Chapter 5

Desert and Tropical Environment

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5.1 INTRODUCTION

When thinking of a hot environment, one usually starts with the impressions of the vast, sandy desert areas around the world. One thinks of the imminent danger of dehydration and death due to lack of potable water.

However, in case of the high altitude deserts in Chile (Atacama) or central Antarctica, extremely cold temperatures can prevail, especially during winter and night-time. From a meteorological-geographical perspective, deserts can be differentiated as extremely arid, semi-arid, and arid areas. Thus, not only the ambient temperature but also the amount of precipitation must be taken into account (Figure 5.1).

From a physiological point of view and in respect to the availability of freshwater, the largest deserts for humans are the oceans. A shipwrecked human, although immersed and surrounded by a gigantic mass of water, cannot drink a drop of it because it would vastly increase dehydration due to its high salt content (3.5%) [1]. Another general misconception—at least for those people who grew up in the moderate latitude of this planet such as the Caucasians—is that living in the tropics is often taken as a synonym for an easy life style and a place for vacations. Due to the permanent (12 months) mean high ambient temperatures (18 °C and higher) combined with an average precipitation of at least 60 mm of rain (high relative humidity), and less air movement [2], the tropical rainforest environment is a very stressful climate for nonadapted humans [3–5]. From a physiological point of view, the cardiovascular system is permanently stressed, day and night. Therefore, it is not surprising that the tropical climate is also called the "white man's grave."

Because thermoregulation in humans is quite complex, a basic physical understanding of heat transfer, that is, convection, conduction, radiation, and evaporative heat loss, is needed, as well as knowledge about the multiple autonomic pathways involved in maintaining core body temperature.

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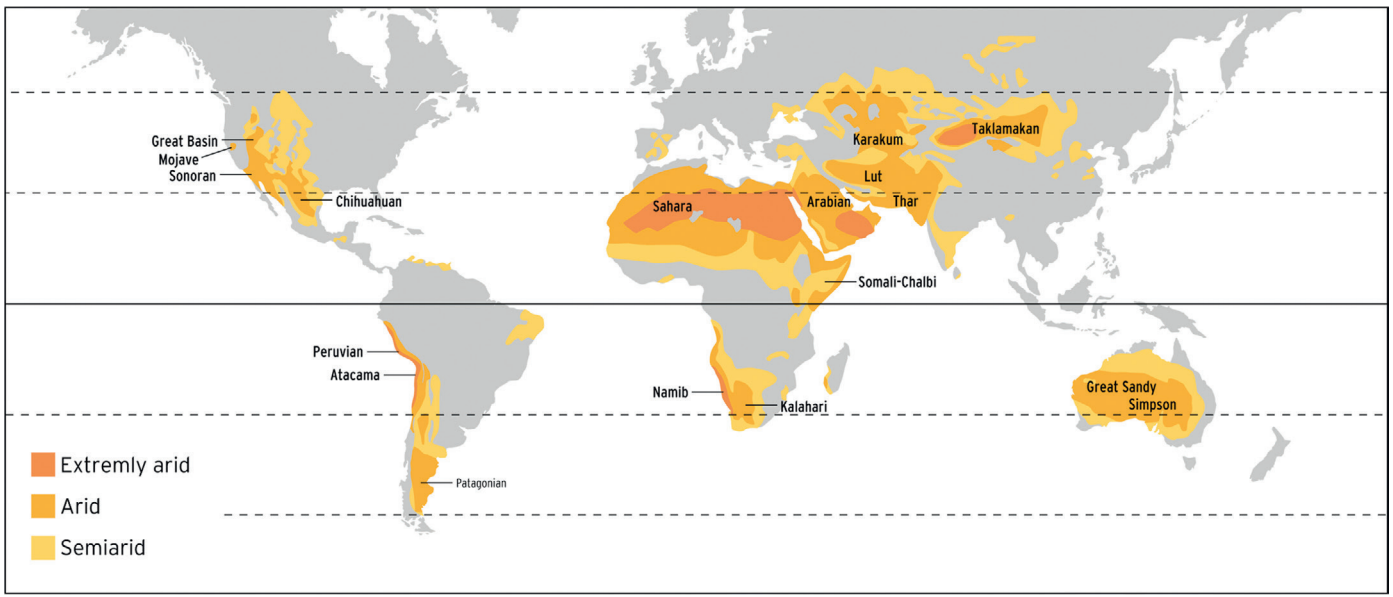

FIGURE 5.1 The main great deserts around the world, with exception of the polar deserts.

5.2 THERMAL BALANCE

5.2.1 Ectothermic and Endothermic Metabolism

Humans are endothermic organisms. This means that in contrast to the ectothermic (poikilothermic) animals such as fishes and reptiles, humans are less dependent on the external environmental temperature [6,7]. Endothermic organisms have much higher basal energy consumption, which is mainly necessary to keep their body temperature constant within a wide range of different environmental temperatures. In the body core (cranial, thoracic, abdominal cavities), the human body temperature is around 37 °C. In the periphery (extremities), it is lower and exhibits regional differences (28-36 °C) (Figure 5.2). Under ambient environmental conditions, the core body temperature of 37 °C is maintained by

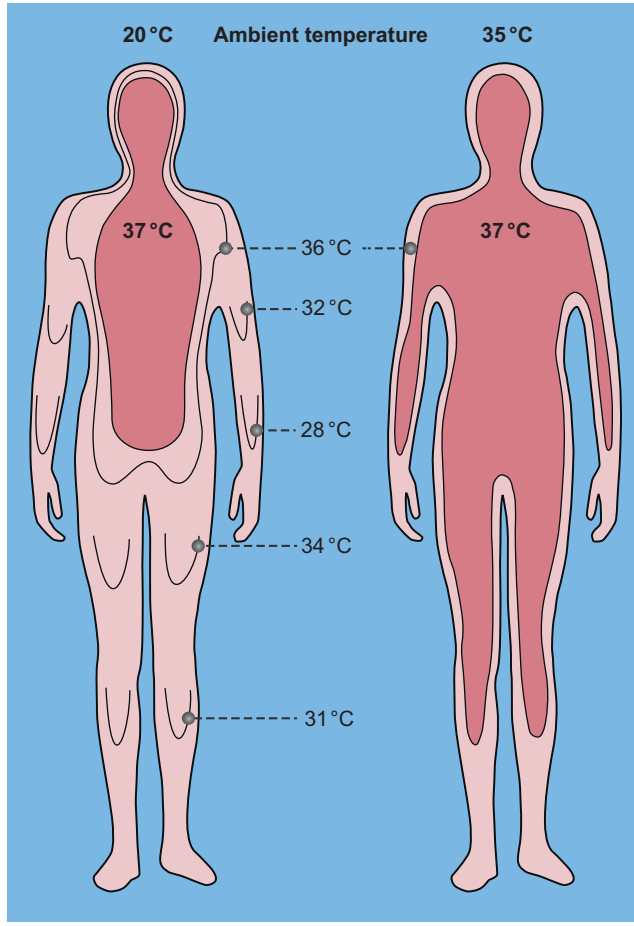

FIGURE 5.2 Body core and body shell temperatures under different ambient temperatures in a cold (20 °C, left panel) and a hot environment (35 °C, right panel). *Adapted from [94].*

the permanent metabolic active internal organs such as brain, heart, liver, and gastrointestinal tract through a fine-tuned thermoregulatory system that mainly adjusts peripheral perfusion of the skin and evaporation by the sweat glands to the thermal needs of the body [6,8]. Heat loss, under resting conditions, occurs mainly via radiation. In a warm/hot environment and under strenuous exercise, the organism depends on the evaporative pathway (Figure 5.2).

However, the core body temperature is not consistently regular. In the course of the day, the core body temperature shows cyclic changes (circadian rhythm), and in women a monthly pattern occurs due to the menstrual cycle. In ectothermic organisms (amphibia, reptiles, fishes), the temperature gradient compared to the environment is low (<5 °C). Their body temperature, and thus their activity, depends largely on the prevailing external environmental conditions (thermo-conform). Thus, these organisms remain viable (eurytherm) over a wide temperature range and, due to a low metabolism (bradymetabolism), they can overcome longer phases of food shortage. The temperature field of humans can be subdivided into a body shell and a body core (Figure 5.2) [6,8,9]. A core body temperature of more than 37.5 °C is defined as hyperthermia; below 35.5 °C is known as hypothermia. By definition, the parts of the body where the tissue temperatures exhibit predominantly 37 °C are considered the body core (homeothermic). Particularly in the metabolically active organs such as the heart, brain, and liver, heat is produced permanently. In the limbs and skin, mean tissue temperature can vary extensively (poikilothermic body shell). The tissue temperature of the body shell decreases with increasing distance from the body core, and the shell crowds around it like onion layers (isotherms). The body shell functions on the one hand as heat insulation of the body core. On the other hand, the heat exchange with the environment takes place at the surface. The relation of body core to the shell is not kept constant. If heat is to be emitted, the body core expands to the skin level, therefore facilitating heat transfer via the enlarged subcutaneous vascular bed. In a cold environment, heat loss has to be reduced to prevent hypothermia. Thus, the body shell has to be enlarged via vasoconstriction, which leads to a better insulation of the core (Figure 5.2). Humans require a constantly high core body temperature (homiothermic = equally warm) between 36.4 and 37.4 °C. Variations of the core body temperature are tolerated only in a very small range (stenothermic). In order to assure this constant core body temperature in a cold environment, insulating layers are necessary to reduce heat losses [8]. Furthermore, a correspondingly high heat production via endothermia and tachymetabolism is required. This causes the metabolic rate of endothermic organisms to be three to four times higher than that of ectothermic organisms. Fluctuations in air temperature, relative air humidity, and airflow, as well as internal heat production require an effective system of thermoregulation. Through these mechanisms, endothermic organisms maintain a high temperature gradient compared to environmental temperatures. This enables them to be more active than ectothermic organisms at the cost of a higher energy consumption [8,10].

5.2.2 Neutral Temperature Zone

The neutral or indifferent temperature zone is defined as a range of environmental temperatures in which a naked, resting person can keep an even heat balance through changes in skin blood flow. It is standardized to basal metabolic conditions, that is, a healthy, unclothed, resting adult at a relative air humidity of 50% minimal air movement and a temperature of approximately 27-31 °C (Figure 5.3) [10].

Above an ambient temperature of 31 °C, heat dissipation is mainly regulated by evaporation, whereas the contributions of radiation, conductance, and convection decrease. At an air temperature >37 °C, the body actually gains heat from the environment (reversal heat flux) [8]. As shown in Figure 5.3, the indifference temperature for an unclothed, resting human under atmospheric conditions lies around 27-31 °C. In water, however, the indifferent temperature is defined as an even smaller range (34.5-35.5 °C). This is caused by the special physical properties of water: Water has a thermal conductivity 25 times higher and a specific heat capacity 4000 times higher than air [6,8]. In contrast to air, heat transfer for convection and conductance is increased in water. Thus, submerging in water temperatures below 25 °C, heat losses cannot be compensated for by means of internal heat production [6]. Once hypothermia begins, the viscosity of blood and musculature activity increase. In addition, the heat

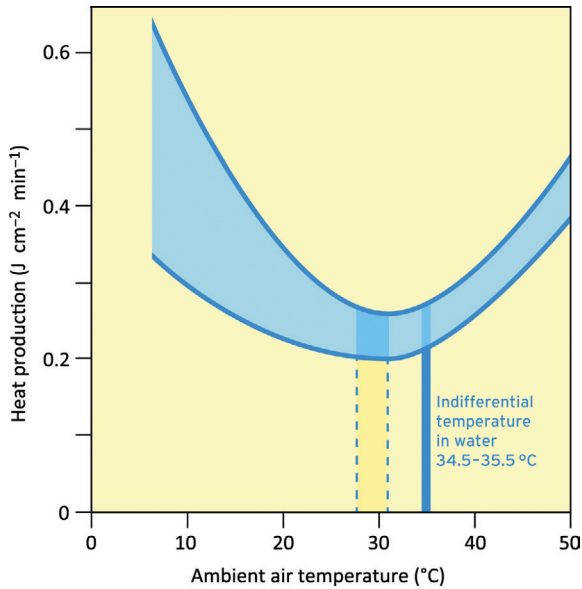

FIGURE 5.3 The thermal neutral or indifferent zone in a resting human under atmospheric conditions (scattered range) and the changes in metabolic heat production. As indicated, in water (solid line) the thermal neutral zone is much higher and very narrow. For further information, see text. *Adapted from Ref. [10].*

loss coefficient is approximately tripled with active swimming movements. The velocity in which nerve signals are conducted are reduced. A falling blood temperature shifts the oxygen binding curve to the left, thus improving the uptake of oxygen in the lungs, but the delivery of oxygen to the tissue, however, is thereby strongly diminished. On the other hand, hot water immersion leads to a rapid increase in core body temperature.

