heat to heat-related emergencies. A worker who requires therapeutic medications should be under the supervision of the responsible health-care provider who understands the potential ramifications of drugs on heat tolerance. In such instances, a worker taking therapeutic medications who is exposed only intermittently or occasionally to a hot environment should seek the guidance from the responsible healthcare provider. See Table 4-2 for additional information on proposed mechanisms of action of drugs implicated in intolerance of heat.

(2) Alcohol and Caffeine

It is hard to separate drugs used therapeutically from those used socially. Alcohol use (combined with heat stress) commonly has been associated with the occurrence of heat stroke [Leithead and Lind 1964]. It is a drug which interferes with central and peripheral nervous function and is associated with dehydration by suppressing ADH production. The ingestion of alcohol prior to or during work in the heat should not be permitted because it reduces heat tolerance and increases the risk of heat-related illnesses.

There are many drugs other than alcohol that are used on social occasions and have been implicated in cases of heat disorder, sometimes leading to death [Khagali and Hayes 1983]. Caffeine may be considered a socially accepted drug found in common beverages and foods (eg., coffee, tea, soft drinks, energy drinks, cocoa, chocolate) and in some over-the-counter analgesics that are consumed worldwide to enhance alertness, reduce fatigue, enhance athletic performance, augment the effects of mild analgesics and for simple enjoyment [Undem 2006; Taylor et al. 2008]. Coffee is one of the most widely consumed beverages in the world, and contains caffeine, which has a mild diuretic effect and should not be provided to workers to replace fluids lost to sweating. Moreover, coffee is generally consumed as a hot beverage and has the potential to exacerbate heat stress. In the past, caffeine was considered to contribute to heat stress by reducing fluid volume and result in cardiovascular strain during exposure to the heat [Serafin 1996].

Recent studies present evidence that caffeine may have less effect on heat tolerance than previously suspected [Roti et al. 2006; Armstrong et al. 2007a; Ely et al. 2011]. Armstrong et al. [2007a] propose that caffeine consumption does not result in water-electrolyte imbalances and does not reduce exercise-heat tolerance. They also suggest that "caffeinated fluids contribute to the daily human water requirement in a manner that is similar to pure water." Ely et al. [2011] similarly found that a caffeine dose of 9 $\mathrm{mg \cdot kg^{-1}}$ did not substantially alter heat balance during work in a hot environment. Caffeine appeared not to interfere with dry heat gains or evaporative heat losses, and total caffeine levels of 9 $\mathrm{mg \cdot kg^{-1}}$ or less (approximately the amount found in one cup of coffee) could be considered safe in hot, dry environments. Roti et al. [2006] concluded that there was no evidence that dehydration or impaired thermoregulation resulted from chronic caffeine ingestion prior to or during exercise in the heat.

Although these studies present evidence that caffeine use may be harmless and acceptable for those exerting themselves in a hot environment, water is still the preferred hydrating beverage for before, during, and after work. However, virtually any nonalcoholic beverage is better than drinking nothing during heat exposure. Further research is needed on the effects of large doses of caffeine consumed at one time and the differences between modes of caffeine delivery (i.e., capsules, various

Table 4-2. Drugs implicated in intolerance to heat

Drug or drug class	Proposed mechanism of action
Anticholinergics (e.g., benzotropine, trihexyphenidyl)	▪ Impaired sweating
Antihistamines	▪ Impaired sweating
Phenothiazines	▪ Impaired sweating, (possibly) disturbed hypothalamic temperature regulation
Tricyclic antidepressants (e.g., imipramine, amitriptyline, protriptyline)	▪ Impaired sweating, increased motor activity and heat production
Amphetamines, cocaine, ecstasy	▪ Increased psychomotor activity, activated vascular endothelium
Analgesics (e.g., acetaminophen, aspirin)	▪ Liver or kidney damage
Ergogenic stimulants (e.g., ephedrine/ephedra)	▪ Increased heat production
Lithium	▪ Nephrogenic diabetes insipidus and water loss
Diuretics	▪ Salt depletion and dehydration
Calcium channel blockers (e.g., amlodipine, verapamil)	▪ Reduced skin blood flow and reduced blood pressure
Ethanol	▪ Diuresis, possible effects on intestinal permeability
Barbiturates	▪ Reduced blood pressure
Antispasmodics	▪ Impaired sweating
Haloperidol	▪ Tachycardia, altered central temperature regulation, and hyponatremia
Laxatives	▪ Dehydration
Beta blockers (atenolol, betaxolol)	▪ Reduced skin blood flow, reduced blood pressure, and impaired sweating
Narcotics	▪ Excessive sweating, salt depletion and dehydration
Levothyroxine	▪ Excessive sweating, salt depletion and dehydration

Adapted from *Heat Stress Control and Heat Casualty Management* [DOD 2003].

beverages, and solid food) [Armstrong et al. 2007a]. Caffeine-containing fluids are now marketed to the public as energy drinks. These drinks contain higher than normal doses of caffeine (more than what is found in a cup of coffee or soft drink) and have been used extensively among competitive athletes prior to participation in athletic events [Burke 2008]. The lethal oral dose for caffeine in humans has been reported to range between 18 and 50 grams [HSDB 2011]. Other than the diuretic effects, this dose of caffeine is capable of inducing cardiac arrhythmias [Undem 2006], which could be potentiated by heat stress (i.e., as a result of already existing cardiovascular strain). It seems that there would also be a tendency to drink several energy drinks just to alleviate thirst as they are often available in smaller containers with less liquid, thus inadvertently overdosing on caffeine.

4.1.6.5 Non-heat Disorders

It has long been recognized that individuals suffering from degenerative diseases of the cardiovascular system and other diseases, such as diabetes or simple malnutrition, are at increased risk when they are exposed to heat and when stress is imposed on the cardiovascular system. The outcome is readily seen during sudden or prolonged heat waves in urban areas where there is a sudden increase in mortality, especially among older individuals, who supposedly have age-related reduced physiologic reserves [Leithead and Lind 1964; Henschel et al. 1969; Ellis 1972; Kilbourne et al. 1982]. In prolonged heat waves, the mortality is higher in the early phase of the heat wave [Henschel et al. 1969; Ellis 1972]. While acclimatization may play a part in the decrease in mortality during the latter part of a prolonged heat wave, the increased death rate in the early days of a heat wave may reflect an "accelerated mortality," with the most vulnerable more likely to succumb at that time rather than more gradually as a result of degenerative diseases.

Any sicknesses or diseases in which GI permeability is compromised could result in greater susceptibility to leakage of endotoxin and the cascade of immune reactions and death that occur with heat stroke. For example, quercetin ingestion during heat acclimation prevented many of the benefits of heat acclimation, whereas glutamine ingestion improved GI barrier function and prevented endotoxin absorption and GI distress [Zuhl et al. 2014].

4.1.6.6 Individual Variation

In all experimental studies of the responses of humans to hot environmental conditions, a wide variation in responses has been observed. These variations are seen not only between individuals but also, to some extent, in the same individual exposed to high environmental stress on different occasions. Such variations are not totally understood. It has been shown [Wyndham 1973] that the influence of body size and its relationship to aerobic capacity in tolerance to heat could account for about half of the variability, leaving the remainder to be determined. Changes in hydration and salt balance might be responsible for some of the remaining variability [Buskirk and Bass 1980]. However, the degree of variability in tolerance to hot environments is poorly understood. Nevertheless, some individuals exhibit poor tolerance to heat and, of this group, some may have hormonal issues that disrupt their ability to control fluids and electrolytes which increases their susceptibility to heat injury [Rosner and Kirven 2007].

4.1.7 Heat-Related Illnesses and Work

The incidence of occupational heat-related illness in the United States is not well-documented by an existing surveillance system specific to occupational injury and illness. According to BLS data from 2010, 4,190 cases of illness and injury from exposure to environmental heat, resulting in one or more days of lost work, occurred among private industry and state and local workers [Bureau of Labor Statistics 2011]. In that same year, 40 workers died from exposure to environmental heat. Eighteen of these workers died in the construction industry; 6 workers died in the natural resources field (including agriculture) and mining; 6 died in professional and business services (including waste management and remediation); and 3 died in manufacturing. In addition, the National Fire Protection Association (NFPA) reported 2,890 cases of "thermal stress" in 2008, but this category includes both frostbite and heat exhaustion as per their definition [NIOSH 2010].

In the following sections, various worksites that may have heat exposure are evaluated and presented in short case study reports.

4.1.7.1 Health Hazard Evaluation (HHE) Reports

National Park

(The full report is available at http://www.cdc.gov/niosh/hhe/reports/pdfs/2013-0109-3214.pdf.)

Park management requested NIOSH to evaluate employees working in extreme heat (e.g., employees involved in maintenance of asphalt parking lots, housing repairs, grounds maintenance, or archaeological surveys), to review current and proposed heat stress management policies, and to recommend ways of preventing heat-related illnesses [NIOSH 2014b]. July temperatures in the park ranged from 31.1°C to 46.7°C (88°F to 116°F). Employees were asked to participate in work and medical history questionnaires, which also asked about health symptoms. Core body temperatures and heart rate were measured. Blood was analyzed daily for markers of muscle breakdown and dehydration during 4 workdays and the following 3 rest days. Estimates of how hard the workers were working were noted and daily temperatures and humidity were measured. The park's heat stress policies and records of work-related injuries and illnesses were reviewed.

One worker was found to have a core body temperature above the ACGIH defined heat stress criteria (>38.5°C [101.3°F]); however, no workers were determined to be dehydrated or experiencing significant muscle breakdown. Several workers had sustained maximum heart rates. The heat stress policy was found to lack appropriate work/rest scheduling, and workers were not consistent in following the policy (i.e., failing to observe the buddy system rule).

The NIOSH HHE made the following recommendations for managers and workers:

Managers

- Avoid moderate to heavy outdoor tasks during summer months, or if necessary, work at night.
- Reduce the amount of time workers work in extremely hot weather.
- Revise the park's heat stress policy to include work/rest schedules based on WBGT and workload.
- Require workers to conduct self-monitoring.
- Develop a workgroup (i.e., workers, resonsible healthcare provider, and safety manger) to make decisions on self-monitoring options and standard operating procedures.

Workers

- Follow the heat stress policy.
- Carry a radio at all times.
- Avoid working alone (i.e., buddy system).
- Learn the signs and symptoms of heat-related illnesses.
- Self-monitor and document signs and symptoms of heat-related illnesses.
- Tell your supervisor if you have symptoms or if you note symptoms in a coworker.
- Drink plenty of fluids, and take rest breaks as needed.
- Volunteer to be on the work group to develop self-monitoring guidance for working in the heat.

Aluminum Smelter Potrooms

(The full report is available at http://www.cdc.gov/niosh/hhe/reports/pdfs/2006-0307-3139.pdf.)

NIOSH assessed workers' exposure to heat while working in the potrooms at an aluminum smelter [NIOSH 2006a]. In the smelting process, alumina is reduced to nearly pure aluminum at an operating temperature of approximately 982.2°C (1,800°F). Workers were interviewed and completed questionnaires about their medical history, work history, and symptoms experienced during the shift on which they were monitored. Monitoring included core body temperature and heart rate. In addition, urine specific gravity and blood electrolytes were measured before and after shifts. WBGTs were monitored in several locations inside the potrooms, and outdoor weather conditions were monitored.

The mean outdoor temperature for the 5 days of evaluation was 25.6°C (78°F). The WBGT measurements ranged from 28.3° C to 48.9°C (83°F to 120°F), with dry bulb air temperatures reaching 56.7°C (134°F) and radiant temperatures reaching 86.7°C (188°F). High radiant heat means that the workers were absorbing rather than radiating heat, unless proper shielding was provided. Metabolic rates of employees were estimated to be light to moderate (115–360 watts). Except for the crane operator, portions of all tasks were found to exceed the NIOSH recommended ceiling limit (NOTE: the NIOSH recommended ceiling limit has been discontinued) and the ACGIH threshold limit value (TLV®) for working in a hot environment. Common symptoms reported during shifts included racing heartbeat or palpitations, headache, muscle cramps, and lightheadedness or dizziness. Post-shift values for blood bicarbonate, blood urea nitrogen (BUN), creatinine, and urine specific gravity increased significantly. Plasma volume depletion was suggested by the substantial decrease over the shift of the BUN to creatinine ratio and potassium level, and may have been caused by excessive sweating. Many of the workers were found to not be sufficiently hydrated. In addition, several participating workers had evidence of acute kidney injury, which may have been a result of or affected by volume depletion rhabdomyolysis caused by excess heat stress exposure or extreme physical activity.

The NIOSH HHE made the following recommendations for managers and workers:

Managers

- Reduce the physical demands on workers working in the potrooms.
- Require the use of heat-reflective personal protective equipment.
- Install cooling recovery areas in the potrooms.
- Do not use outdoor air to cool workers when it is over 35°C (95°F) outside.
- Follow the heat stress management program.
- Stop 8-hour overtime shifts during extremely hot weather.

Workers

- Use reflective personal protective equipment.
- Use the cooling recovery areas when on breaks.
- Take time to work safely.

Automobile Parts Manufacturing Facility

(The full report is available at http://www.cdc.gov/niosh/hhe/reports/pdfs/2003-0268-3065.pdf.)

The painting department of an automobile parts manufacturing facility was assessed in part for workers subjected to high heat [NIOSH 2003a]. WBGT monitors were placed in the loading and unloading area among the workers and in the cafeteria (for comparison) for the entire work shift. Heat stress was measured for six workers over 2 days, through the use of wireless devices that are swallowed and monitor core body temperature. In addition, heart rate and skin temperature were monitored with other devices. Pre- and post-shift body weights were measured on both days to determine degree of dehydration.

Four of the six participating workers exceeded the ACGIH core body temperature's lower limit (38°C [100.4°F]) six times, and one worker exceeded its upper limit (38.5°C [101.3°F]) once. Of the 13 measurements taken over the 2 days in participating workers, nine showed signs of dehydration (post-weight was less than pre-weight). Three of these measures met or exceeded the 1.5% guideline for adequate hydration. The dry bulb temperatures ranged from 26.9°C to 30.1°C (80.5°F to 86.2°F) in the loading and unloading areas and 21.2°C to 21.5°C (70.2°F to 70.7°F) in the cafeteria. Inadequate ventilation was suspected by the steadily increasing temperature in the loading and unloading areas.

The NIOSH HHE made the following recommendations for managers:

- Allow workers to rest during the rest portion of the work/rest regiment, and do not assign any duties during this time.
- Position fans above workstations, not directly in front of the workers.
- Hire a consultant familiar with ventilation in hot processes to reduce heat.

Glass Bottle Manufacturer

(The full report is available at http://www.cdc.gov/niosh/hhe/reports/pdfs/2003-0311-3052.pdf.)

A manufacturer of glass containers for the beer, spirit, juice, and tea industries was assessed by NIOSH because of concern regarding heat-related illnesses among employees exposed to hot working environments in the hot end of the plant, including the forming department [NIOSH 2003b]. In this department, raw materials are melted together in a furnace at temperatures of 1260°C to 1537.8°C (2,300°F to 2,800°F). The manufacturer used various controls such as fans that supply cooler air from the basement, evaporative cooling fans, sports drinks, two 25-minute worker rest breaks (plus additional breaks at management's discretion), and a review of heat safety during safety meetings and through displayed posters. WBGT measurements were collected in the forming department, metabolic rates of workers were estimated, and employees were interviewed.

The highest WBGT reading was 30.7°C (87.2°F), with a dry bulb temperature of 30.6°C (87.0°F) and a globe temperature of 46.5°C (115.7°F). These results indicated that most surfaces in the department were at an elevated temperature and acted as radiant heat sources. The nearby break room's WBGT was 21.3°C (70.3°F), with a dry bulb temperature of 24.2°C (75.5°F). NIOSH guidelines were used to estimate the metabolic heat produced by the workers (186 kcal·h^{-1}), resulting in a light workload rate. WBGTs and metabolic rates were then compared to those listed in the NIOSH RELs and ACGIH TLVs®, and both recommended a continuous work schedule in similar environments. Eighteen workers were interviewed, with two having experienced heat-related symptoms on a hot day a few months earlier (i.e. heart racing, lack of sweating, persistent headache). Other workers mentioned symptoms in previous years, including those related to heat exhaustion, cramping, and nausea. Some workers mentioned that new workers typically start work in June and are not given enough time to acclimatize, resulting in some quitting. In addition, workers noted that the fans were useful (particularly the evaporative coolers) but they were not well maintained and some were not functional.

The NIOSH HHE made the following recommendations for managers:

- Place the fans that supply cooler air from the basement and the evaporative cooling fans on a preventative maintenance schedule to ensure they are operational throughout the summer months.

- Develop a heat acclimatization program to decrease the risk of heat-related illnesses.
- Develop continuing education programs to ensure that all workers potentially exposed to hot environments and physically demanding job activities stay current on heat stress and heat stress prevention information.
- Monitor environmental heat exposures during the hottest months with a WBGT monitor at (or as close as possible to) the area where the workers are exposed.
- Establish criteria for the declaration of a heat alert.
- Develop a heat-related illness surveillance program, which includes establishing and maintaining accurate records of any heat-related disorder events and noting the environmental and work conditions at the time of disorder.
- Ensure that workers stay hydrated and do not lose more than 1.5% body weight during their shift.
- Create a buddy system so that workers can monitor each other for symptoms of heat disorders.
- Allow workers to take unscheduled breaks if they report feeling weak, nauseated, excessively fatigued, confused, and/or irritable during work in hot temperatures.

Truck Drivers

(The full report is available at http://www.cdc.gov/niosh/hhe/reports/pdfs/2011-0131-3221.pdf.)

An airline catering facility that included truck drivers was evaluated for ergonomic risk factors, heat and cold exposure, and job stress [NIOSH 2014a]. Although heat stress conditions in the food delivery trucks were not specifically evaluated, several employees and management representatives discussed temperatures in the trucks. Employees reported that the trucks were poorly maintained (e.g., some trucks had no air conditioning or had windows that did not open or close). Employees reported that the heating system in some of the trucks could not be turned off so it operated throughout the year.

Managers reported that a typical delivery job took about 2 hours and included loading the truck at the dock, driving approximately 25 minutes to the airport, going through airport security, driving to the plane parked on the tarmac, unloading carts from the truck to the plane, and then driving back to the dock. The delivery could take longer if the plane was not ready to receive the carts. Employees were expected to do three or four deliveries during their shift. Employees could take a break between deliveries in an air conditioned room at the airline catering facility. However, managers stated that on busy days employees sometimes skipped these breaks to keep up with job demands. Some employees also reported that they skipped breaks to shorten their shift so they could leave work early.

Drivers, loaders, and sanitation (i.e., autoclave operators, dishwashers) employees were potentially exposed to hot temperatures. Heat measurements in the autoclave area suggested a continuous work schedule is acceptable for acclimatized employees with moderate workloads when outdoor temperatures are in the mid-70s. For the temperatures measured in these work areas, acclimatization for most people occurred in 4 days by exposing them to progressively longer periods in a hot work environment. The company had heat-related training materials available. The training included sources of heat, symptoms of heat-related disorders, and recommendations for preventing heat stress, such as taking breaks in a cool area and drinking one glass of water every 20 minutes. However, breaks were limited to two per 8-hour shift, and beverages were prohibited in most work areas.