5.3 HEAT PRODUCTION

5.3.1 Metabolic Heat Production and Metabolic Rate

Living cells absorb high-energy nutrients, metabolize them, and finally excrete substances of lower energy [10]. The process of transformation of the energy from nutrients into internal energy forms and their utilization is described as basal energy rate. Two terms have to be distinguished in this process: (i) metabolic heat production = transformation of chemical energy into heat, and (ii) metabolic rate = rate of transformation of chemical energy into heat and mechanical work. Under resting conditions, the metabolic rate corresponds to the metabolic heat production. The chemical energy of the ingested food products can thus be utilized for heat production, muscle activity, and for the synthesis of adenosintriphosphate (for further details see Chapter 3 and Ref. [10]). Because the efficiency factor of mechanical muscle work lies only around 20-30%, approximately 70-80% of the chemical binding energy of the nutrients burned for external work accumulates as additional heat. This strongly influences the thermal comfort of the subject [8]. In order to remain within thermal balance, heat loss also has to be increased. The higher the basal energy rate and insulation value of the clothing, the lower is the ambient temperature of thermal comfort. For the basal energy rate during different activities, the unit MET (metabolism) is used: 1 MET designates the energy rate during sitting activity, approximately 300 mL of O_2 consumption, equivalent to an energy need of 400 kJ/h. At the same time, the influence of air movement and the kind of clothing have to be taken into account, because the boundary air layer lying around the body (microclimate) contributes to the heat insulation. An insulation value of 1 clo (deduced from the English word "clothing") corresponds to 0.155 m^2 K/W. One might use as a rough approximation: thermal insulation (clo) = 0.08 × total number of layers + 0.51 [5]. This insulation value quickly decreases with higher wind velocities. This is mainly because the boundary layer is thinner and the laminar heat convection along the body axis passes into a turbulent current [6].

5.3.2 Thermal Balance under Different Physical and Environmental Settings

Approximately 80% of the heat within the internal organs is produced at physical rest, whereas the other parts of the body contribute only 20%. Under resting conditions, the organs with high heat production (60-140 J 100 $g^{-1}min^{-1}$) are

situated exclusively in the cranial, thoracic, and abdominal cavities, thus within the body core. The body shell, on the other hand, contributes only slightly to heat production. With physical work, heat dissipation changes fundamentally. Up to 90% of the entire heat production can then be ascribed to the working musculature, and thus the tissue temperature in muscles can be distinctly above core body temperature. Because subcutaneous fat in limb muscles is rather sparse, and the tissue is well supplied with blood, heat can be dissipated rather easily. Thus, perfusion of the working muscles not only serves for delivery and removal of metabolic products, but also for thermoregulation. Because the thermal conductivity of nonperfused tissue has the same dimension as fat tissue, the dissipation of heat depends mainly on the variation in this perfusion, specifically by opening arteriovenous anastomoses in the skin serving as as "thermal windows" of the body [8].

5.3.3 Influencing Factors

A constant core body temperature presupposes that heat production and heat losses are in balance. For other mammals and birds, this core body temperature is independent of their weight and volume, lying between 36 and 40 °C. The necessary basal metabolism increases with the body mass. This metabolism of an endothermic organism is determined both by the heat-producing body mass and the heat-emitting body surface. The specific metabolic rate of an organism is obtained by dividing the metabolic rate by the body mass, which means, for example, that the specific metabolic rate of the Etruscan shrew (0.002 kg) per time unit is approximately 175 times larger than that of the elephant (10,000 kg; law of metabolic reduction). This is due to the surface-volume ratio. As the body mass, i.e. body volume, increases with the third power (m^3), the body surface, however, only with the second power (m^2), accordingly small organisms (i.e., newborn infants) have an unfavorable surface-volume relation [10].

5.4 METHODOLOGIES OF CORE BODY TEMPERATURE MEASUREMENT

The temperature measurements practiced most frequently, sublingual and axillary, exhibit the largest source of errors. As a rule, by using axillary temperature measurement, one determines only the peripheral temperature and not the core body temperature. For a correct recording of the core body temperature, the sensor would have to be tightly pressed into the axilla for 30-40 min, which is the time it takes the body core to extend into the axilla (Figure 5.2). When measuring sublingual temperature, the position of the thermometer at the root of tongue varies by approximately 0.6-0.8 °C, according to [6,8]. Under experimental conditions, the core temperature is measured by inserting a thermosensor in the esophagus, nasopharnyx, rectum, or tympanum/auditory meatus. However, none of these methods are really applicable outside the laboratory, especially during field research

in extreme environments. This is due to the fact that the requirements for a method to accurately measure core body temperature are demanding. Basically, a thermosensor has to be (i) noninvasive, (ii) easy to handle, (iii) meeting basic hygiene standards, and (iv) not biased toward various environmental conditions, while (v) changes should quantitatively reflect small changes in arterial blood temperature and (vi) last but not least, the response time of the thermosensor to temperature changes should be as short as possible [11,12]. These requirements are essential. Previous studies in humans have shown that when high environmental temperatures and humidity prevail, the heat load will cause a rapid rise in core body temperature. This may result in heat stress-related injuries such as orthostatic collapse or even heat stroke [8]. If there are thermoregulatory impairments due to fever or drugs, the deleterious effects may occur even faster [13,14].

The tissue temperature of each organ depends on local metabolism and perfusion of the respective organ. Both factors are able to evoke measurable temperature differences within one organ. Thus, it becomes evident that the temperature of the organs in cranial, thoracic, and abdominal cavities deviates approximately 0.4 °C from the arterial blood temperature as reference value. Currently, there is no accurate and easy method to measure core body temperature in a field setting. The relative advantages and disadvantages of core temperature measurements and measurement sites, including the time response of the different kind of sensors, have been intensively discussed ever since the first benchmark investigations on this topic by Claude Bernard in 1876 [15–20]. Most thermal physiologists agree that the esophageal temperature is close to the best noninvasive index of core temperature for humans. It correlates well with changes in central venous blood temperature [12,16,21]. The esophageal temperature is obtained by inserting a catheter containing a thermocouple or thermistor through the nasal passage into the throat and then swallowing it. Although appropriate for research settings and during surgery, it is highly inappropriate in ambulatory or field assessments. This holds true for other sites such as rectal or tympanic probes as well, because they are all impractical for use in the field. Rectal temperature is obtained by inserting a temperature sensor at a minimal depth of 8 cm past the anal sphincter [14,22]. During exercise it takes approximately 25-40 min to achieve a steady-state rectal temperature value [15,22,23]. This is 0.2-0.3 °C higher than simultaneously measured nasopharyngeal and esophageal temperatures under resting and thermoneutral (27 °C) environmental conditions [17,19,23,24]. Rectal as well as esophageal temperatures are largely independent of the environmental temperatures [19,25]. As a result, the steady-state rectal temperature provides a good index to assess body heat storage [24,26]. The main problem with the rectal temperature is that it shows a slow response in comparison to the other measurement sites, a fact that recently was proven again in 60 patients who underwent a post-operative rewarming [26]. The reason for the slow response is probably (i) a low rate of blood flow to the rectum compared to other measurement sites [21,27] and (ii) the mass of

organs located in the body cavity. This greater mass of tissue in the lower abdominal cavity requires a far greater amount of energy to cause a rapid temperature change. Tympanic temperature is obtained by inserting a small temperature sensor into the ear canal in direct contact with the tympanic membrane. Some subjects perceive this to be uncomfortable [8]. In addition, there are reports of the temperature sensor's perforating the tympanic membrane [28–30]. Because of the potential discomfort and trauma, as well as placement problems associated with tympanic measurements, some investigators chose to measure the temperature at the external auditory meatus. For this measurement, a temperature sensor is placed in an ear plug. Proper placement of the probe is vital because there is a substantial (~0.5 °C) temperature gradient along the wall of the meatus. In addition, several studies have shown that the auditory meatus temperature measurements do not provide a reliable index of the level of core body temperature during either rest or exercise [11,22,23,31] In regard to environmental conditions, these temperature values might be lower or higher than simultaneously measured steady-state rectal [22] and esophageal temperature values [31–33]. In addition, local heating of the head and/or increased air flow to the face will bias the tympanic temperature [22,23,31]. The best and most reliable method of assessing thermal state in operational environments is direct measurement of core body temperature using a network-enabled ingestible core temperature sensor [34]. However, the use of the latter is impractical for routine application. Thus, these devices are reserved for use during high thermal stress missions, while encapsulated in nuclear, biological, and chemical protective suits, and/or if use is indicated by medics during combat casualty care such as cooling interventions in case of heat injuries [20]. Skin surface can be accessed much easier than other core body temperature measurement sites mentioned above, although this site has many flaws because skin temperature is largely affected by cutaneous blood flow and/or sweat evaporation. Furthermore, environmental changes such as air temperature, humidity, wind speed, and radiation will alter skin temperature as well. Therefore, thermal physiologists prefer to determine mean skin temperature as a sum of weighted individual skin temperatures taken at different skin surface areas up to 16 sites, and for certain questions even more [8]. It is obvious that for most cases such a complex, heavy wired temperature measurement setup is currently highly impractical. Therefore, most recently Gunga et al. have introduced a combined skin temperature and heat flux sensor (Double Sensor) [35,36] (Figure 5.4).

In contrast to similar methodological attempts in the past [37,38], this new miniaturized heat flux sensor can be used without extra heating and comes in a special capsule [35]. It is placed at the vertex or at the front of the head and has been tested under various physical and environmental conditions, such as changing workloads and ambient temperatures of 10, 25, and 40 °C. A measurement comparison of the new sensor with the rectal temperature revealed that the Double Sensor (i) differed up to 0.06 °C from the average of the rectal temperature, (ii) showed with increasing ambient temperatures increasing concordance

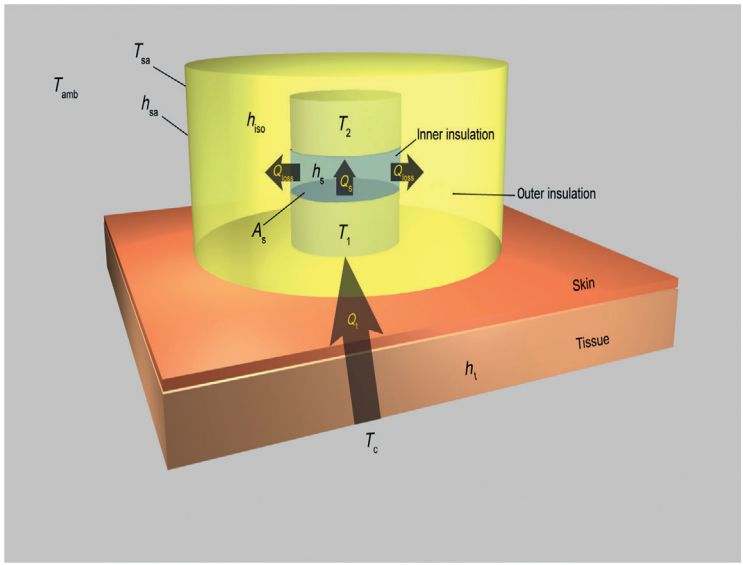

FIGURE 5.4 The technical scheme of the heat flux sensor (Double Sensor) placed at the front of the head to monitor continuously noninvasive core body temperature (T_c) under different environmental and physical conditions. T_c is calculated by measuring the heat flux dQ/dt from the body core to the outer sensor T_2. Heat loss ($\dot{Q}_{loss}$) is taken into account (see Refs. [35,36]).

correlation coefficients, and (iii) exhibited a more rapid response to the core body temperature changes for all resting periods and at all ambient conditions as compared to rectal temperature. However, we observed limitations of the heat flux sensor in cold environments, which have to be investigated further.

5.5 BASICS OF HEAT TRANSFER

5.5.1 Internal Heat Transfer

The heat exchange between two objects is proportional to the difference of their temperatures. For the process of heat loss, internal heat has to be transported from the various tissues and organs to the cooler body surface. Heat transfer from the body core to the shell is called internal heat transport. With regard to thermoregulation, heat loss is under sympathetic control. Local mechanisms are also involved in addition to the systemic regulation of blood circulation. Under high physical strain, the skin vessels, for example, in the thorax, are dilated to excess [8]. This increased vasodilation is induced by bradykinin and other mediators excreted with sweat. An increase in internal heat production and/or external heat stress therefore also leads to an increase in skin perfusion. Due to their geometry, these parts of the body possess a large surface in relation to their volume, which allows an easier dissipation of heat. Under heat stress the deep arteriovenous anastomoses in the acrae are largely closed, and the arterial

blood from the body core is redirected into the opening venous vascular bed of the skin [6,9]. Thus, this anatomical particularity promotes heat loss to the environment.

On the other hand, closure of deep arteriovenous anastomoses can inhibit the return of heat to the body core and prevent overheating. The high efficiency of the acrae to regulate the internal heat current lies, above all, in the large variability of their blood circulation (finger 1:600, hand 1:30, trunk 1:7) [10].

5.5.2 External Heat Transfer

Environmental conditions such as ambient air temperatures, radiation, relative air humidity, or wind velocity, as well as the skin temperature and the effective body surface are parametric for heat transfer from the body surface to the environment. In this external heat transfer, four different pathways play a role: convection, conduction, radiation, and evaporation (Figure 5.5) [8,10].

5.5.2.1 Convection

The convective heat exchange between body surface and environment takes place mainly in an air layer (boundary layer) that is merely a few millimeters thick and lies above the skin. In convective heat transfer, two forms have to be distinguished: natural and forced convection.