The NIOSH HHE made the following heat stress-related recommendations for managers and workers:

Managers

- Train workers on the health effects of exposure to hot temperatures and ways to be more comfortable at work. Inform employees and supervisors of OSHA heat safety tools found at http://www.osha.gov/SLTC/heatillness/heat_index/heat_app.html. The website provides information on protective measures they can take on the basis of the heat index at the worksite.
- Develop and implement a heat stress prevention program. Establish mandatory breaks and access to fluids for workers exposed to heat.
- Ensure new trucks have air conditioning and repair systems in existing vehicles.

Workers

- Drink plenty of fluids when exposed to heat.
- Take regular breaks to recover from extreme temperatures.
- Take part in safety committees.
- Report symptoms to supervisors and medical staff as soon as they occur.

4.1.7.2 Case Studies

Landscaping Case Study

A 30-year-old male landscape mowing assistant collapsed and died of heat stroke after a day of caring for residential lawns [NIOSH 2002]. Two hours before his death he had complained of feeling light-headed and short of breath, but he refused assistance offered to him by his partner. The worker was on medication that had a warning about exposure to extreme heat, and this might have interfered with body temperature regulation. The landscape worker had been wearing two pairs of work pants on the day he died, but his partner did not notice any profuse sweating or flushed or extremely dry skin. Upon collapse, the victim was treated by emergency medical services (EMS) personnel at the site and then transported to the hospital. There he was pronounced dead, with an internal temperature of 42°C (107.6°F). On the day of the incident, the maximum air temperature was 17.2°C (81°F).

The following recommendations were made after the incident:

- Employers should ensure that supervisors/managers monitor workers during periods of high heat stress.
- Identify workers with risk factors that would predispose them to heat-related illnesses.
- Train workers about heat stress, heat strain, and heat-related illnesses.
- Ensure all workers are able to recognize the signs and symptoms of heat-related illnesses in themselves and in others.
- Stress the importance of drinking nonalcoholic beverages before, during, and after working in hot conditions.
- Periodically remind workers of the signs of heat-related illness and encourage them to drink copious amounts of water during hot conditions.

Migrant Farm Worker Case Studies

A male Hispanic worker aged 56 years died of heat stroke after hand-harvesting ripe tobacco leaves for 3 days on a North Carolina farm [CDC 2008]. On the third day, the man started working at 6:00 a.m. and took a short midmorning break and a 90-minute lunch break. Mid-afternoon, a supervisor observed the man working slowly and reportedly instructed him to rest, but the man continued working. An hour later, the man appeared confused and coworkers carried him to the shade and tried to get him to drink water. The man was taken by ambulance to an emergency department, where his core temperature was recorded as 42.2°C (108°F) and, despite treatment, he died. On the day of the incident, the local temperature was approximately 33.9°C (93°F) with 44% RH and clear skies. The heat index (a measurement of how hot it feels when both actual temperature and RH are considered) was in the range of 30°C to 44.4°C (86°F to 112°F) that day.

In an additional, similar case study, a male Hispanic migrant worker aged 44 years died of heat stroke while working at another North Carolina tobacco farm [NIOSH 2006b]. He had been working in the fields for about the last week of July. On August 1, the heat index was between 37.8°C and 43.3°C (100°F and 110°F). Around 3 p.m. the worker complained to the crew leader that he was not feeling well. He drank some water and was driven to the workers' housing and left alone. He was found unconscious approximately 45 minutes later. Emergency medical personnel responded within 5 minutes and the worker was taken to the hospital and pronounced dead. His core body temperature was recorded at 42.2°C (108°F).

The following recommendations were made after the incident:

- Agricultural employers should develop, implement, and enforce a comprehensive safety and health program that includes standard operating procedures for prevention of heat-related illnesses.
- Train supervisors and workers on how to prevent, recognize, and treat heat illness, using a language and literacy level that workers can understand.
- Establish a hydration program that provides adequate potable water (or other appropriate hydrating fluid) for each worker and which encourages workers to drink at regular intervals.
- Monitor environmental conditions and develop work/rest schedules to accommodate high heat and humidity.
- Provide an appropriate acclimatization program for new workers to a hot environment, workers who have not been on the job for a period of time, and experienced workers during a rapid change in excessively hot weather.
- Provide prompt medical attention to workers who show signs of heat-related illness.

Construction Case Study

A 41-year-old male construction laborer was sawing boards to make concrete forms that were to be part of an addition to a factory [NIOSH 2004]. At 5 p.m. the worker collapsed in the parking lot on the way to his vehicle. He was found 30 minutes later by a factory worker, who then returned to the factory and reported the situation to a supervisor. The receptionist was instructed to call EMS while the supervisor administered emergency care to the collapsed worker. The worker's body temperature was recorded as 41.7°C (107°F) by the EMS and as 42.2°C (108°F) when admitted to the hospital. The worker died the next day from heat stroke.

The following recommendations were made after the incident:

- Train supervisors and workers to recognize symptoms of heat exhaustion/stroke when working in high heat index and/or humid conditions.
- To avoid dehydration and heat exhaustion/stroke, workers should be given frequent breaks and be provided drinking water and other hydrating drinks when working in humid or hot conditions.

- Work hours should be adjusted to accommodate environmental work conditions such as a high heat index and/or high humidity.

Fire Fighter Case Study

During the construction of a fire line during a small wildland fire, a 21-year-old fire fighter died from heat stroke, another was overcome by heat stroke and survived, and two others suffered heat exhaustion [NIOSH 1997]. The crew had initially started their day by exercising 1 to 1.5 hours as part of their physical training regimen. The 21-year-old had been sick with a suspected viral or bacterial infection in recent days. After physical training, the crew practiced constructing a fire line for about 1 hour. At 11:45 a.m., the crew arrived at the actual fire that had been reported and constructed a fire line as a precaution in an area with no fire. Crews carried canteens and could drink when desired. Around 1:45 p.m., the crew took a 15-minute break and drank water or Gatorade. At 2 p.m. they resumed the fire line, and 15 minutes later one member fell from heat exhaustion and broke his shoulder. He was administered first aid and transported to the hospital. Prior to this, a member of another crew suffered heat exhaustion, was treated by paramedics, and was returned to his base of operation. Around 2:30 p.m., the 21-year-old moved off the line as though he was going to relieve himself. However, 5 minutes later he was found on the ground thrashing. The crew leader found that he was semiconscious and suffering from heat stroke. His clothing was removed, water was dumped on his skin, and chemical cold packs were applied to his body. At 2:50 p.m. paramedics arrived and continued to administer aid. At 3 p.m. another crew member experienced symptoms of heat stroke. An ambulance transported the 21-year-old to the hospital at 3:20 p.m., but he died early the following morning. The maximum measured temperature during the incident was 36.7°C (98°F).

The following recommendations were made after the incident:

- Require supervisors to regularly medically monitor fire fighters, using generally accepted techniques, during periods of high heat stress.
- Ensure fire fighters' workloads are appropriate for their level of acclimatization.
- Ensure fire fighters' workloads are appropriate for the ambient weather conditions and clothing.

4.2 Acute Heat Disorders

Although heat disorders are interrelated and seldom occur as discrete entities, each has unique clinical characteristics [Minard and Copman 1963 (p. 253); Leithead and Lind 1964; Minard 1973; Lind 1977; Dinman and Horvath 1984; Springer 1985]. These disorders range from simple postural heat syncope (fainting) to the complexities of heat stroke. A common feature in all the heat-related disorders (except simple postural heat syncope) is some degree of elevated body temperature, which may be complicated by deficits of body water. The prognosis depends on the absolute level of the elevated body temperature, the promptness of treatment to lower the body temperature, and the extent of deficiency or imbalance of fluids or electrolytes. A summary of classification, clinical features, prevention, and first-aid treatment of heat-related illnesses is presented in Table 4-3.

4.2.1 Heat Stroke

Heat stroke can occur as either classic or exertional. Classic heat stroke includes (1) a major disruption of central nervous system function (unconsciousness or convulsions); (2) a lack of sweating; and (3) a rectal temperature

Table 4-3. Classification, medical aspects, and first aid of heat-related illness

Signs and symptoms	Examples of predisposing factors	Underlying physiologic disturbance	First aid
(1) Temperature regulation			
Heat stroke			
▪ Confusion, altered mental status, slurred speech ▪ Loss of consciousness (coma) ▪ Hot, dry skin or profuse sweating ▪ Seizures ▪ Very high body temperature ▪ Fatal if treatment delayed	▪ Sustained exertion in heat ▪ Obesity and lack of physical fitness ▪ Recent alcohol intake ▪ Dehydration ▪ Individual susceptibility ▪ Chronic cardiovascular disease	▪ Failure of the central drive for sweating, leading to loss of evaporative cooling and an uncontrolled accelerating rise in temperature	▪ A medical emergency: call 911 for emergency medical care ▪ Someone should stay with worker until emergency medical services arrive ▪ Move the worker to a shaded, cool area and remove outer clothing ▪ Cool the worker quickly with a cold water or ice bath if possible; wet the skin, place cold wet cloths on skin, or soak clothing with cool water ▪ Circulate the air around the worker to speed cooling ▪ Place cold wet cloths or ice on head, neck, armpits, and groin; or soak the clothing with cool water
(2) Circulatory hypostasis			
Heat syncope			
▪ Fainting (short duration) ▪ Dizziness ▪ Light-headedness during prolonged standing or suddenly rising from a sitting or lying position	▪ Dehydration ▪ Lack of acclimatization	▪ Pooling of blood in dilated vessels of skin and lower parts of body	▪ Sit or lie down in a cool place ▪ Slowly drink water, clear juice, or a sports drink

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See footnotes at end of table.