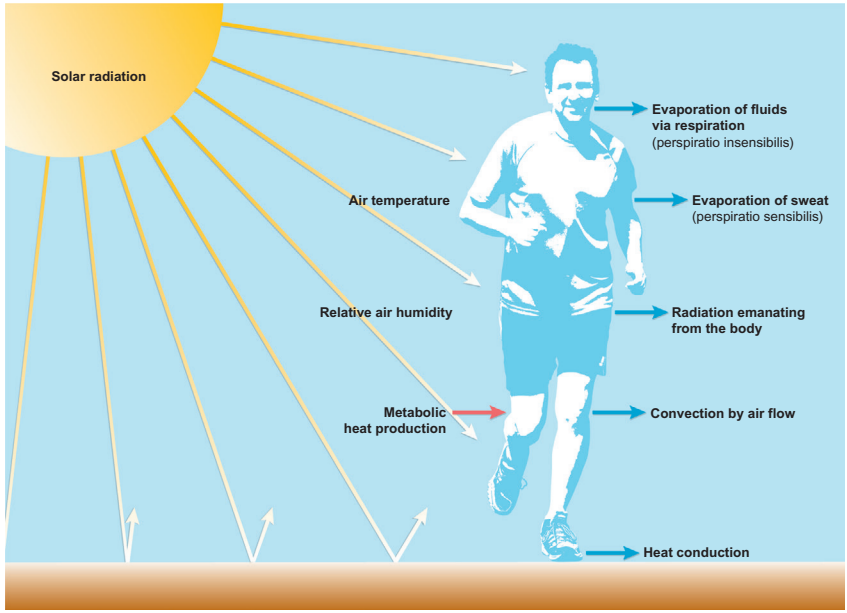

FIGURE 5.5 Parameters affecting heat balance during exercise and different heat transfer mechanisms.

5.5.2.1.1 Natural Convection

If a warm body is situated in a cooler medium, for example, air or water, a mass movement along the body axis of cooler parts toward warmer parts occurs in an upward direction in the medium. In doing so, the medium transports heat convectively. Under atmospheric conditions in an unclothed person, this convective mass transport amounts to approximately 600 L air per minute. In space, that is, weightlessness, this mass transport does not take place in astronauts, which contributes to a thermal discomfort (for further details, see Chapter 7). The amount of heat emitted in humans can be estimated by the assumption that 3 W of heat are dissipated in 1 m^2 effective exchange surface area per centigrade temperature difference between skin and ambient temperature. Thus, at 25 °C ambient temperature and a mean skin temperature of 33 °C a total heat loss of 24 W/m^2 will be achieved. With a surface-related basal metabolic rate of an adult of 0.1-0.2 $J/(cm^2\ min)$, this amounts to 4000-4350 kJ/day (45-50 W/m^2) [6].

5.5.2.1.2 Forced Convection

Forced convection exists when a body is transferred into a moving medium (wind, water current) or it is moved through this medium. Thus, the size and shape of the object play an important role in heat loss, meaning that the heat loss per square unit in small organisms (such as a mouse) by far exceeds that of large organisms (such as an elephant). In small organisms, forced convection can thus quickly lead to disturbances of the heat balance, especially when the thickness of the boundary layer is decreased by the forced convection and a laminar current in the boundary layer is transferred to a turbulent one. Convective heat losses also develop via the respiratory tract. However, in humans this mechanism of heat loss is small compared to other mammals (dog, horse).

5.5.3 Conduction

The direct heat transport between two solid substances in physical contact is termed conduction. The heat flows from the substance with a higher temperature to that with a lower. On an atomic level, heat is exchanged in the form of kinetic energy among the neighboring atoms. This means that, in contrast to the convective heat transport, no mass is transported. The rate of conductive heat transport between two objects depends on their temperature difference, the effective exchange area, and the material properties, as well as their special thermal conductivity. Silver, for example, has a very high thermal conductivity (430 W/m K), whereas air possesses merely a very low conductivity (0.024 W/m K) [6,8]. Based on its low heat conductivity, air can function as an excellent insulator. The amount of heat transported by the blood from the body core to the skin surface is conductively absorbed in the resting boundary layer and dissipated convectively by the air movement. In the external heat transport, conduction only takes place when skin surface areas come into direct contact with materials of high heat conductivity (e.g., metals) [6]. In such cases, an

unprotected contact can lead to immediate burning or freezing, respectively. The thicker the resting boundary layer around the body, the lower the heat transport between body surface and air. By choosing proper clothing, humans can increase or decrease the thickness of their boundary layer. Here the amount of the air enclosed within the clothing is decisive for the insulation value. The higher the latter, the larger the enclosed amount of air [10].

5.5.4 Radiation

Each substance with a temperature above the absolute zero point (−273.15 °C) emits an electromagnetic radiation of a certain wavelength. The wavelength emitted depends on the surface temperature and is inversely proportional to the latter. Short wavelengths are thus radiated by hot objects, and long wavelengths are radiated by cool objects [8]. Humans and animals are relatively cool objects within the temperature spectrum, thus they radiate in the long-wave infrared range [6]. The surface temperature is not only decisive for the wavelength emitted, but also for the rate at which a body emits radiation energy. Under resting conditions with an air temperature of 20-25 °C, relatively low air humidity and low windspeed, humans can emit approximately 50-60% of their total heat production via infrared radiation to the environment (Figure 5.6) [6].

The remaining portions are distributed more or less equally to convection/conduction and evaporation (Figure 5.6) [6]. For the amount of heat loss or heat

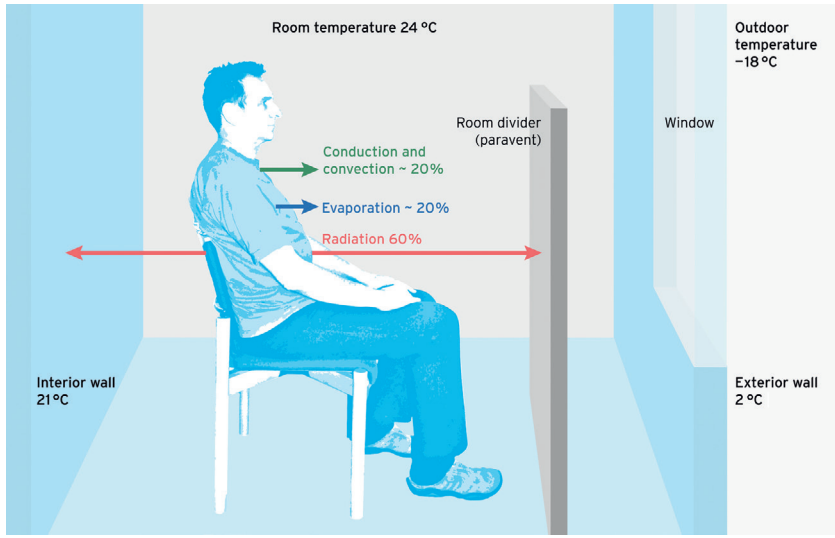

FIGURE 5.6 Heat loss pathways and their percentage in a lightly clothed human (0.5-0.6 clo) under resting conditions. Conduction and convection (green), radiation (red), and evaporation (blue) at a room temperature of 24 °C are shown in the figure. A screen decreases the heat loss by radiation. Further details are given in the text. *Adapted from Ref. [10].*

gain via radiation, the surface temperatures of the objects and walls nearest to the body surface are decisive, because the greater the temperature difference between the two, then the greater the heat loss or heat gain would be, respectively. With cool external or window temperatures, a screen or a curtain placed before it with a surface temperature aligning with the mean room temperature can considerably decrease the heat loss (Figure 5.6) [6]. An unprotected exposure of the body to significantly cooler surfaces leads to a decrease of the local skin temperature, which leads to the activation of cold receptors in the skin and to vasoconstriction. Then the concerned skin areas and the musculature lying below the skin proceed to cool further, producing a "cold sensation." Chilling and muscle tension can occur as a consequence of longer exposure. On the other hand, objects with a surface temperature higher than the skin (mean human skin temperature 32-33 °C) supply the organism with heat by means of radiation (radiant heater, furnace) [6,8].

5.5.5 Evaporation

Passively, the organism loses water by diffusion through the skin and the mucosa of the respiratory system (perspiratio insensibilis, extraglandular release of water) [10].

5.5.5.1 Perspiratio Insensibilis

The amount of fluid lost in an adult human by perspiratio insensibilis adds up to approximately 20-30 mL/h, that is, 400-600 mL/day at 33 °C ambient temperatures. Under thermoneutral environmental conditions and with 50% relative air humidity, this amount of evaporation leads to a simultaneous heat loss of about 20-30% of the daily metabolic rate (passive evaporative heat loss) at sea level. At high altitude or in extreme cold environments these losses can be much higher, especially during physical exercise [10].

5.5.5.2 Perspiratio Sensibilis

Physically active humans can excrete fluid via the sweat glands (perspiratio sensibilis, glandular release of water). By means of the evaporation of sweat, the organism can lose a considerable amount of heat, because in the transition from a liquid to a gaseous state (water vapor) energy is required (endothermic process). With complete evaporation, an amount of sweat of about 2 g/min is sufficient to dissipate the entire heat production of an adult under resting conditions (80-90 W). Because adults can produce a maximum of 10-15 g/min sweat per square meter, evaporation is the key mechanism of heat dissipation during heavy physical exercise and/or external heat stress [10]. In addition to adequate hydration, it is essential that the water vapor pressure produced by the sweat glands lies above that of the environment. The higher the water vapor pressure in the environmental air (high air humidity, tropical climate), the more difficult the heat loss via evaporation gets. That is why tropical climates are regarded as very stressful environments for nonadapted Caucasians, for example. With a low relative humidity in

the air (dry desert climate), however, humans can also tolerate extremely high air temperatures and external heat load for a short time, because the gradient of the water vapor pressure from the skin to the environment is very large. According to Folk and Semken [39], this special anatomical-physiological feature (sweat glands) found in primates evolved in humans during the Tertiary Age in concert with bipedalism and a smooth hairless skin. Today, the following kinds of glands can be distinguished: (i) sebaceous (oily), (ii) apocrine, and (iii) eccrine (sweat) glands. The sebaceous glands can be found evenly distributed along the body axis with exception of the palms and soles. In some areas of the body, such as the scalp and face, the number of sebaceous glands can be extremely dense (900/cm^2). As shown in the schematic cross cut of the human skin, sebaceous and apocrine glands are anatomically close together (Figure 5.7) [39]. The latter, however, is usually located deeper in the dermis and invariably linked with a hair follicle, mainly in adults in the axilla (axillary organ) and in the pubic, anal, and auditory meatus (ear) canals as well. This organ, characterized by a nearly equal number of eccrine and apocrine glands in the axilla, is a feature that interestingly can only be found in our closest relatives in nature, the chimpanzees and gorillas.

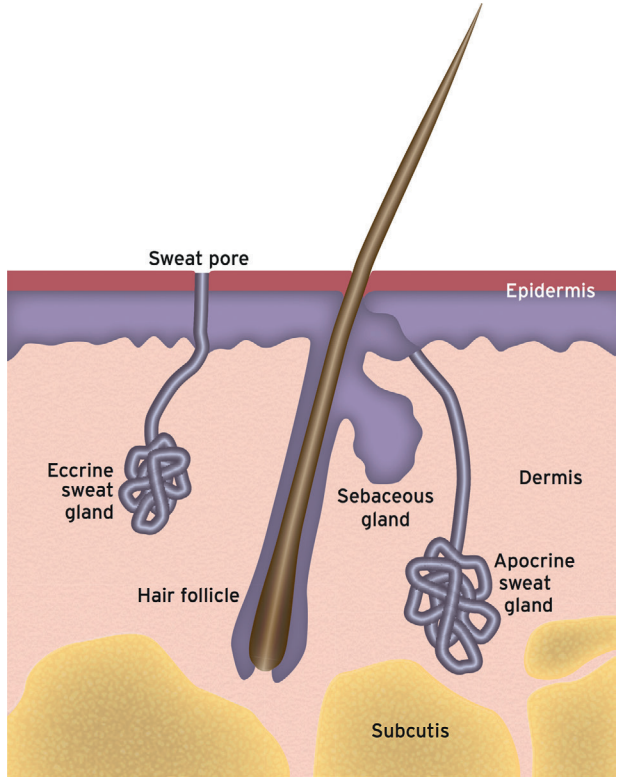

FIGURE 5.7 Schematic cross cut through the human skin.

They are also rudimentary in orangutans and are completely missing in all other mammals. As described by Folk and Semken [39], they have a tubular form and secrete a milky, viscid, gray, or reddish fluid. In the human fetus, apocrine glands can be found everywhere over the body up to the 5-5.5 month, then they disappear except those regions mentioned above. Eccrine glands (sweat glands) (i) have also a tubular form, (ii) are smaller than apocrine glands, and (iii) are located in the outer region of the dermis (Figure 5.7).

The number of sweat glands varies between 2-5 million over the whole body surface area with the highest density at the palms/soles. A smaller number of sweat glands can be found at the head/trunk, and the lowest numbers at the extremities. On the average 150-340/cm^2 can be found in the human skin [39]. However, the absolute number of eccrine glands can vary distinctly between different ethnic groups [35]. In general, populations living in colder environmental conditions have a smaller number of sweat glands than those living permanently in a hot and/or tropical environment. So, for example, the Ainu in the Arctic regions have in total only 1.4 million sweat glands, whereas Caucasians have >3 million [4,40]. Similar observations in different ethnic groups living in the circumpolar regions were made by other authors, as well. So, for example, Schäfer et al. conducted specific research on the activity and number of sweat glands in *Eskimos* and Caucasians [41]. Specifically, they studied the functioning sweat glands in 17 skin sites on the face, body, and limbs of 37 adult male *Eskimos* and 21 Caucasian controls. They found that *Eskimos* showed greater numbers and greater activity of functioning sweat glands on exposed parts of the face such as nose and cheeks, while responding with significantly less sweat gland activity on all body surfaces that are normally heavily clothed in winter. According to Schaefer et al., trunks, arms, hands, legs, and feet showed a progressive reduction of sweat gland response in the order of one-half on the trunk to one-fifth on feet when comparing mean sweat gland counts per square centimeter in *Eskimos* and controls. The comparative reduction of sweat gland response in the *Eskimos* progressed in the same order as the distance of the part from the body core and as the risk of the part to freezing. They concluded from the study that this reduction of sweat gland activity may represent a morphological and/or functional adaptation to environmental conditions including climate and clothing [41]. In addition, during the process of heat acclimatization, the sweat glands are targets of specific metabolic/biochemical changes. Before heat acclimatization, the sweat has an osmolality of about one-third (100 mosm/L) of that of plasma (285-295 mosm/L) [35]. Normal sweat composition consists of water, minerals, lactate, and urea. On average, the mineral (including trace minerals) composition of sweat is as follows: sodium (0.9 g/L), potassium (0.2 g/L), calcium (0.015 g/L), magnesium (0.0013 g/L), zinc (0.4 mg/L), copper (0.3-0.8 mg/L), iron (1 mg/L), chromium (0.1 mg/L), nickel (0.05 mg/L), and lead (0.05 mg/L) [42]. After an acclimatization period of about two weeks, (i) the threshold of sweating will be decreased (earlier starting of sweating), (ii) the regional pattern of sweating will change (Figure 5.8), (iii) the absolute amount of sweat produced by the eccrine sweat

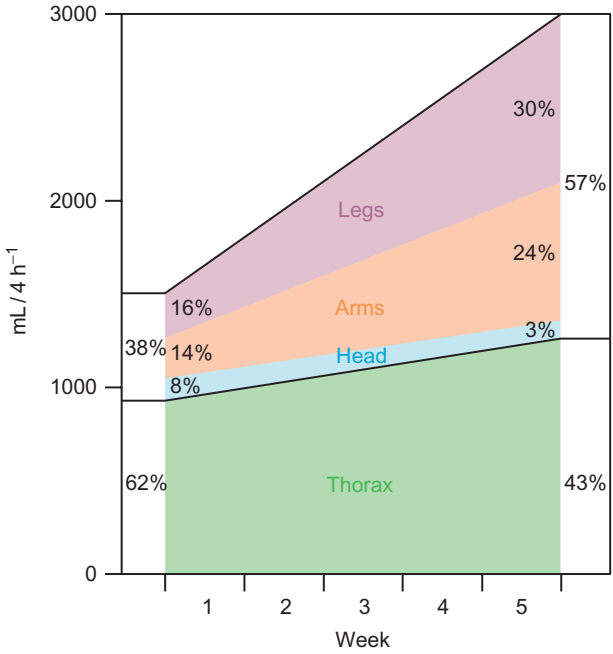

FIGURE 5.8 Changes of regional sweating pattern after a four-week lasting exposure to a tropical climate. Please note that especially legs and arms show a remarkable increase. *Adapted from Ref. [43].*

glands will increase from 1.5 to 4 L/h, and (iv) the sweat composition will be attenuated, showing a lower osmolality [20,44]. In fully heat-acclimatized subjects, the latter can be reduced to as low as 10 mosm/L, thereby (i) saving essential electrolytes for the human body and (ii) avoiding eventually deleterious hyponatraemia during prolonged exercise in the heat [43,45–47].

However, although the anatomy and physiology of human sweat glands have been studied intensively by different research groups in the past [48–60], such research has focused mainly on young males [51]. Indeed, much less information is available regarding females, children, and the elderly [51]. Therefore, recently, Havenith et al. [61] and Smith and Havenith [62,63] studied the regional variation in sweating over the body under resting conditions and exercise. They used a modified absorbent technique to collect sweat at two exercise intensities [55% (I1) and 75% (I2) $\dot{V}O_2max$ in moderately warm conditions (25 °C, 50% relative humidity, 2 m/s air velocity). At I1 and I2, highest sweat rates were observed on the central (upper and mid) and lower back, with values as high as 1197, 1148, and 856 g/(m h), respectively, at I2. Lowest values were observed on the fingers, thumbs, and palms, with values of 144, 254, and 119 g/(m² h), respectively at I2. Sweat mapping of the head demonstrated high sweat rates on the forehead (1710 g/(m² h) at I2) compared with low values on the chin (302 g/(m² h) at I2) and cheeks (279 g/(m² h) at I2). Sweat rate increased significantly in

all regions from the low to high exercise intensity, with exception of the feet and ankles. They observed no significant correlation between regional sweat rates and regional skin temperature, nor did regional sweat rate correspond to known patterns of regional sweat gland density. Aside from a detailed mapping of regional sweat rates over the whole body, their study demonstrated a large intra- and intersegmental variation and the presence of consistent patterns of regional high versus low sweat rate [63]. In Caucasian females [62] regional sweat rates were determined at two exercise intensities (60% (I1) and 75% (I2) $\dot{V}O_2max$) in moderately warm conditions (25 °C, 45% relative humidity, 2 m/s air velocity). A comparison was made between the females and males. The results are summarized in Figure 5.9 [63].

They found that female I1 regional sweat rate was highest at the central upper back, heels, dorsal foot, and between the breasts (223, 161, 139, and 139 g/(m^2 h), respectively). Lowest values were over the breasts and the middle and lower outer back (<16 g/(m^2 h)). At I2, the central upper back, bra triangle, and lower back showed the highest regional sweat rate (723, 470, and 333 g/(m^2 h), respectively). Regions of the breasts and palms had the lowest regional sweat rate at I2 (<82 g/(m^2 h)). Significantly greater gross sweat loss and thus regional sweat rates were observed in males versus females at both exercise intensities. For the same metabolic heat production (male I1 vs. female I2), absolute and normalized regional sweat rates showed a significant region-sex interaction,

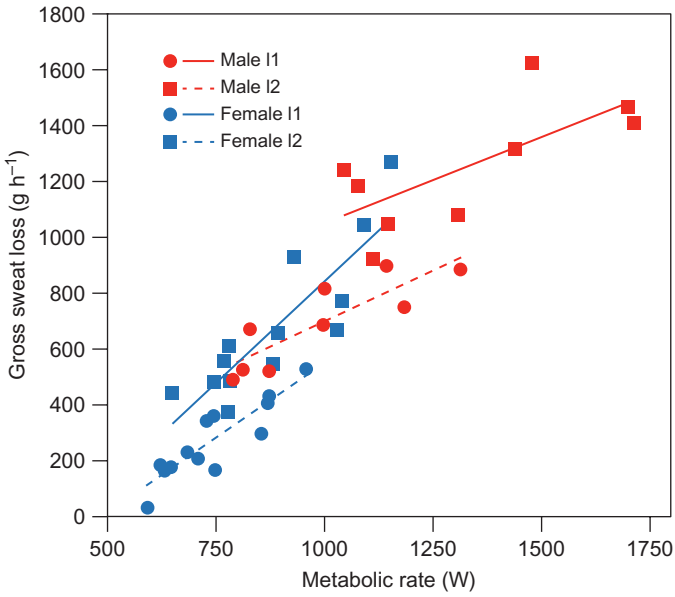

FIGURE 5.9 Absolute mean gross sweat rate (g/h) and absolute mean metabolic rate (*W*) for trained females and males at exercise intensity 1 (I1) and intensity 2 (I2). *Male data have been adapted from Smith and Havenith [63].*

with a greater distribution toward the arms and hands in females versus males. They concluded from their studies that despite some differences in distribution, both sexes showed highest regional sweat rates on the central upper back and the lowest toward the extremities. Furthermore, regional variation in sweat rates neither correlates with regional skin temperature nor does it correspond with regional sweat gland densities reported in the literature [62,63].

5.6 THERMOREGULATION

The task of the thermoregulatory system is to keep the body temperature constant within narrow limits, so that a balance exists between heat production and heat losses. The control variable is the core body temperature, an integrative value resulting from the local temperatures of many parts of the body. The autonomic mechanisms of temperature regulation (skin blood flow, sweating) permit a certain degree of acclimatization under extreme environmental conditions, although to a limited extent. Most important are adequate cognitive behavioral adaptations. Otherwise, under extreme environmental conditions, a failure of the regulatory mechanisms can rapidly occur, leading to hypo- and/or hyperthermia. This pertains especially to children and elderly people. For the registration of external environmental and internal core body temperatures, the skin is supplied with cold and heat receptors, unequally distributed over the human body (Table 5.1). Via afferent sensory nerve fibers, these external (body shell) and internal (body core) thermoreceptors are connected with the spinal cord and the hypothalamus, which is regarded as the regulatory center of temperature regulation. Most cold receptors are located approximately 0.2 mm below the skin and are, with exception of the scrotum, more numerous than the heat receptors [6]. Following fast increases of the skin temperature, the heat receptors in the first instance react with an excessive discharge rate of approximately 10-50 impulses/s [6,35]. Afterwards their impulse rate decreases quickly (to approximately 20 impulses/s). Cold receptors have a considerably lower spontaneous discharge rate (2-10 impulses/s) than heat receptors. During the acute excitation (excitatory) phase of either heat or cold receptors, the other receptors in the skin are inhibited. To date, not much is known about the mechanisms of signal transduction of the temperature signal. There are indications that changes of the sodium pump and the passive Na^+/K^+ conductivity might play an important part [6,35].

Anatomically, the preoptic area of the anterior hypothalamus (POAH) seems to be where the actual temperatures of the body shell and the body core are compared to a so-called setpoint value. In technical systems (e.g., air-conditioning systems) this setpoint value is set by means of a temperature reference signal placed within the control circuit. In the hypothalamus, special neurons are supposed to exist producing this signal independently of the temperature [6,8]. Such neurons in the hypothalamus, however, until recently could not be verified in a sufficient number. When actual temperature and setpoint value deviate from each other, various control elements in the control

TABLE 5.1 Distribution of Warm and Cold Points in Different Body Areas

	Distribution of Warm and Cold Points	
Body Region	Cold Points per cm²	Warm Points per cm²
Forehead	5.5-8	2
Nose	8-13	1
Mouth	16-19	–
Remaining face	8.5-9	1.7
Chest	9-10.2	0.3
Forearm	6-7.5	0.3-0.4
Palm of hand	1-5	0.4
Finger, outer face	7-9	1.7
Finger, inner face	2-4	1.6
Thigh	4.5-5.2	0.4

Data are Taken from Kunsch and Kunsch, 2007 [95]

circuit (motor system, brown adipose tissue, vasomotor activity, sweat secretion, pilomotor activity) are changed by the autonomic nervous system via efferent vegetative nerve fibers in the circuit of positive and/or negative feedback [6,35]. A decrease of the core body temperature below the setpoint value set by the hypothalamus leads to (i) a vasoconstriction of the skin and shell vessels (negative feedback), whereby the heat release via the body shell is reduced; (ii) a piloerection of the hair (goose bumps), which enlarges the insulating boundary layer above the skin and thus decreases the heat loss; and (iii) an increased heat production by shivering. When the actual value, on the other hand, lies above the setpoint value, all those mechanisms that might evoke a further increase of the body temperature (motor system) are extenuated (negative feedback), and the mechanisms of heat loss are strengthened (vasodilatation in the body shell, increase of sweat secretion). These different defense mechanisms for the maintenance of the core body temperature are reflexes and cannot be influenced entirely arbitrarily (autonomic control). The conscious sensations of thermal comfort or discomfort are generated in the sensory cortex, which receives the excitations of the internal and external cold and heat receptors via the tractus spinothalamicus and the unspecific medial thalamic regions. With distinct thermal discomfort, not only a stimulation of the autonomic countermeasures is initiated, but also, mediated via the cortex, changes in behavior, which leads to selection of warmer clothing or taking shelter in a heated room in a cold environment [6,8,35].

5.6.1 Cardiovascular Regulation

Particularly under extreme environmental conditions such as high ambient temperature and/or high relative air humidity, the simultaneous demands of metabolically active musculature and the thermoregulatory system (increased blood circulation of the skin for heat dissipation) put high stress on the cardiovascular system. The skin blood circulation can then increase to several liters per minute and constitute a considerable part of the cardiac output per minute. This is illustrated in Figures 5.10 and 5.11 [20,64].