(Continued)

Table 4-3 (Continued). Classification, medical aspects, and first aid of heat-related illness

Signs and symptoms	Examples of predisposing factors	Underlying physiologic disturbance	First aid
(3) Water and/or salt depletion			
Heat exhaustion ▪ Headache ▪ Nausea ▪ Dizziness ▪ Weakness ▪ Irritability ▪ Thirst ▪ Heavy sweating ▪ Elevated body temperature ▪ Decreased urine output	▪ Sustained exertion in heat ▪ Lack of acclimatization ▪ Failure to replace water lost in sweat	▪ Dehydration ▪ Depletion of circulating blood volume ▪ Circulatory strain from competing demands for blood flow to skin and to active muscles	▪ Take worker to a clinic or emergency room for medical evaluation and treatment ▪ If medical care is unavailable, call 911 ▪ Someone should stay with worker until help arrives ▪ Remove worker from hot area and give liquids to drink ▪ Remove unnecessary clothing, including shoes and socks ▪ Cool the worker with cold compresses or have the worker wash head, face, and neck with cold water ▪ Encourage frequent sips of cool water

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See footnotes at end of table.

(Continued)

Table 4-3 (Continued). Classification, medical aspects, and first aid of heat-related illness

Signs and symptoms	Examples of predisposing Factors	Underlying physiologic disturbance	First aid
Heat cramps			
▪ Muscle cramps, pain, or spasms in the abdomen, arms, or legs	▪ Heavy sweating during hot work ▪ Drinking large volumes of water without replacing salt loss	▪ Loss of electrolytes in sweat ▪ Water intake dilutes electrolytes ▪ Muscle spasm	▪ Drink water and have a snack and/or carbohydrate-electrolyte replacement liquid (e.g., sports drinks) every 15 to 20 minutes ▪ Avoid salt tablets ▪ Get medical help if the worker has heart problems, is on a low sodium diet, or if cramps do not subside within 1 hour
Hyponatremia			
▪ Symptoms range from none, to minimal, to severe, including encephalopathy, cerebral and pulmonary edema, respiratory distress, and death	▪ Heavy sweating during hot work ▪ Drinking large volumes of water without replacing salt loss	▪ Loss of electrolytes in sweat ▪ Low plasma sodium ▪ Osmotic disequilibrium	▪ Drink water and have a snack and/or carbohydrate-electrolyte replacement liquid (e.g., sports drinks) ▪ Avoid salt tablets ▪ Get medical help if the worker has heart problems or is on a low-sodium diet, or if cramps do not subside within 1 hour
(4) Skin eruptions			
Heat rash (*miliaria rubra*, **“prickly heat,” “sweat rash”**)			
▪ Looks like red cluster of pimples or small blisters that usually appears on the neck, upper chest, groin, under the breasts, and in elbow creases	▪ Unrelieved exposure to humid heat with skin continuously wet with unevaporated sweat	▪ Plugging of sweat gland ducts with retention of sweat and inflammatory reaction	▪ When possible, a cooler, less humid work environment is best treatment ▪ Keep rash area dry ▪ Powder may be applied to increase comfort ▪ Ointments and creams should not be used

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See footnotes at end of table.

(Continued)

Table 4-3 (Continued). Classification, medical aspects, and first aid of heat-related illness

Signs and symptoms	Examples of predisposing Factors	Underlying physiologic disturbance	First aid
Heat rash (*miliaria profunda,* “wildfire”)			
▪ Extensive areas of skin which do not sweat on heat exposure, but present gooseflesh appearance, which subsides with cool environments ▪ Associated with incapacitation in heat	▪ Weeks or months of constant exposure to heat with previous history of extensive heat rash and sunburn	▪ Skin trauma (heat rash; sunburn) causes sweat retention deep in skin, reduced evaporative cooling causes heat intolerance	▪ No effective treatment ▪ Recovery of sweating occurs gradually on return to cooler climate
(5) Muscle tissue injury			
Rhabdomyolysis			
▪ Muscle cramps/pain ▪ Abnormally dark (tea or cola colored) urine ▪ Weakness ▪ Exercise intolerance ▪ Asymptomatic	▪ End result of any process that damages skeletal muscle, such as the following: ▪ Prolonged, intense physical exertion ▪ Elevated body temperature (associated with heat stroke) ▪ Use of certain prescription and over-the-counter medications ▪ Use of certain dietary supplements like creatine and caffeine ▪ Use of illicit drugs that can reduce blood flow to muscle tissue, such as cocaine and methamphetamine ▪ Direct injury to the muscle (i.e., trauma, burns) or infections	▪ Leakage of muscle cell contents into the bloodstream, which may result in seizures, abnormal heart rhythms, nausea, vomiting, fatigue, and kidney damage ▪ Injured muscles located in muscle fascial compartments may swell and cut off blood supply to entire muscle group, which may result in loss of function and permanent disability	▪ Stop activity ▪ Increase oral hydration (water preferred) ▪ Seek immediate care at the nearest medical facility ▪ Ask to be checked for rhabdomyolysis (i.e., blood sample analyzed for creatine kinase)

Adapted from Minard 1973; DOD 2003; Cervellin et al. 2010; OSHA-NIOSH 2011.

in excess of 41°C (105.8°F) [Minard and Copman 1963 (p. 253); Leithead and Lind 1964; Shibolet et al. 1976; Khagali and Hayes 1983]. The 41°C (105.8°F) rectal temperature is an arbitrary value for hyperpyrexia because observations are made only after the admission of patients to hospitals, which may occur from about 30 minutes to several hours after the event. Exertional heat stroke occurs in physically active individuals who will often continue sweating [DOD 2003; Armstrong et al. 2007b; Navy Environmental Health Center 2007]. With exertional heat stroke, the skeletal muscle often rapidly breaks down, which is called acute rhabdomyolysis (see 4.2.1.1), and also results in renal failure [DOD 2003]. The risk of renal failure is about 25% for those suffering from exertional heat stroke [Navy Environmental Health Center 2007]. For additional comparisons between classic and exertional heat stroke see Table 4-4. The metabolic and environmental heat loads that give rise to heat stroke are highly variable and are often difficult or impossible to accurately reconstruct. Also, the medical outcomes vary among patients, depending on the caregiver's knowledge, understanding, skill, and available facilities.

Heat stroke is a medical emergency, and rapidly cooling the affected worker is imperative. If possible, cool the worker quickly with an ice water bath. Placing the affected worker in a shady area, removing outer clothing and wetting or applying ice to the head, neck, armpits, and groin areas, and increasing air movement to enhance evaporative cooling are all important activities to perform while waiting for or transporting to medical care. A worker experiencing heat exhaustion or heat stroke should not be sent home or be left unattended without a specific order from a responsible healthcare provider.

Frequently, by the time a worker is admitted to a hospital, the disorder has progressed to a multisystem emergency affecting virtually all tissues and organs [Dukes-Dobos 1981]. In the typical clinical presentation, the central nervous system is disorganized and there is commonly evidence of fragility of small blood vessels, possibly coupled with the loss of integrity of cellular membranes in many tissues. The blood-clotting mechanism is often severely disturbed, as are liver and kidney functions. It is not clear, however, whether these events are present at the onset of the disorder or whether they develop over time. Postmortem evaluation indicates that few tissues escape pathological involvement. Early recognition of the disorder or its impending onset, when combined with appropriate treatment, considerably reduces the death rate and the extent of organ and tissue involvement [DOD 2003; Navy Environmental Health Center 2007].

4.2.1.1 Rhabdomyolysis

Rhabdomyolysis is a medical condition associated with heat stress and prolonged physical exertion, resulting in the rapid breakdown of muscle and the rupture and necrosis of the affected muscles. When muscle tissue dies, electrolytes and large proteins that formed the muscle's contractile mechanism are released into the bloodstream [Khan 2009; Cervellin et al. 2010]. Potassium is the main electrolyte released into the blood by the death of muscle tissue, and high levels can cause irregular and dangerous heart rhythms and seizures. In addition, the large muscle proteins can damage the delicate filtration system of the kidneys.

Classic symptoms of rhabdomyolysis are muscle pain, cramping, swelling, weakness, and decreased range of motion of joints. One of the signs of rhabdomyolysis is dark or tea-colored urine [Brudvig and Fitzgerald 2007; Khan 2009; Cervellin et al. 2010]. However, symptoms can vary between individuals, with some experiencing nonspecific symptoms such as fatigue, exercise intolerance,

Table 4-4. Comparison of classic and exertional heat stroke

Patient characteristics	Classic	Exertional
Age	Young children or elderly	Typically 15–45 years
Health	Chronic illness or debilitation common	Usually healthy
Prevailing weather	Frequent in prolonged heat waves	Variable
Drug use	Diuretics, antidepressants, anticholinergics, phenothiazines	Usually none; sometimes ergogenic stimulants or cocaine
Activity	Sedentary	Strenuous exercise
Sweating	Usually absent	Often present
History of febrile illness	Unusual	Common
Acid–base disturbances	Respiratory alkalosis	Lactic acidosis
Acute renal failure	Fairly rare	Common
Rhabdomyolysis	Seldom severe	Common; may be severe
Hyperuricemia	Modest	Marked
Creatinine: blood urea nitrogen ratio	1:10	Elevated
Creatine kinase (CK), aldolase	Mildly elevated	Markedly elevated
Hyperkalemia	Usually absent	Often present
Hypocalcemia	Uncommon	Common
Disseminated intravascular coagulation (DIC)	Mild	May be marked
Hypoglycemia	Uncommon	Common

Adapted from *Heat Stress Control and Heat Casualty Management* [DOD 2003].

abdominal pain, back pain, nausea or vomiting, and confusion, while others might have no symptoms [Huerta-Alardin et al. 2005; Brudvig and Fitzgerald 2007]. In one study, only half of patients with confirmed rhabdomyolysis reported muscle pain or weakness [Cervellin et al. 2010]. Because muscle cramps and dark urine after prolonged exertion may be the only symptom and sign, rhabdomyolysis may be mistaken for another heat-related illness and dehydration. This was confirmed by a study of cases in which rhabdomyolysis was initially misdiagnosed; it found that the most common diagnoses given were heat stress and dehydration [Gardner and Kark 1994]. Delays in recognizing rhabdomyolysis due to misdiagnosis are problematic. The more serious the case is and the longer the delay in making the correct diagnosis and initiating treatment, the greater the risk for increased complications, some of which may be permanent such as reduced or loss of kidney function due to compartment syndrome.