With a simultaneous increase of the body core and body shell temperature, the compliance in the venous system of the skin is augmented and vasomotor tone decreases. This entails a decrease of the central blood volume, which is

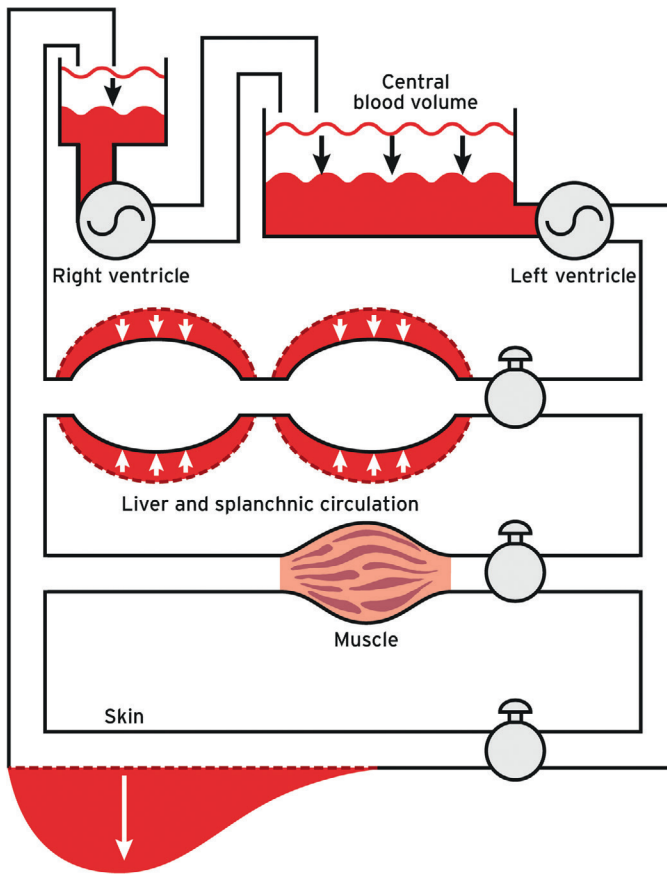

FIGURE 5.10 A schematic view of the different parts of the cardiovascular system. Please note the skin as a highly variable volume compartment of the cardiovascular system. If volume is markedly increased during, for example, heat stress, concomitantly the blood volume in the right ventricle will be decreased. *Adapted from Ref. [64].*

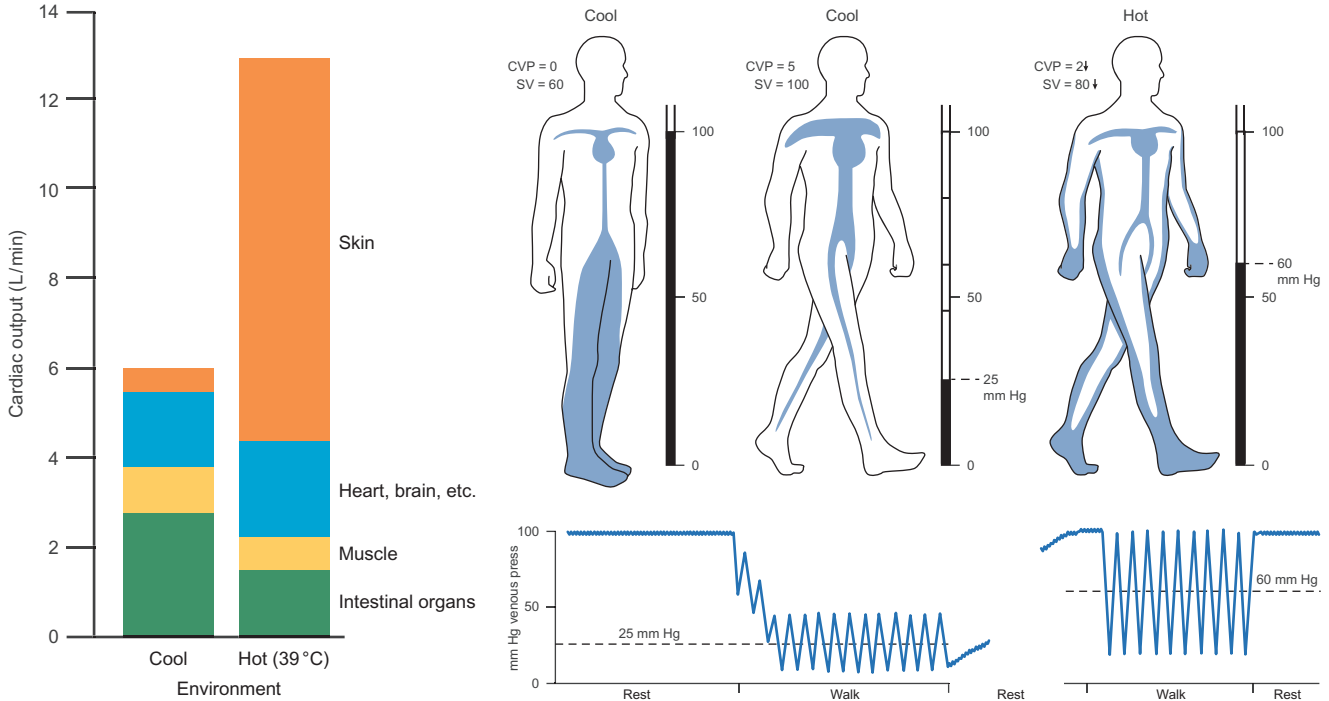

FIGURE 5.11 Circulatory changes in blood flow under different environmental conditions (left panel) and changes in central blood volume and central venous pressure in a cold and a hot environment (right panel). *Adapted from Ref. [64].*

essential for the physical capacity. In a human at rest in an upright position and at an indifferent ambient air temperature, approximately 70% of the blood volume is below the heart and 85% thereof in the low pressure system [65]. Physical work under the extreme environmental conditions thus leads to a disturbance of the volume distribution (heat collapse) with a descent of arterial pressure, filling pressure, and stroke volume [64,65].

An orthostatic intolerance thus has to be considered as an emergency reaction leading to unconsciousness. Placement into a horizontal position leads to a new redistribution of the blood volume from the periphery to the center. In the case of a heat collapse, this redistribution from the periphery to the center can be supported by an elevation of the extremities. Furthermore, as can be seen in Figures 5.10 and 5.11, the additional blood volume to maintain a higher perfusion of the skin is taken from the liver and splanchnic region. Because all food (i) is absorbed by the intestines and (ii) processed in the liver, any reduction in blood supply to these tissues and organs is as well accompanied by a reduction in the efficiency of local immune system. That is why (i) parasites, apart from the fact that the liver is a very nutrient-rich organ, prefer to go and stay there; and (ii) the immune deficiency in the intestines can very rapidly lead to a systemic infection (endotoxemia). The latter effect is illustrated in Figure 5.12, which shows that a decline in consumed plasma antilipopolysaccharide (LPS) concentrations occurred already at rectal temperatures as low as 39-40 °C [66].

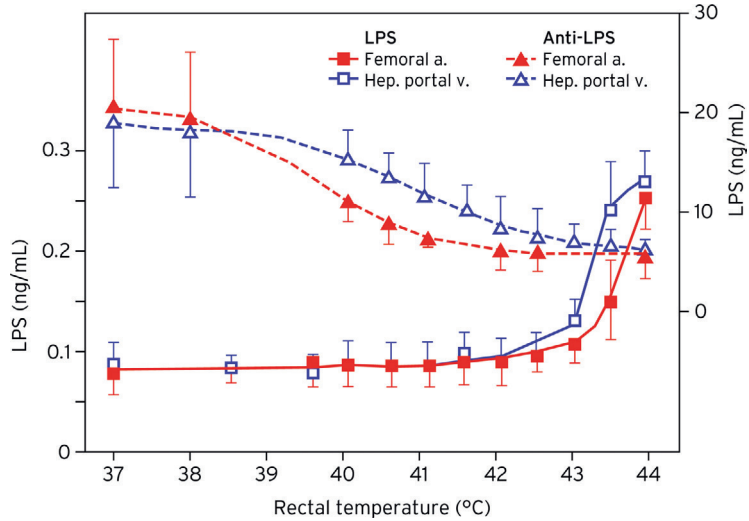

FIGURE 5.12 Endotoxemia following intestinal ischemia in an anesthetized nonhuman primate. Please note that (i) at a rectal temperature of 42-43 °C, plasma lipopolysaccharide (LPS) concentrations increase first in the hepatic portal vein, (ii) about 10-15 min later also in the systemic circulation; however, (iii) the decline in consumed anti-LPS antibodies already occurred at rectal temperatures as low as 39-40 °C. *Adapted from Ref. [66].*

5.6.2 Water/Salt Homeostasis

Evaporation, as outlined before, is a significant mechanism of heat loss for humans and can amount to up to several liters per hour. The organism also loses electrolytes with sweat in addition to body fluid. This can exert a substantial influence on the water/salt balance. If the loss in fluid and electrolytes is not compensated for, there is a risk of dehydration and hyponatremia/hypocalcemia. In the early stages, plasma volume is used for the production of sweat in which strong evaporation reduces the circulating blood volume with the adverse effects on the cardiovascular system (decrease of filling pressure and stroke volume). A hypotonic sweat facilitates the necessary fluid shifts for the maintenance of the circulating blood volume, because the loss entails an increase of the intravasal colloid-osmotic pressure. This is the driving force for the influx of water from the interstitial space and, with more extensive evaporation, also from the intracellular compartment [64]. The body temperature plays a decisive role in the function of fine motor skills as well as gross motor skills. If the body temperature drops, fine motor skills are restricted. With the onset of shivering, gross motor skills are likewise seriously disturbed. Due to this fact, sporting events are held preferably in the afternoon and evening hours because during this period the circadian core body temperatures are highest and the environmental conditions rather moderate—important prerequisites for peak motor performance.

5.7 SPECIAL TEMPERATURE REGULATION

5.7.1 Heat Production During Physical Work

Heat production during physical work maximum oxygen uptake ($\dot{V}O_2max$) depends on the maximum heart rate (HF_{max}), the maximum stroke volume (SV_{max}) and the maximum arteriovenous oxygen difference ($AVo_2diffmax$):

$$\dot{V}O_2\,max = HF_{max} \times SV_{max} \times AV_{O_2diffmax}$$

The environmental conditions (PO_2, air temperature and radiation temperatures, air humidity, air velocity) can have a substantial impact on human performance. If in the course of a physical endurance exercise, for example, blood volume decreases due to strong sweating and insufficient fluid intake, the stroke volume decreases, thus $\dot{V}O_2max$. If blood circulation in the body shell has to be increased for thermoregulatory reasons, this blood volume is lost for the metabolically active musculature, for example, in the course of a physical endurance exercise, the blood volume decreases due to strong sweating and insufficient fluid intake, stroke volume, thus $\dot{V}O_2max$, likewise declines. If for thermoregulatory reasons the blood circulation in the body shell has to be increased, this blood volume is lost for the metabolically active musculature, and the maximum $AVo_2diffmax$ is diminished. Under resting conditions, the oxygen uptake

of the skeletal muscle lies around 1.5 mL/(min kg min) and can be increased to tenfold during physical activity. The entire heat production of the adult human under resting conditions corresponds to a performance of approximately 80 W and can increase to up to 1000 W [8]. With sufficient energy reserves and/or continuous fluid supply, this physical exercise can be maintained over several hours, for example, in a marathon or triathlon. If these augmented mechanisms of heat loss, such as evaporation, had not evolved in humans, internal heat production would limit the endurance capacity to approximately 20 min because every 5-8 min the core body temperature would increase by about 1 °C, resulting in a rapidly occurring lethal hyperthermia [20].

5.7.2 Heat Loss

The total heat loss from the body core to the skin surface is comprised of two components: (i) fixed value transmitting the heat by conduction (passively) via the inactive musculature and the subcutaneous skin layers, and (ii) strongly variable heat transport by means of the convective heat transport of blood/circulation. Increased heat dissipation from the body core to the body shell is mainly ensured by means of an augmented blood flow from the muscles to the skin and under physical and/or warm environmental conditions by an increased evaporation of sweat [64]. The skin blood circulation can vary in different parts of the body. Thus, for example, it can be increased in the trunk by a factor of 7, at the hand by a factor of 30, and at the fingers by a factor of 600. Although the hands are merely a small part of the total body surface, they play a decisive role in thermoregulatory system of the human body because they serve as "thermal windows." Furthermore, per gram of evaporated sweat, an ultrafiltrate of the plasma, the organism loses approximately 2.5 kJ, the sweat glands being able to produce about 2-4 L/h, thus an amount of 30 g/min of sweat. The maximum sweat production, as well as the composition of the sweat, is variable. Besides a generally lower normal temperature, trained organisms adapted to heat have a lower sweating threshold. The trained athlete begins to sweat earlier so that his or her body temperature is lower under comparable conditions than that of less well-trained persons. Under the same environmental conditions and load, the athlete is thus able to maintain a performance for a longer period of time. Furthermore, the cooling of the body shell by sweating is important to maintain a lower skin temperature and thus a heat gradient from the body core to the body shell. If the body shell is warmer than the body core, the body core is supplied with heat from the body shell via the vessels (sauna effect). The heat flux is converted, and the core body temperature increased. The amount of heat that can be emitted by respiration to the environment plays only a minor role in the total balance of heat fluxes to prevent hyperthermia. However, considerable amounts of heat can thus be withdrawn from the body at very cold air temperature and heavy exercise, i.e. high respiratory breathing rates.