Rhabdomyolysis is diagnosed by measurement of creatine kinase (CK), also known as creatine phosphokinase (CPK), in the blood by a licensed health care professional. The severity of rhabdomyolysis depends upon damage to other organ systems and the peak CK level. Long-term health consequences of rhabdomyolysis vary widely and largely depend on speed of recognition and treatment [Line and Rust 1995]. Mild rhabdomyolysis can be treated by drinking lots of fluids [George et al. 2010]. Severe cases require hospitalization and aggressive treatment with intravenous fluids to dilute the proteins and thus minimize their damage to the kidney; monitoring of the heart for dangerous rhythm changes from the surge of electrolytes; and monitoring of kidney function [Sauret et al. 2002]. In severe cases, the kidneys may fail and immediate dialysis is needed to mechanically remove proteins and electrolytes from the blood [Bosch et al. 2009]. The drop in kidney function may be temporary, but in some cases kidney function does not recover, leaving a formerly healthy individual facing a lifetime of dialysis or possibly a kidney transplant. Up to 8% of cases of rhabdomyolysis are fatal [Cervellin et al. 2010]. In the past, rhabdomyolysis screening involved a urine test that was positive if it contained myoglobin but no red blood cells. However, because myoglobin is quickly excreted and levels may return to normal within 6 hours of muscle injury, it is not considered a reliable test for the condition. One study showed that only 19% of patients diagnosed with rhabdomyolysis had elevated urine myoglobin levels at the time of diagnosis [Counselman and Lo 2011].

Risk factors for rhabdomyolysis include elevated core body temperature from environmental heat, from heat generated by physical exertion, or from medical conditions that raise the body's temperature (e.g., malignant hyperthermia); dehydration; prescription medications (e.g., cholesterol-lowering statins and antidepressants); over-the-counter medications (e.g., antihistamines, non-steroidal anti-inflammatory medications, omeprazole); excessive caffeine intake; use of dietary supplements (e.g., creatine and Hydroxycut™); use of medications, alcohol, or amphetamines; underlying medical conditions (e.g., sickle cell trait or lupus); and concurrent bacterial or viral infections (e.g., influenza, Epstein-Barr, or Legionella) [Wrenn and Oschner 1989; Line and Rust 1995; Huerta-Alardin et al. 2005; Melli et al. 2005; Do et al. 2007; Makaryus et al. 2007; Dehoney and Wellein 2009; Nauss et al. 2009; Chatzizisis et al. 2010; George et al. 2010; de Carvalho et al. 2011].

Differentiation between the types of heat exposure that led to the rhabdomyolysis is important because they have different signs, symptoms, and potential sequelae. Victims of classic heat stroke usually do not experience rhabdomyolysis as severe as those with exertional heat stroke. Individuals with exertional heat stroke with rhabdomyolysis often have higher levels of CK, presence of lactic acidosis, significant drop in blood calcium levels (while showing a dangerously high spike in blood potassium levels that can cause heart rhythm irregularities), increased incidence of kidney failure (20%–30% vs <5%), and increased risk for a potentially fatal coagulation disorder called disseminated intravascular coagulation [DOD 2003].

Some jobs intrinsically have risk factors for heat related illness and rhabdomyolysis, such as fire fighting. Fire fighters are at increased risk because they routinely encounter several heat sources as they do their jobs: heat from the fire, ambient heat when working during hot weather, and heat generated by prolonged physical exertion during fire-suppression efforts. Physical exertion is often increased by the effort of carrying additional weight. Structural fire fighters may carry an additional 40 to 60 pounds of turnout gear, self-contained breathing apparatus (SCBA) air tanks, and various equipment (e.g., ventilation hooks can weigh 20–80 lb). Although wildland fire fighters do not wear turnout gear because of the remoteness of the fires they fight, they must be able to be self-sufficient once they are at the fire location, which can mean carrying 40- to 110-lb packs while hiking over steep terrain for prolonged periods. In a 2011 investigation into the heat stroke death of a wildland fire fighter belonging to an interagency "hotshot crew." NIOSH reviewed agency records and found that they had documented 255 nonfatal cases of heat-related illness in the previous 12 years [NIOSH 2012]. NIOSH investigated the exertional heat stroke death of a 26-year-old structural fire fighter who collapsed at the end of a 4.4-mile jog during his cadet training course in 22.8°C (73°F) ambient temperature. On arrival at the emergency department he had a core temperature of 40.7°C (105.3°F) and was found to have rhabdomyolysis. He subsequently developed acute kidney failure and disseminated intravascular coagulation before he died 5 days later.

The incidence of symptomatic exertional rhabdomyolysis in the general population is unknown [Alpers and Jones 2010]. Individuals in occupations that require prolonged, intense physical activity, such as military personnel/recruits, law enforcement, and athletes, are at increased risk for developing rhabdomyolysis [CDC 1990; Gardner and Kark 1994; Walsh and Page 2006; O'Connor and Duester 2011]. Most cases of acute exertional rhabdomyolysis among military trainees occur during the first week of training [Olerud et al. 1976]. More recent data, from the Armed Forces Health Surveillance Center, reported 378 cases of "rhabdomyolysis likely due to physical exertion or heat stress" in 2013 alone, representing a 33% increase in the annual incidence rate between 2009 and 2013. It was noted that 71% of these cases occurred between May and September [Armed Forces Health Surveillance Center 2014]. It is a recurrent issue during summer football practice, as this involves intense physical training regimens often in full protective gear during hot weather.

Repetitive exhaustive exercise regimens including "incentive training" with high-intensity repetitive exercises such as push-ups are more likely to lead to rhabdomyolysis than prolonged submaximal exercise regimens [Olerud et al. 1976]. Although it is not uncommon for individuals in the general population who engage in exertional activities higher than their baseline level of fitness to develop exertional rhabdomyolysis, it also occurs in highly conditioned individuals who may engage in supramaximal (i.e., high intensity, usually short duration) exercise or who have other risk factors concurrent with an exertional activity [Walsh and Page 2006].

Aside from addressing the cause of rhabdomyolysis (i.e., cool the body down if temperature is high), the common theme of treatment of rhabdomyolysis is fluids. Mild cases of rhabdomyolysis can be treated with oral rehydration fluids, which serve to flush the harmful proteins out of the blood before they can harm the kidneys while restoring the volume depletion that often accompanies rhabdomyolysis. Treatment of more severe cases of rhabdomyolysis require fluid intake rates for durations that cannot be accomplished by drinking, which is why inpatient treatment with intravenous fluids is necessary. The treatment goal is to obtain urine output of 200 to 300 mL per hour while monitoring serum CK, potassium, and kidney function markers. Patients with severe rhabdomyolysis should be placed on cardiac monitors, because the elevated levels of potassium can produce potentially fatal heart-rhythm abnormalities. Low blood calcium levels are common with exertional rhabdomyolysis and can result in seizures. Consultation with a nephrologist may be needed for emergency hemodialysis if the kidney function declines. Kidney function may or may not recover, and if not, the patient will require regular dialysis for the rest of her or his life.

Another potential serious complication of rhabdomyolysis is compartment syndrome, which can occur if the damaged muscle is inside a fibrous sheath with other muscles. Most of these sheaths are found in the arms and legs [Cervellin et al. 2010]. All muscles within the same sheath are considered to be in the same compartment. During rhabdomyolysis, the injured muscle becomes inflamed and swollen. Because the fibrous sheath of the compartment cannot expand, the pressure from this swelling can become severe enough to prevent blood from entering the sheath, endangering all muscles inside that compartment. Unless the sheath is cut open by a surgeon to relieve the pressure and restore blood flow, all the muscles in that compartment could die, resulting in permanent loss of function of the affected limb(s) [Walsh and Page 2006].

Patients with compartment syndrome may present with some of the classic "5 P's" of vascular insufficiency: pain, pallor, pulselessness, paresthesias (i.e., sensation of tingling, numbness, or burning, usually felt in the hands, feet, arms, or legs), and paralysis. Pain is the most common of these symptoms and is often severe to the point of being difficult to control even with narcotic analgesics. This pain is worsened with passive extension, and onset may be delayed for hours to days after the initial injury because it can take time for the swelling of the damaged muscle to become severe enough to block the blood flow into the compartment. On examination, the affected limb is swollen, tense, and very tender to applied pressure (although compartment syndrome may occur in the abdomen and neck, it most often occurs in the lower extremities). Swelling of the affected area may also be evaluated by measurement of that extremity's circumference and comparison with that of the unaffected side. However, if compartment syndrome is suspected, then immediate surgical consultation is indicated, along with measurement of the pressure within the compartment. Compartment pressures should not exceed 30 mmHg (4 kP_a); when they do, it is an indication for immediate transfer to surgery for fasciotomy. Prompt treatment is critical because permanent damage to the muscle occurs when its blood flow is blocked for more than 8 hours [Haller 2011].