5.7.3 Age-Dependent Temperature Regulation

With an environmental temperature of 32-34 °C (relative air humidity approximately 60%), the thermal neutral zone of the newborn is distinctly higher than that of adults. The reason for this is that newborns have a very unfavorable surface-volume relation (threefold larger) compared to adults and only a very thin subcutaneous fat tissue. Newborns, however, can activate the shiver-free heat production in the brown adipose tissue via the sympathetic nervous system. Six to eight weeks after birth, distinct circadian variations of the core temperature can already be observed with lowest values between 2 and 4 o'clock in the morning. Sweat production increases distinctly with the beginning of puberty being about 350 g/(m^2 h) prior to puberty (ages between 7 and 11 years old) and between 500 g/(m^2 h) after puberty (ages 13-15 years old) [51]. Falk [67], Falk and Dotan [68], and Sinclair et al. [69] have addressed this topic in reviews and different studies. They conclude that prepubertal children's ability to thermoregulate when exposed to hot and humid environments is deficient compared to adults. However, definitely—although difficult to perform for ethical reasons—more research is urgently needed in this field. Not only is the younger population endangered in hot environments but also elderly people (Larose J 2013[70]; Popkin BM [71]) (Figure 5.13). Much like newborns, the elderly have a larger requirement for warmth. This might be due to their overall decreasing metabolic rate, the decrease in water content of the skin, a thin subcutaneous fat tissue, and/or a reduced vasomotor activity. Elderly people are endangered especially

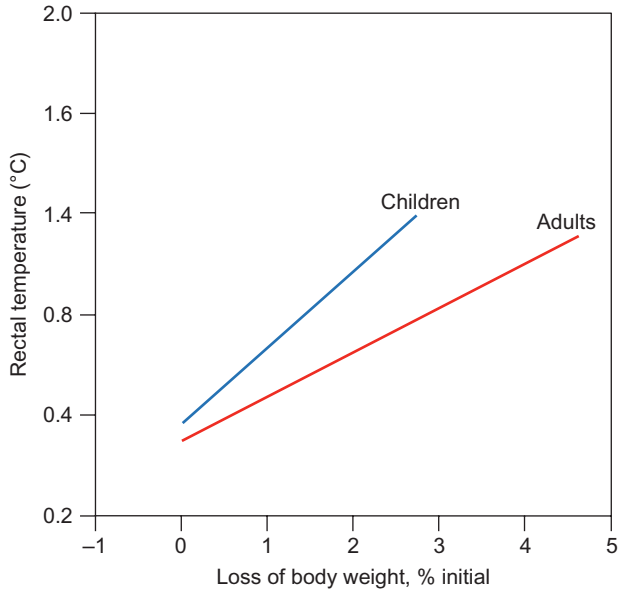

FIGURE 5.13 Comparison of core temperature changes in children and young healthy adults. *Adapted from Ref. [51].*

in the first 1-3 days of heat wave during the summer period (Leon LR 2012 [96]; Larose J [70]; Popkin BM [71]).

5.8 ADAPTATION, ACCLIMATIZATION, ACCLIMATION, AND HABITUATION

5.8.1 Definitions

An organism must be adequately adapted to all kind of environmental stressors in order to survive. Four major distinct terms are used to describe this process in biology and have to be decisively distinguished: adaptation, acclimatization, acclimation, and habituation [9].

An adaptation is a trait with a current functional role in the life history of an organism. Adaptation is an evolutionary process by which an organism becomes adjusted to its environment and becomes fitted through natural selection for some special activity. Adaptations contribute to the overall fitness and survival of the organism. Such changes can encompass genotypic and phenotype adaptations as well as behavioral responses to environmental stressors such as heat, cold, and hypoxia, to mention a few conditions.

The process of acclimatization takes a short period of time (days to weeks). Acclimatization, compared to adaptation, occurs within the organism's lifetime. The individual organism adjusts gradually during this time span to the natural environment, allowing it to maintain performance across a range of environmental conditions.

Acclimation is a term used to describe adaptive physiological responses to experimentally induced changes, in particular, climatic factors such as heat or hypoxia by means of an artificial exposure, for example, in a hypobaric chamber [35]. In general, organisms can adjust their morphological, behavioral, physical, and/or biochemical traits in response to changes in environmental challenges [9]. Among those responses might be changes in the biochemistry of cell membranes, making them more rigid in cold temperatures and more porous in warm temperatures by increasing the number of membrane proteins. Specific proteins, those so-called chaperones, are, for example, the well-known heat shock proteins (HSP 70, HSP 72, HSP 90). The molecular substances (i) bind to normal proteins to prevent them from deforming and (ii) can even unfold damaged proteins back to their original shapes. The number of heat shock proteins increases within about an hour or so after heat stress and expand the range of survival of the cellular structures by approximately 1.5-2.0 °C. Chaperones are therefore essential in maintaining cell functions under periods of extreme stress, especially in organisms living in the hot deserts (Kamler, 2004) [99]. It has been shown that organisms that are acclimated to high or low temperatures display relatively high resting levels of heat shock proteins so that when they are exposed to even more extreme temperatures, the proteins are readily available (Leon, 2012) [96]. Other examples of biochemical adaptations are neurohormonal factors such as dopamine or 5-hydroxytryptamine.

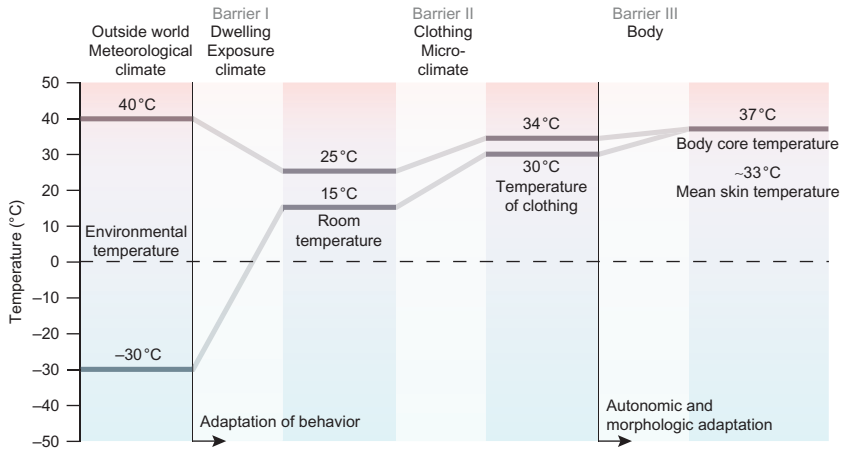

FIGURE 5.14 The range of the autonomic adaptations in humans in comparison to behavioral adaptations. *Adapted from Ref. [10].*

However, under extreme environmental conditions behavioral adaptations are the main avenue by which humans are able to survive (Figure 5.14). For example, the autonomic responses to overcome extreme thermal strains, heat and cold, are very limited, and without other counter measures, will lead rapidly to a fatal hypo- or hyperthermic state of the organism.

As humans evolved in a relatively dry, moderate altitude environment with a limited food and water availability, they were forced to run about 20 km daily (for details, see Chapters 1 and 3) in order to acquire resources. Thus, it is not surprising that humans possess hardly any natural biochemical, physiological, and/or anatomical-morphological protective mechanisms against the cold (hypothermia), such as thick fur or thick subcutaneous adipose tissue. Humans however, are equipped against heat strain (hyperthermia). The major mechanisms to avoid the latter include (i) a decrease in the sweating threshold, (ii) an increased amount of sweat (Figure 5.15), (iii) a decrease of the electrolyte content of the sweat (Figure 5.16), (iv) a decrease in heart rate, and finally, (v) an increase in total blood volume. The Zeitgang (time course) of these major adaptations is shown in Figure 5.16.

In humans, the maximum sweat production in relation to the body surface area is markedly higher than in any other organisms. The lower electrolyte content of the sweat has several effects: (i) electrolytes are preserved in the organisms, that is, a deficiency in supply of the organism is counteracted; and (ii) the sweat evaporates more easily from the skin. The augmented amount of plasma proteins in the long run increases the plasma volume by 10-20%. Hematocrit decreases correspondingly, which lowers blood viscosity, so that the cardiovascular system is thus in a better initial position to operate much more efficiently and effectively. Therefore, comparable loads during physical activity can be accomplished with lower heart rates. All mentioned factors contribute to the

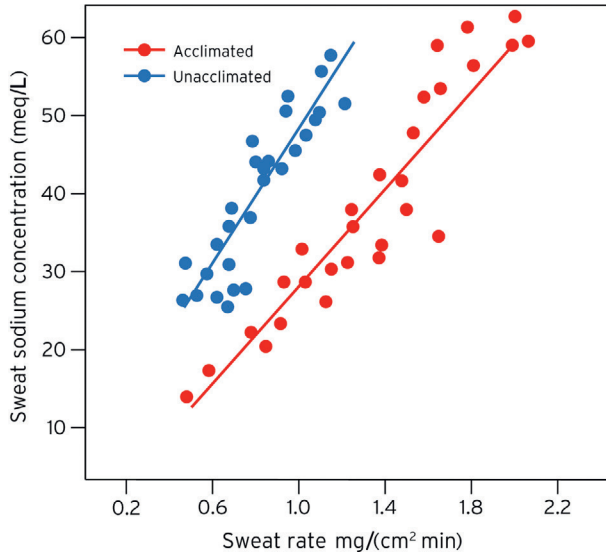

FIGURE 5.15 Relationship between sweat sodium concentration and back sweating rate before and after heat acclimation. *Adapted from Ref. [45].*

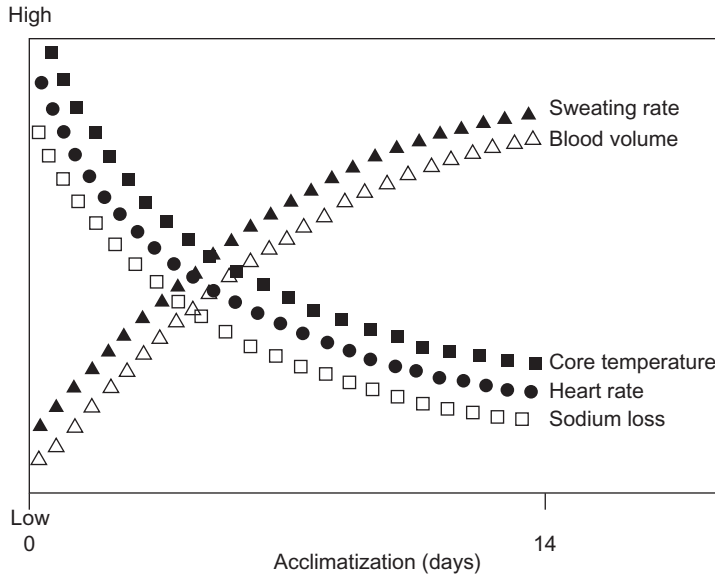

FIGURE 5.16 Thermal acclimatizations in humans to a hot environment and their time course.

fact that increases in body core temperature under physical exercise is slower than in nonadapted persons. The long-term heat acclimatization processes thus bear a strong resemblance to those adaptations observed in endurance athletes.

5.8.2 Special Adaptations

5.8.2.1 Adaptation of Behavior

The capacity of autonomic adaptations is relatively limited in humans as mentioned before. That is why these autonomic adaptations have to be supplemented by corresponding behavioral adaptations. During a stay in the desert, physical activities should be carried out either in the early morning hours, in the late evening hours, or at night. This avoids high load peaks during the day in which the long-wave radiation maxima of the sun is present. Fluid intake to compensate and—with very strong heavy sweat losses and restricted availability of a balanced diet—also an additional salt uptake have to be made consciously and repeatedly. Thirst and appetite for salt are inadequate with strong losses. Nightly cramps in the calves indicate corresponding deficiency symptoms in the electrolyte balance. In Figure 5.17 the different fluid losses associated with different kinds of activities in a hot desert climate are illustrated [9,35].

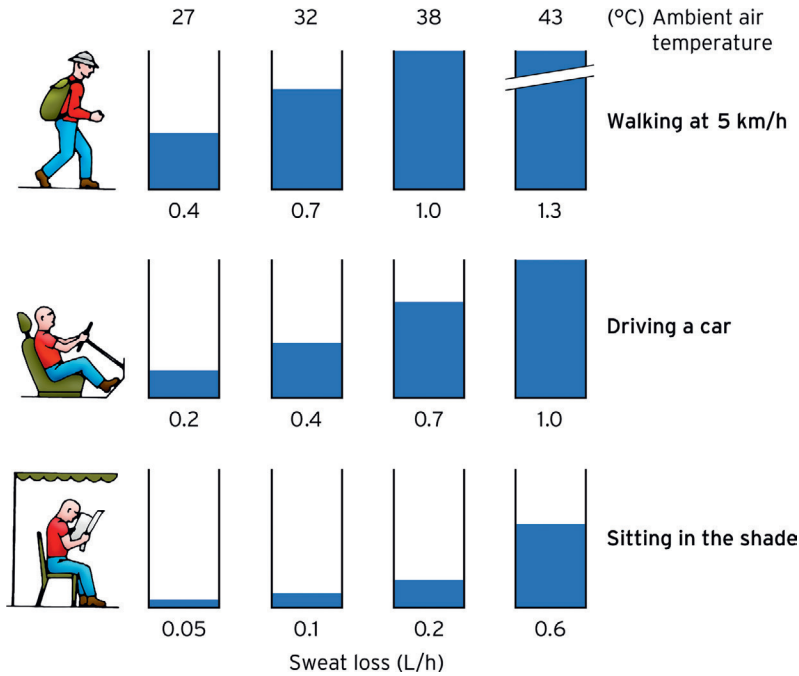

FIGURE 5.17 Fluid losses under different environmental and physical conditions. *Adapted from Ref. [44].*

The clothing should cover as much of the skin surface as possible and be permeable to water vapor and loose-fitting in order to permit air circulation along the body axis (natural convection, pleasant microclimate). Shkolnik et al. [73] investigated whether black robes help the Bedouins to minimize solar heat loads in a hot desert and reported that the amount of heat gained by a Bedouin exposed to the hot desert is the same whether wearing a black or a white robe. The additional heat absorbed by the black robe was lost before it reached the skin. The higher temperature in the loose black clothing as compared to the white clothing increased the natural convection between the different layers of Bedouins coats. The color of the clothing is therefore of less importance than its material and style as nicely shown in Figure 5.18. The important is that in dark, loose-fitting multilayer clothing, the higher absorption of energy in the outer layers increases the convection within the clothing. This effect is so strong that in the end, the surface temperatures on the skin, which are important for a pleasant feeling, that is, the microclimate, is the same as dressed in white clothing, which physically absorbs less energy from radiation.

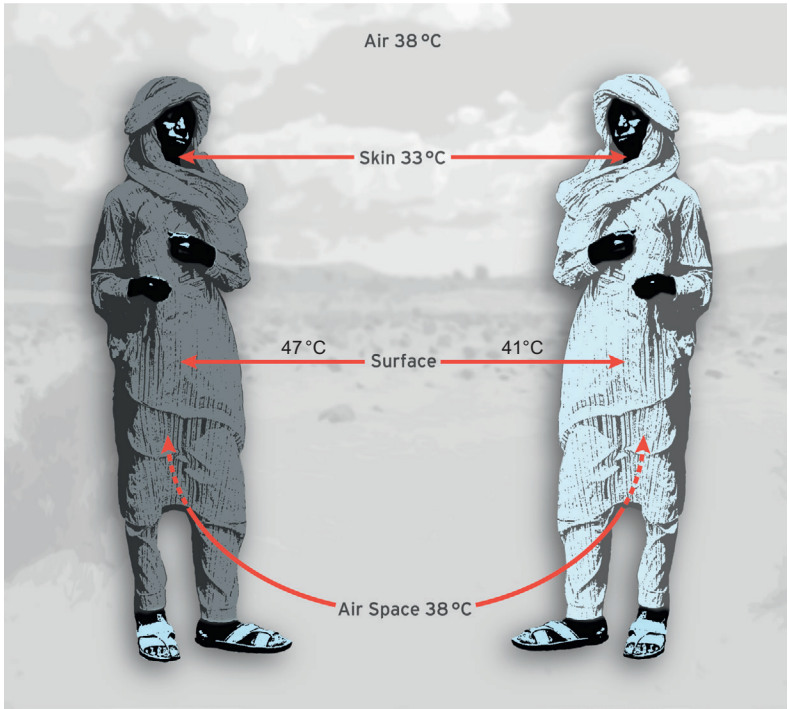

FIGURE 5.18 At an ambient temperature of 38 °C the surface temperature of a black Bedouin coat (left person) is higher compared to a white one. The skin temperature is in both cases 33 °C at the face as indicated by the arrows. *Adapted from Ref. [73].*

A special form of heat adaptation is observed in large animals (giraffes, camels) in hot climate zones, the so-called adaptive heterothermia. These animals increase their sweating threshold and decrease their shivering threshold, thus widening the range in which changes of the core body temperature are tolerated. This procedure reduces the quantity of sweat and subdues an early heat production by shivering during decreasing of the core body temperature. During the day, the body stores large amounts of heat and releases it at night through convection and radiation to the cool environment. In addition, a wide range of change in their core temperatures could be observed during a 24-h period, and this could be attributed to a well and dehydrated state of the organism (Figure 5.19) [74,75].

In addition, large desert animals, such as the camel and donkey, have a very low evaporative water loss in terms of percentage of their body weight in comparison to humans and other animals as summarized in Figure 5.20.

Under arid environmental conditions with low availability of water these are very reasonable strategies to survive. Furthermore, in indigenous humans living in the tropics, an increase in the sweating threshold has been detected. This can also be interpreted in the sense of economization. In general, humans tolerate a dry-hot climate, that is, a desert climate, better than the tropical climate (warm/hot ambient air temperature, high moisture, still air) because physically a high vapor pressure has to be reached to fully evaporate the sweat thereby cooling the body surface. Furthermore, due to the high ambient humidity, not all sweat is fully evaporated, but instead sweat drops fall to the ground. This sweat is lost for cooling because under these circumstances the cooling effect of sweating cannot be achieved and the permanent high skin perfusion, as mentioned above, is a heavy burden for the circulatory system. Older subjects with cardiovascular diseases are especially prone to suffer under these climate conditions.

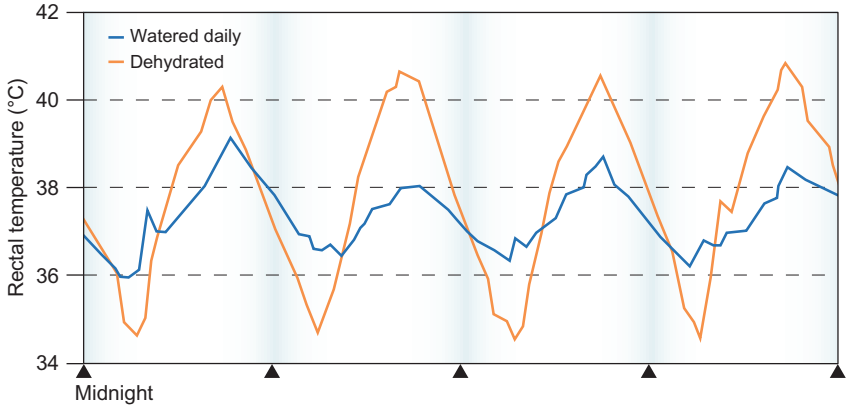

FIGURE 5.19 Circadian core body temperature changes in a hydrated and dehydrated camel. *Adapted from Ref. [74].*

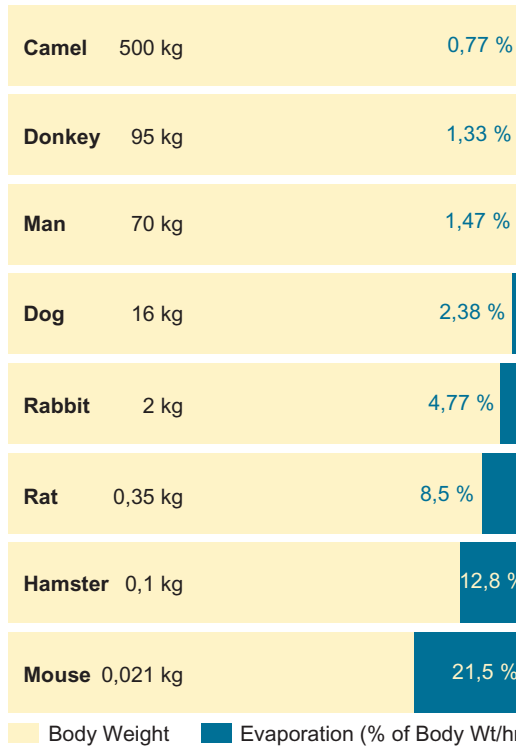

FIGURE 5.20 Differences of evaporative fluid loss in humans and some animals living in different environmental settings. *Adapted from Ref. [97].*

5.8.2.2 Torpor, Hibernation, and Estivation

We will now focus on adaptations in animals living in extreme environments in order to show the range of extraordinary adaptations enabling them to tolerate and survive under stressful environmental conditions. For example, some mammals and birds fall into dormancy every winter or in very hot summer months. According to its length and the environmental conditions, a distinction is drawn among torpor, hibernation, and estivation. Daily phases of rigor with reduced body temperature and metabolic activity are designated as torpor. This phenomenon can be observed, for example, in bats and colibris. Mammals (hamsters, kangaroo, rats) decrease their metabolic rate and thus body temperature not only for hours, but rather for weeks and months. This state is called hibernation. For that kind of adaptation, it is necessary that the animals built up large energy reserves (body fat) during the summer months. Furthermore, these animals hibernate mostly in deep burrows that provde considerably enlarged thermal insulation to the harsh environment prevailing outside this self-made cave. Nevertheless, the body temperatures of the

hibernators fall below the ambient temperature in the cave, which might be barely above the freezing point of water. If the ambient temperature tends to decrease markedly below 0 °C, there is a definite danger for the animal to die by freezing. But before that occurs, the hibernating animal starts to produce heat by nonshivering thermogenesis, for example (see below). The organism then reaches its normal core body temperature (approximately 36 °C) for a short time and afterwards reduces its metabolism and other physiological parameters of the cardiovascular system once again. The regulation of body temperature is thus entirely intact in these organisms, only the threshold of heat production seems to be decreased drastically. It is therefore assumed that this is a defined reduction of the mean setpoint value in the hypothalamus. The process of waking up can take place several times in the winter months. Even though these waking phases are metabolically expensive for the organism, hibernation on the whole is a very effective measure to overcome meager and hostile seasons by means of low metabolic efforts. This saves approximately 80% of the energy required in comparison to normothermia. Small organisms with a large surface-body volume relation particularly use hibernation or torpor as survival strategies. Most hibernators have a body mass of about 85 g. The smallest hibernators weigh merely 5 g, the largest more than 100 kg, if bears are included. Besides torpor and hibernation, some organisms fall into a summer dormancy (estivation). Snails, for example, are thus able to survive longer dry periods [9,35].

5.9 HYPERTHERMIA AND FEVER

5.9.1 Hyperthermia

5.9.1.1 Pathophysiology

Hyperthermia is characterized by a disproportion between heat loss and internal heat production or external heat supply without a shift of the setpoint value as observed with fever [6]. With increased physical exercise or external heat supply (sauna), body temperatures above 41 °C can be observed. After the end of the physical exertion (load) or after the end of the heat exposure, the body temperature returns to its initial value. Recent investigations have shown that especially heat exposure of longer duration and high temperature displays the highest mortality rate. In the past 100 years, the frequency of these extreme climatic conditions has increased significantly in regions with normally moderate climate due to global warming. During the day, these heat waves lead to heating of the densely built inner cities, so that the temperatures at night do not fall below 28-30 °C. The population living in these areas is exposed to a permanent 24-h heat stress, particularly in the attic floors. Children (<1 year), elderly people, diabetics, and persons suffering from cardiovascular diseases are especially affected. For those who are also immobile (bedridden), the convective heat loss is decreased. The circulation is forced to maintain the heat transport from the core to the periphery via a steadily

increased cardiac output per minute in order to guarantee heat transmission from the skin to the environment via an increased skin perfusion. As a result of high environmental temperatures, heat cramps, heat collapse, heat exhaustion, and heat stroke may result [72,76].

5.9.1.2 Heat Cramps

With heavy physical work, especially with environmental temperatures above 27 °C and high relative humidity, the fluid loss by evaporation can amount to 5-10 L/day. Electrolytes are also lost in sweating, particularly Na^+, Cl^-, Mg^{2+}, and Ca^{2+}. Drinking low-electrolyte water further augments the decrease of extracellular ion concentrations. The loss of Na^+ and Mg^{2+} may result in heat cramps in the calf musculature. Occasionally, the abdominal muscles are affected by cramps and thus an abdominal emergency situation seems to exist. The loss of Cl^- ions promotes a hypoacidity of the stomach. Especially in a tropical climate, this achylia promotes the incorporation of pathogens into the gastrointestinal tract [72,76]. Daily fluid intake should be sufficient to guarantee a daily urine output of 800-1000 mL/day.

5.9.1.3 Heat Collapse

The most frequently observed heat illness is heat collapse. This is an orthostatic circulatory disorder occurring in particular during the first days of a stay in hot climate. Heat collapse is caused by a failure of the cardiovascular reaction to high environmental temperature, thus creating a disturbance of the volume distribution of circulation. As an expression of the disproportion between heat production, heat absorption, and heat loss, first of all, the skin blood circulation is increased by peripheral vasodilatation. Even at rest, this requires an increase of the cardiac output per minute. Due to peripheral vasodilatation, the intrathoracic volume is diminished, which can lead to a heat collapse following a phase of orthostatic instability. In contrast to the heat stroke (see below), sweat secretion is intact so that the skin is moist. The body temperature is lower (38.5-41 °C) in comparison to a body temperature of above 41 °C in the case of heat stroke. As possible indications of a circulatory collapse, one has to consider blood pressure decrease, bradycardia (weakness, dizziness, tiredness), headache, lack of appetite, nausea, vomiting, and an urge to defecate. Further symptoms, such as piloerection (goose bumps), observed on the chest and at the upper arms, hyperventilation, muscle cramps, and sometimes also neuronal signs such as ataxia and incoherent speech. Laboratory tests reveal, among others, hemoconcentration, hypernatremia, hypophosphatemia, and hypoglycemia [72,76].