4.2.2 Heat Exhaustion

Heat exhaustion is often considered a precursor to the more serious heat stroke. This disorder has been encountered frequently in experimental assessment of heat tolerance. Usually, it is accompanied by a slightly elevated core body temperature (38°C–39°C, or 100.4°F–102.2°F). The symptoms of headache, nausea, vertigo, weakness, thirst, heavy sweating, irritability, and a decreased urine output are common to both heat exhaustion and the early stage of heat stroke. There is wide variation in the ability to tolerate an increased mean body temperature; some individuals cannot tolerate rectal temperatures of 38°C–39°C (100.4°F–102.2°F) and others continue to perform well at even higher rectal temperatures [Joy and Goldman 1968].

Failure to replace water predisposes the individual to one or more of the heat disorders, especially heat exhaustion and heat stroke. Data suggest that cases of heat exhaustion can be expected to occur some 10 times more frequently than cases of heat stroke [Khagali and Hayes 1983].

4.2.3 Heat Cramps

Heat cramps are not uncommon in individuals who work hard in the heat. The exact cause or causes have not been determined, but heat cramps may be attributed to or associated with a continued loss of salt in the sweat, accompanied by a copious intake of water without appropriate replacement of salt. Other electrolytes, such as magnesium, calcium, and potassium, may also be involved. Cramps often occur in the muscles principally used during work and can be readily alleviated by rest, the ingestion of water, and the correction of any body fluid electrolyte imbalance (e.g., with sports drinks containing carbohydrates and electrolytes [serum sodium concentration <136 $mEq \cdot L^{-1}$]). Salt tablets should not be taken. Salt losses are best replaced by the ingestion of normal salted foods or fluids over many hours [DOD 2003].

4.2.4 Heat Syncope

Heat syncope (fainting) usually occurs with prolonged standing or sudden rising from a sitting or supine position; in such instances, temporary circulatory failure, due to pooling of blood in the peripheral veins, results in a decrease in diastolic filling of the heart [DOD 2003]. Symptoms of heat syncope include light-headedness, dizziness, and fainting. Factors that may contribute to heat syncope include dehydration and lack of acclimatization. Workers who have fainted will usually recover rapidly if they sit or lay down; however, complete recovery of stable blood pressure and HR may take an hour or two [DOD 2003].

4.2.5 Heat Rashes

The most common heat rash is prickly heat (miliaria rubra), which appears as red papules, usually in areas where clothing is restrictive, and gives rise to a prickling sensation, particularly as sweating increases. It occurs in skin that is persistently wetted by unevaporated sweat, apparently because the keratinous layers of the skin absorb water, swell, and mechanically obstruct the sweat ducts [Pandolf et al. 1980b, 1980a; DiBenedetto and Worobec 1985]. If untreated, the papules may become infected and develop secondary staphylococcal infections [DOD 2003]. Another skin disorder (miliaria crystallina) appears with the onset of sweating in skin previously injured at the surface, commonly in sunburned areas. The damage prevents the escape of sweat and results in the formation of small to large watery vesicles, which rapidly subside once sweating stops; the problem ceases to exist once the damaged skin is sloughed.

Miliaria profunda occurs when sweat ducts are blocked below the skin surface. This rash also occurs following a sunburn injury but has been reported to occur without clear evidence of previous skin injury. Discrete, pale elevations of the skin, resembling gooseflesh, are present.

In most cases, these rashes disappear with return to a cool environment. When a substantial part of the day is spent in cool and/or dry areas so that the skin surface can dry, the rashes are less likely to occur or occur with diminished frequency.

Although heat rashes are not dangerous in themselves, each can impair areas of skin and reduce sweating that reduces evaporative heat loss and impacts thermoregulation. Wet and/or damaged skin can also absorb toxic chemicals more readily than dry, unbroken skin. In experimentally induced miliaria rubra, sweating capacity recovered within 3 to 4 weeks [Pandolf et al. 1980b, 1980a].

4.3 Chronic Heat Disorders

Some long-term effects from heat stress (noted from anecdotal, historical, epidemiologic, and experimental evidence) have been suggested. Severe heat-related illness may cause permanent damage to a person's organs, such as the heart, kidneys, and liver, which may result in a chronic disorder.

One study compared a cohort of U.S. Army personnel hospitalized for heat-related illness with those that had appendicitis. Those with heat-related illness had a 40% higher risk of all-cause mortality than the appendicitis patients [Wallace et al. 2007]. Furthermore, it was found that males with heat-related illness were at an increased rate of death from cardiovascular disease and ischemic heart disease, compared to the appendicitis cases.

More studies are needed to increase our understanding of long-term effects of heat-related illness. The severity of illness, the duration of exposures, and the etiology and prevention of heat cramps are just a few of many factors that may have an effect on a worker's chronic condition.

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Appendix A

Heat Exchange Equation Utilizing the SI Units

This appendix is referred to in Chapter 3 of the current document. It is reprinted in full from the 1986 NIOSH criteria document:

NIOSH [1986a]. Criteria for a recommended standard: occupational exposure to hot environments: revised criteria 1986. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control, National Institute for Occupational Safety and Health.

Convection (C) SI Units

The rate of heat exchange between a person and the ambient air can be stated algebraically:

$$C = h_c(t_a - \bar{t}_{sk})$$

Where

h_c is the mean heat transfer coefficient
t_a = air temperature (°C)
$\bar{t}_{sk}$ = air temperature (°C)

The value of h_c differs for the different parts of the body [Nishi 1981], depending mainly on the diameter of the body part; for example, at the torso the value of h_c is about half of what it is at the thighs. The value used for h_c is generally the average of the h_c values for the head, chest, back, upper arms, hands, thighs, and legs. The value of h_c varies between 2 and 12, depending on body position and activity.

Other factors that influence the value of h_c are air speed, air direction, and clothing. The value of $\bar{t}_{sk}$ can also vary according to the method used for the measurements, the number and location of the measuring points over the body, and the values used for weighting the temperatures measured at the different locations.

Numerous investigations have tried to simplify the calculation of convection heat exchange. The ISO Working Group on the Thermal Environment (ISO-WGTE) developed a draft standard for the Analytical Determination of Heat Stress [ISO 1982b]*. One of the simplifications they adopted is to use only the following three values for h_c, which are expressed in units of $Wm^{-2}°C^{-1}$, corresponding to the SI system.

(a) When air speed is very low and is due only to natural convection,
$h_c = 2.38(\bar{t}_{sk} - t_a)^{0.25}$

(b) In forced convection when relative air speed (V_{ar}) is less than 1m·,
$h_c = 3.5 + 5.2V_{ar}$

(c) In forced convection,
when V_{ar} is greater than 1m·,
$h_c = 8.7V_{ar}^{0.6}$

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*This standard has been revised. See *Ergonomics of the Thermal Environment: Analytical Determination and Interpretation of Heat Stress Using Calculation of the Predicted Heat Strain* (Standard No. ISO 7933) [ISO 2004b].

The expression V_{ar} is defined as the ratio of the air velocity relative to the ground and the speed of the body or parts of the body relative to the ground. If the body movement is due to muscular work, V_{ar} can be calculated by the following equation:

$$V_{ar} = V_a + 0.0052(M - 58)$$

where

V_a = air velocity in ms^{-1}
M = metabolic heat production (Wm^{-2})

For simplicity, however, it is recommended to add 0.7 ms^{-1} to V_a as a correction for the effect of physical work.

The ISO-WGTE recommends also to include in the equation for calculating the convection heat exchange a separate coefficient for clothing, called a reduction factor for loss of sensible heat exchange due to the wearing of clothes (F_{cl}), which can be calculated by the following equation:

$$F_{cl} = 1/1 + (h_c + h_r)I_{cl} \text{ (dimensionless)}$$

where

h_r = the heat transfer coefficient for radiant heat exchange
I_{cl} = the thermal insulation of clothing

Both h_r and I_{cl} will be explained later in this appendix in more detail. The ISO-WGTE recommended the use of 36°C (96.8°F) for t_{sk} on the assumption that most workers engaged in hot jobs would have a t_{sk} very close to this temperature; thus, any error resulting from this simplification will be small. They also assumed that most corrected for different body positions when calculating the convective heat exchange of workers.

The final equation for C to be used according to the ISO-WGTE is

$$C = h_c F_{cl} (t_a - 36) \text{ } (Wm^{-2})$$

Radiation (R) SI Units

The rate of radiant heat exchange between a person and the surrounding solid objects can be stated algebraically:

$$R = h_r (T_r - T_{sk})^4$$

where

h_r = the coefficient for radiant heat exchange
T_r = the mean radiant temperature in °K
T_{sk} = the mean weighted skin temperature in °K

The value of h_r depends on the body position of the exposed worker and on the emissivity of the skin and clothing, as well as on the insulation of clothing. The body position will determine how much of the total body surface will be actually exposed to radiation, and the emissivity of the skin and clothing will determine how much of the radiant heat energy will be absorbed on those surfaces. The insulation of clothing determines how much of the radiant heat absorbed at the surface of the garments will actually be transferred to the skin.

The ISO-WGTE recommended a linearized equation for calculating the value of R, using SI units:

$$R = h_r F_{cl} (t_r - t_{sk}) \ (Wm^{-2} / °C^{-1})$$

The effect of insulation and emissivity of the clothing material on radiant heat exchange is covered by the addition of the clothing coefficient F_{cl}, which is also used in the equation for C as described above.

They also recommend a simplified equation for calculating an approximate value for h_r:

$h_r = 4E_{sk} A_r / A_{Du} [(t_r + t_{sk})/2 + 273]^3$
= the universal radiation constant
$= (5.67 \times 10^{-8}) \ Wm^{-2} °K^{-4}$

The effect of the emissivity of the skin on radiant heat exchange is covered by the expression $\underline{E}_{sk}$, which has the value of 0.97 in the infrared range. The effect of body position is covered by the expression A_r/A_{Du}, which is the ratio of the skin surface area exposed to radiation and the total skin surface area, as estimated by DeBois' formula:

$$A_{Du} = 0.00718 \times \text{Weight}^{0.425} / \text{Height}^{0.725}$$

In this equation, body weight must be expressed in kg and height in cm, and the value of A_{Du} is then obtained in m^2. Values given for the ratio A_r/A_{Du} by the ISO-WGTE include these:

Standing: 0.77
Seated: 0.70
Crouched: 0.67

The value of t_r (mean radiant temperature) can be calculated by the following equation:

$$t_r = t_g + 1.8V_a^{0.5}(t_g - t_a)$$

For further simplification, the value of t_{sk} can be assumed to be 36°C, just as it was in the equation for convection.

Evaporation (E) SI Units

E_{req} is the amount of heat that must be eliminated from the body by evaporation of sweat from the skin in order to maintain thermal equilibrium. However, there are major limitations to the maximum amount of sweat that can be evaporated from the skin (E_{max}):

(a) The human sweating capacity
(b) The maximum vapor uptake capacity of the ambient air
(c) The resistance of the clothing to evaporation

As described in Chapter 5[†], the sweating capacity of healthy individuals is influenced by age, sex, state of hydration, and acclimatization.

The draft ISO-WGTE [ISO 1982b][‡] standard recommends that an hourly sweat rate of 650 grams for an unacclimatized person and 1,040 grams for an acclimatized person are the maximum permissible for the average worker while performing physical work in heat. However, these sweating rate limits are not the maximum sweating capacities; they are related to levels of heat stress at which the risk of heat-related illnesses is minimal.

For a full work shift, the total sweat output should not exceed 3,250 grams for an unacclimatized person and 5,200 grams for an acclimatized person to prevent deterioration in performance due to dehydration. It follows from the foregoing that if heat exposure is evenly distributed over an 8-hour shift, then the maximum acceptable hourly sweat rate is about 400 grams for an unacclimatized person and 650 grams for an acclimatized person.

Thus, if the worker's heat exposure remains within the limits of the recommended standard, then the maximum sweating capacity will not be exceeded, and the limitation of evaporation will be due only to the maximum vapor uptake capacity of the ambient air. The E_{max} can be described with the equation recommended by the ISO-WGTE:

$$E_{max} = (p_{sk,s} - p_a)/R_e$$

where

E_{max} = maximum water vapor uptake capacity (Wm^{-2})
$p_{sk,s}$ = saturated water vapor pressure at 36°C skin temperature (5.9 kP_a)
p_a = partial water vapor pressure at ambient air temperature (kP_a)
R_e = total evaporative resistance of the limiting layer of air and clothing ($m^2kP_a\ W^{-1}$), which can be calculated by the following equation:

$$R_e = 1 / 16.7 / h_c / F_{pcl}$$

where

h_c = convective heat exchange coefficient (Wm^{-2} / C^{-1})
F_{pcl} = reduction factor for loss in latent heat exchange due to clothing (dimensionless) which

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[†] Chapter 4 of the current document.

[‡] This standard has been revised. See Ergonomics of the Thermal Environment: Analytical Determination and Interpretation of Heat Stress Using Calculation of the Predicted Heat Strain (Standard No. ISO 7933) [ISO 2004b].

can be calculated by the following equation:

$$F_{pcl} = 1 / 1 + 0.92h_c / I_{cl}$$

where

I_{cl} = Thermal insulation of clothing (m^2 °C W^{-1})

What this means is that the maximum vapor uptake capacity of the air depends on the temperature, humidity, and velocity of the ambient air and clothing worn. However, the relationship of these variables with respect to human heat tolerance is quite complex. Further complications are caused by the fact that in order to be able to evaporate a certain amount of sweat from the skin, it is necessary to sweat more than that amount, because some of the sweat will drip off the skin or will be picked up by the clothing. To calculate the additional amount of sweat required due to dripping, the ISO-WGTE recommended the following equations:

$$S_{req} = E_{req}$$

where

S_{req} = required sweat (Wm^{-2}). This quantity can also be expressed as ($g\ h^{-1}\ m^{-2}$) × 0.68
E_{req} = required evaporation (Wm^{-2}), which can be calculated by the equation $E_{req} = M + C + R$
η = Evaporative efficiency of sweating of a nude person, which can be calculated by the following equation:

$$\eta = 1 - 0.5 / e^{-6.6(1-w)}$$

where

e = the base of natural logarithm
w = E_{req}/E_{max}, also called the "wettedness index"

The experimental data are insufficient to calculate the loss of evaporative efficiency of sweat due to the wicking effect of clothing. However, if the workers wear thin knitted cotton underwear, this can actually enhance the cooling efficiency of sweat, because after wicking the sweat off the skin, it spreads it more evenly over a larger area, thus enhancing evaporation and preventing dripping. Since the thin knitted material clings to the skin, the evaporative cooling will affect the skin without much loss to the environment. If a loosely fitting garment wicks up the sweat, there may be a substantial loss in evaporative cooling efficiency. However, if the heat exposure (M+C+R) remains below the human sweating capacity, then the exposed worker's sweat excretion can increase to compensate for the loss of its cooling efficiency. A compensatory increase of sweating does not add much to the physiologic strain if water and electrolytes are replaced satisfactorily and if the water vapor uptake capacity of the ambient air is not exhausted.

In order to make sure that in the S_{req} index the wettedness modifies the value of S_{req} only to the extent to which it increases physiologic strain, the E_{req}/E_{max} ratio affects the value of S_{req} in an exponential manner.

The closer the value of E_{req} comes to E_{max}, the greater the impact of w on S_{req}. This is in accord with the physiologic strain as well as the subjective feeling of discomfort.

In this manner, the S_{req} index is an improvement over other rational heat stress indices, but at the same time the calculations involved are more complex. With the availability of pocket-sized programmable calculators, the problem of calculations required is greatly reduced. However, it is questionable whether it is worthwhile to perform a complex calculation with variables that cannot be measured accurately. These variables include the mean weighted skin temperature, the velocity and direction of the air, the body position and exposed surface area, the insulation and vapor permeability of the clothing, and the metabolic heat generated by the work.

For practical purposes, simplicity of the calculations may be preferable to all-inclusiveness. Also, the utilization of familiar units such as kcal, Btu, and W, (the British units or metric units instead of the SI suggested), to express energy in heat production may increase the use of the calculations. They can be useful in analysis of a hot job for determining the optimal method of stress reduction and for prediction of the magnitude of heat stress so that proper preventive work practices and engineering controls can be planned in advance.

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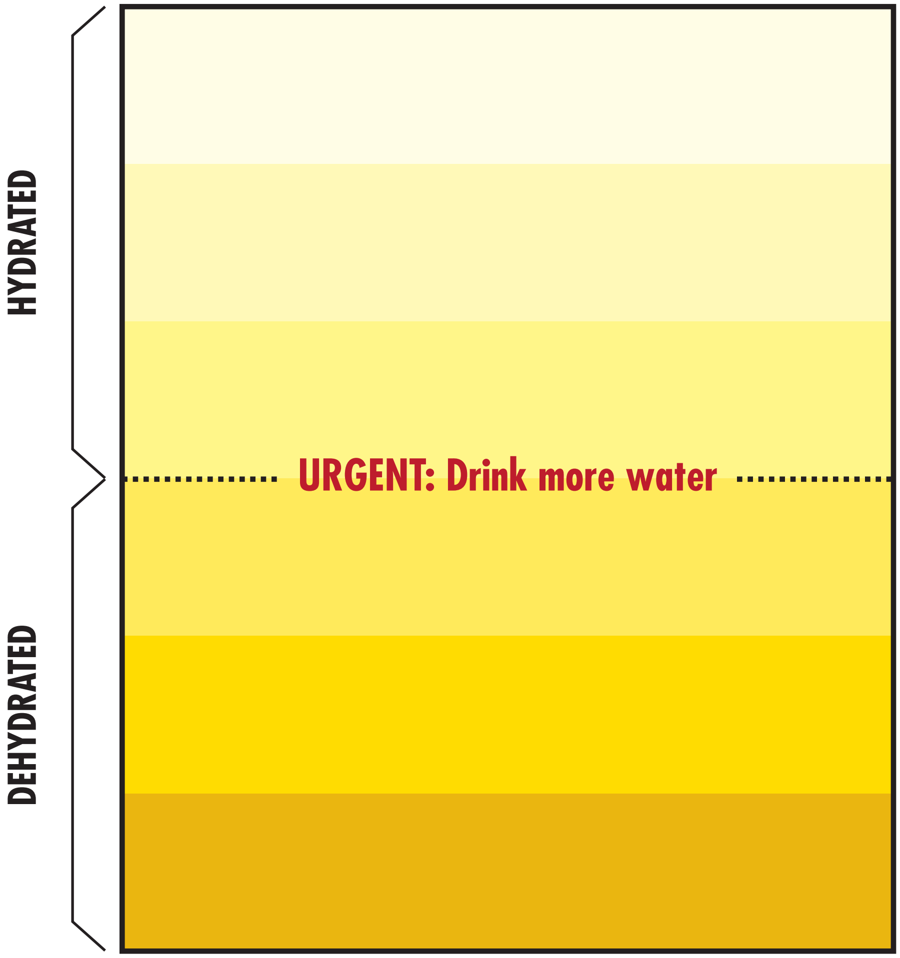
Appendix B

Urine Chart

Urine Color Chart

Are you hydrated?



Urine charts can be implemented as a training tool to demonstrate the concept of color change between the urine of a well-hydrated worker and that of a dehydrated worker. When conducting an investigation to evaluate the validity and sensitivity of urine color, Armstrong et al. [1998] found that urine color was as good an index as urine osmolality, urine specific gravity, urine volume, plasma osmolality, plasma sodium, and plasma total protein for tracking changes in body water and hydration status. In an earlier study, the author suggested that urine color could be used in workplace settings where close estimates of urine specific gravity or urine osmolality are acceptable [Armstrong et al. 1994, 2010].

Although the urine chart is a good indicator of hydration status for most workers with normal pale yellow to deep amber urine, urine color can also be affected by diet, medications, and illnesses or disorders. See Table B-1.

Table B-1. Causes of abnormal colors in urine

Color	Causes Diet	 Medications	 Medical conditions
Clear		▪ Diuretics	
Cloudy or milky			▪ Urinary tract infection ▪ Bacteria ▪ Crystals ▪ Fat ▪ White or red blood cells ▪ Mucus
Yellow	▪ Vitamins		
Orange	▪ B complex vitamins ▪ Carotene ▪ Carrots	▪ Rifampin ▪ Sulfasalazine (Azulfidine) ▪ Phenazopyridine (Pyridium) ▪ Laxatives ▪ Chemotherapy drugs	▪ Medical conditions (liver or bile duct)
Red or pink	▪ Beets ▪ Blackberries ▪ Rhubarb	▪ Rifampin (Rifadin, Rimactane) ▪ Phenazopyridine ▪ Laxatives containing senna	▪ Blood (infection or cancer) ▪ Toxins (chronic lead or mercury poisoning)
Port wine or purple			▪ Porphyria (inherited disease)
Green or blue	▪ Food dyes	▪ Amitriptyline ▪ Indonethacin (Indocin) ▪ Propofol (Diprivan) ▪ Medications containing methylene blue	▪ Familial hypercalcemia (inherited disorder) ▪ Urinary tract infection with *Pseudomonas* sp.
Brown	▪ Fava beans ▪ Rhubarb ▪ Aloe ▪ Kidney or liver disorders (cirrhosis)	▪ Antimalarial drugs (chloroquine, primaquine) ▪ Antibiotics (metronidazole, nitrofurantoin) ▪ Laxatives containing cascara or senna ▪ Methocarbamol (muscle relaxant)	▪ Urinary tract infections

Adapted from Mayo Clinic 2011; Medline Plus 2011; Watson 2011.

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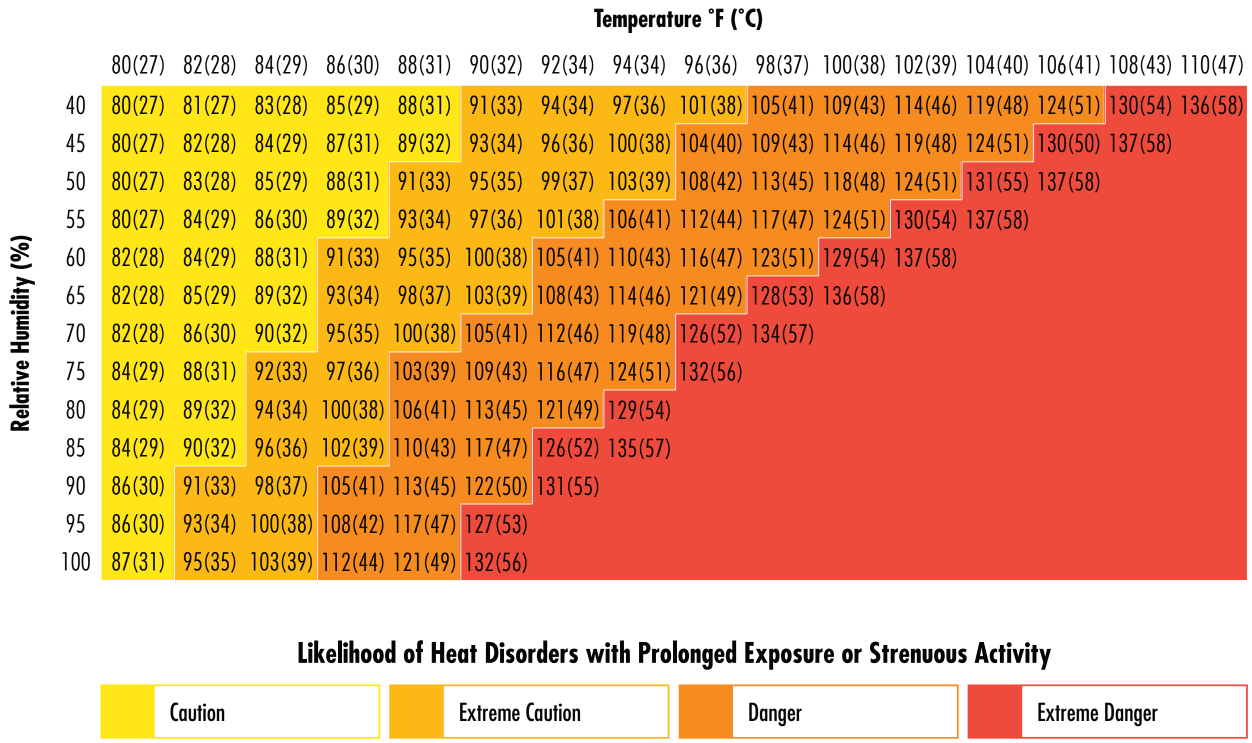
Appendix C

Heat Index

NOAA's National Weather Service

Heat Index



Temperature °F (°C)

Relative Humidity (%)	80(27)	82(28)	84(29)	86(30)	88(31)	90(32)	92(34)	94(34)	96(36)	98(37)	100(38)	102(39)	104(40)	106(41)	108(43)	110(47)
40	80(27)	81(27)	83(28)	85(29)	88(31)	91(33)	94(34)	97(36)	101(38)	105(41)	109(43)	114(46)	119(48)	124(51)	130(54)	136(58)
45	80(27)	82(28)	84(29)	87(31)	89(32)	93(34)	96(36)	100(38)	104(40)	109(43)	114(46)	119(48)	124(51)	130(50)	137(58)	
50	80(27)	83(28)	85(29)	88(31)	91(33)	95(35)	99(37)	103(39)	108(42)	113(45)	118(48)	124(51)	131(55)	137(58)		
55	80(27)	84(29)	86(30)	89(32)	93(34)	97(36)	101(38)	106(41)	112(44)	117(47)	124(51)	130(54)	137(58)			
60	82(28)	84(29)	88(31)	91(33)	95(35)	100(38)	105(41)	110(43)	116(47)	123(51)	129(54)	137(58)				
65	82(28)	85(29)	89(32)	93(34)	98(37)	103(39)	108(43)	114(46)	121(49)	128(53)	136(58)					
70	82(28)	86(30)	90(32)	95(35)	100(38)	105(41)	112(46)	119(48)	126(52)	134(57)						
75	84(29)	88(31)	92(33)	97(36)	103(39)	109(43)	116(47)	124(51)	132(56)							
80	84(29)	89(32)	94(34)	100(38)	106(41)	113(45)	121(49)	129(54)								
85	84(29)	90(32)	96(36)	102(39)	110(43)	117(47)	126(52)	135(57)								
90	86(30)	91(33)	98(37)	105(41)	113(45)	122(50)	131(55)									
95	86(30)	93(34)	100(38)	108(42)	117(47)	127(53)										
100	87(31)	95(35)	103(39)	112(44)	121(49)	132(56)										

Likelihood of Heat Disorders with Prolonged Exposure or Strenuous Activity

🟨 Caution	🟧 Extreme Caution	🟧 Danger	🟥 Extreme Danger

Adapted from NOAA [2012].

The National Oceanic and Atmospheric Administration (NOAA) issues heat alerts based on the heat index values, as seen in the chart above. The Heat Index is a measure of how hot it feels when RH is taken into account with the actual air temperature. Since heat index values were devised for shady, light wind conditions, exposure to full sunshine can increase heat index values by up to 15°F.

NOAA may also issue an extreme heat advisory:

- *Excessive Heat Outlook*
  Extended excessive heat (heat index of 105°F–110°F [41°C–43°C]) over the next 3 to 7 days.
- *Excessive Heat Watch*
  Excessive heat may occur within the next 24 to 72 hours.
- *Excessive Heat Warning*
  The heat index will be life threatening in the next 24 hours. Excessive heat is imminent or has a high probability of occurring.
- *Excessive Heat Advisory*
  The heat index may be uncomfortable but not life threatening if precautions are taken.

NOAA uses four bands of colors associated with four risk levels. The risk level–related measures in Table C-1 have been modified by OSHA for use on worksites.

Table C-1. Heat index–associated protective measures for worksites

Heat index	Risk level	Protective measure
Less than 91°F (33°C)	Lower (caution)	Basic health and safety planning
91°F to 103°F (33°C to 39°C)	Moderate	Implement precautions and heighten awareness
103°F to 115°F (39°C to 46°C)	High	Additional precautions to protect workers
Greater than 115°F (46°C)	Very high to extreme	Even more aggressive protective measures

Adapted from OSHA [2012c].

Additional information about protective measures mentioned in the above table can be found on OSHA's website.

Note: The presence of a radiant heat source may decrease the accuracy and usefulness of the above heat index.

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