5.9.1.4 Heat Stroke

A longer lasting overheating above 40 °C leads to a heat stroke (disorientation, cramps, delirium). Heat stroke is a life-threatening disturbance of temperature regulation occurring particularly often during the first days of a heat wave in elderly people suffering from chronic diseases such as arteriosclerosis, cardiac

insufficiency, or diabetes mellitus. Furthermore, the occurrence of heat strokes is well known after application of anticholinergic drugs for the inhibition of sweat production, under diuretic therapy, as well as in persons with skin diseases who have a hindered heat loss (e.g., ectodermal dysplasia, congenital missing of sweat glands, severe sclerodermia). However, young people can also suffer heat strokes after extreme physical exertion and an uncovered head in strong solar radiation. The temperature regulation in the hypothalamus is disturbed so that, in spite of high thermal stress, a vasoconstriction of the peripheral skin vessels occurs and the sweat production is suspended. Patients suffering from heat stroke thus have dry, hot skin [72,76]. For every 1 °C temperature increase, the basal metabolic rate increases by 7%. At a temperature of 41 °C, this means a metabolic increase of almost 40%. Frequently, blood volume is diminished and hematocrit is increased. In the pulmonary vascular bed, a resistance increase takes place. The warning symptoms include the following appearing at short notice: intense headaches, dizziness, feeling of weakness, abdominal pain, confusion, or hyperpnea. Blood pressure is extraordinarily low. The muscles are flaccid, and the tendon reflexes can be diminished. Pulse rate is increased and respiration is fast and weak. Depending on the severity, lethargy, stupor, and coma can exist. Heat stroke can also be fatal. One important criterion for the existence of a heat stroke is the core body temperature above 40.5 °C. Even cases with temperatures of up to 44.4 °C have been reported. The skin is red, dry, and hot. Mortality with heat stroke lies at least at 10%. The patients die within a few hours after being found or in the following days and weeks from the consequences of various complications, for example, renal failure, cardiac infarction, or bronchopneumonia. If no sufficient heat dispersion is taken care of during hyperthermia, temperature will continue to increase. Because the heat-regulating mechanisms fail and perspiration is no longer possible, external support for heat dispersion has to be applied (ice-water immersion) [72].

5.9.1.5 Preventions

Prophylactic fluid supply before exposure, light clothing, frequent cool baths, cool environment, and reduced physical activity (in particular old and very young people) can help to avoid heat diseases, especially heat stroke. In order to avoid heat cramps and heat collapse, physical exertion at high ambient temperatures (>26 °C) and high humidity as well as low air movements should be restricted or avoided. Under such environmental conditions, long-distance adult runners should drink, for example, approximately 250 mL of slightly salted fluids every 3-4 km also during the competition. A small amount of electrolytes (salt) and carbohydrates should be added to the fluids. This is better than a rehydration of the organism by plain water. The reason is that pure water gets rapidly absorbed from the gut and will lead a decrease in sodium in the plasma. This will induce an increase in urine production and thereby increase the body's fluid losses. The small amount of salt is needed to stimulate the thirst drive. The addition of carbohydrates to the fluids can enhance the intestinal absorption

of water. So, for example, Gisolfi et al. [77] observed that a 6% carbohydrate-electrolyte (2% glucose, 6% sucrose, 20 meq Na^+, 2.6 meq K^+) solution was absorbed sixfold faster than water. However, solutions containing carbohydrate concentrations >10% will cause an osmotic-driven net movement of fluid into the intestinal lumen when such solutions are ingested during exercise. Thus, an effective loss of water from the vascular compartment will occur. This will impair cardiovascular function, leading to a fall in blood flow to muscle, skin, and other tissues [78,79].

5.9.2 Fever

Fever is not hyperthermia because the core temperature setpoint is changed via order of the hypothalamus. Fever is thus the symptom of an acute adjustment of the body to ensure this higher setpoint. The change in the setpoint is initiated by pyrogenic substances [6,80–81]. A distinction is made between exogenous and endogenous pyrogens. Exogenous pyrogens are viruses, bacterial toxins, LPSs, as well as muramyldipeptides of bacterial membranes. These pyrogens stimulate granulocytes and macrophages to release a whole series of large hydrophil polypeptides or proteins (interleukin-1 beta, interleukin-6, TNF-alpha, interferon) into the blood (Figure 5.21).

These cytokines are referred to as endogenous pyrogens. Today it is assumed that not only one single cytokine is responsible for the initiation of fever but that a simultaneous stimulation of various cytokines is necessary (cytokine cocktail). In order to induce fever, these cytokines have to come into contact with the neuronal structures of the preoptic area in the anterior hypothalamus. Due to their size, the mentioned cytokines are actually incapable of passing the blood-brain barrier. The fenestrated capillaries in the organum vasculosum laminae, a structure located in the direct vicinity of the POAH, however, enable a transfer to the endogenous pyrogens. Here the cytokines are supposed to activate monocytes, endothial and glia cells expressed in the tissue that effect an increase in the prostaglandin-E2 production. This prostaglandin-E2 is now able to pass the ependymal blood-brain barrier. It is not entirely clarified whether prostaglandin-E2 will then have a direct effect on the neurons of the POAH or whether also neurons in the organum vasculosum laminae are interposed. The fact that prostaglandin-E2 inhibitors like actylsalicilic acid or indomethacin have a fever-reducing (antipyretic) effect indicates, among other things, that prostaglandin-E2 plays a decisive role in the mediation of an adjustment of the setpoint value. The observation has been made that sometimes after intravenous injection of exogenous pyrogens, an increase of the setpoint value is observed even before a measurable increase of endogenous pyrogens in the blood. This has led to the search for alternative signal transduction pathways. A possible neural pathway could lead from the liver via the nervus vagus (N. vagus) to the POAH. The Kupffer cells possess macrophages that come into contact with the exogenous pyrogens in the circulating blood. Via mediators released by the Kupffer stellate cells a stimulation of

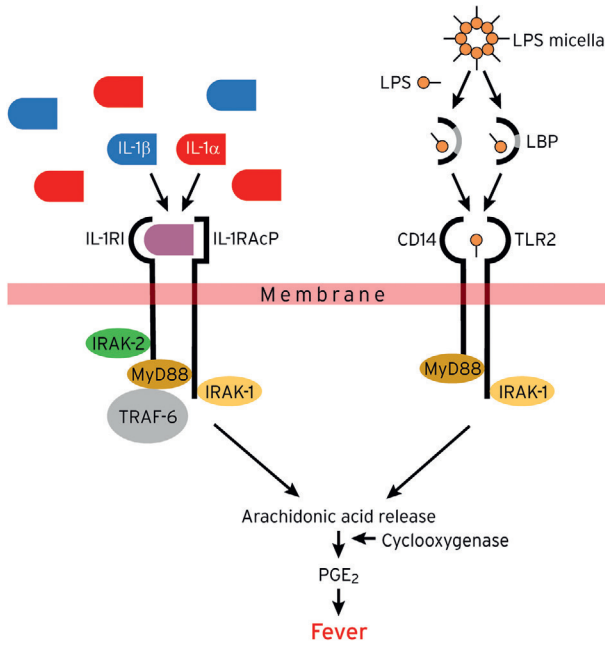

FIGURE 5.21 Fever and presumable pathways of fever induction via IL-1 alpha and IL-1 beta cytokines (pathway A, left axis) as well as lipopolysaccharides (LPSs), CD14, and toll-like-receptor 2 (TLR2) (pathway B, right axis). According to Netea et al. [81]2000 and Dinarello [80]2004, in pathway A IL-1 (IL-1α and β) binds to its cellular receptor type I (IL-1RI) and the IL-1 receptor accessory protein (IL-1RacP). This leads to signal transduction via receptor associated proteins IRAK-1 and -2, MyD88, and TRAF-6 with release of arachidonic acid and prostaglandin-E2 (PGE2) and finally induction of fever. In pathway B the LPS complex acts with LPS-binding protein, which enables binding of LPS to CD14 and toll-like receptor 2 (TLR2). Thereafter, the signal transduction pathway is very similar to that of the IL-1 receptor. Here too, MyD88 and IRAK-1 are activated, arachidonic acid is released, and fever is induced through PGE2. *Adapted from Ref. [81].*

the afferent parts of N. vagus occur. The N. vagus could, via its nuclei in the medulla oblongata and from there via projections to the POAH, change the setpoint directly or via a stimulation of the prostaglandin-E2 production in the POAH, which, as the main control center of thermoregulation in humans, would induce an adjustment of the core temperature setpoint (Figure 5.22) [6].

In addition to this classical model, others have postulated there might be alternative models. These pathways are summarized in Figure 5.23 [81].

However, if fever is induced, three phases can be distinguished: (i) increase of fever, (ii) plateau phase of fever, and (iii) decrease of fever (Figure 5.24). Usually, fever follows the normal fluctuation pattern of body temperature, only on a distinctly higher level. That is why fever, just like the normal body temperature, is higher in the evening (evening "peaks") than in the morning. After the setpoint has been shifted to a higher level (first phase), for example, a core

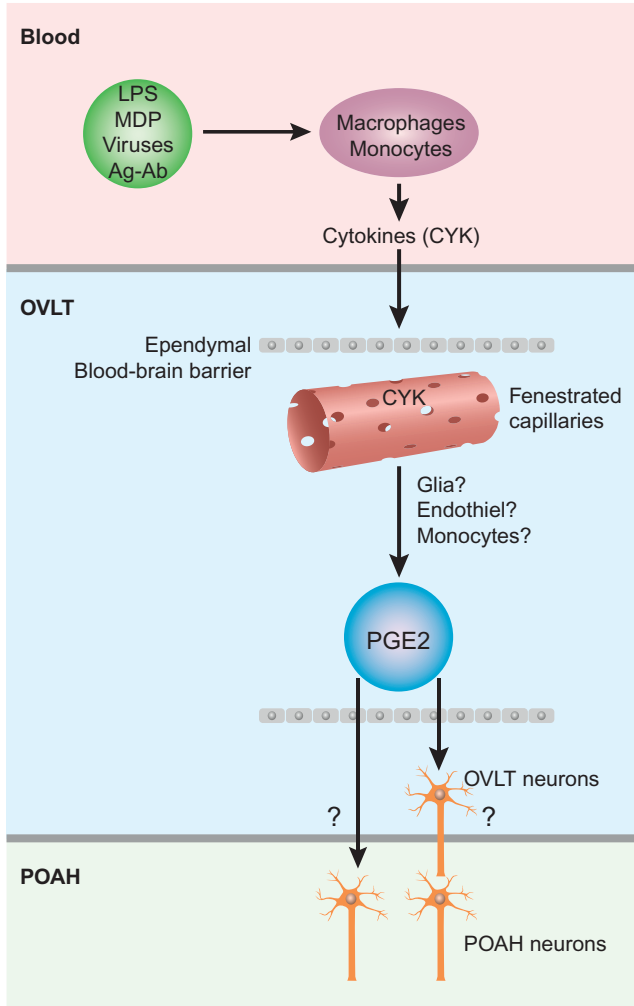

FIGURE 5.22 Hypothetical scheme showing the anatomical structures and physiological transmitters involved in the induction of fever in humans according to Jessen (Jessen 2001). Exogenous pyrogens (lipopolysaccharides [LPS], muramyl dipeptide [MDP], viruses, antigen-antibody complexes [Ag-Ab]) in the blood come in contact with immune-competent cells (macrophages, monocytes). These immune cells release thereafter cytokines such as interleukin-1 alpha (IL-1 alpha), interleukin-1 beta (IL-1 beta), and/or interleukin-6 (upper panel). Through the fenestrated capillaries of the organum vasculosum laminae terminalis (OVLT) in the vicinity of the POAH, these relative large cytokines can pass into the perivascular space. There, the cytocines are activating resident monocytes, endothiel, or glia cells that trigger the production of prostaglandin-E2 (PGE2). It is currently not fully understood how PGE2 acts on the POAH. Either the PGE reaches the POAH via diffusion and/or special neurons, which then induce the upward change of the setpoint of core body temperature. *Adapted from Ref. [6].*

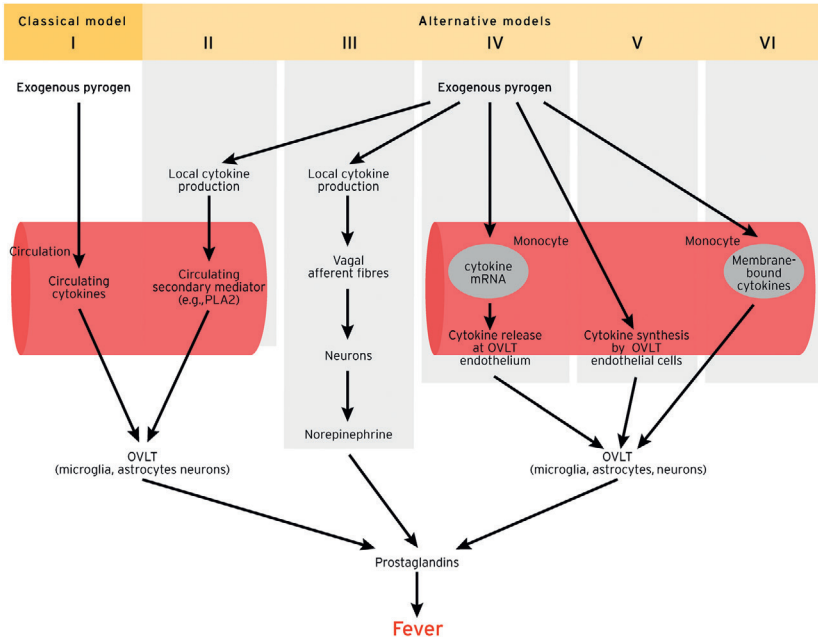

FIGURE 5.23 Additional pathways might be able to trigger OVLT (organum vasculosum laminae terminales). *Adapted from Ref. [81].*

body temperature of 40 °C, the body reacts in the same way as if it were exposed to a cold environment. This leads to an increased cold sensation, a strong vasoconstriction in the skin vessels (reduction of heat loss), and the occurrence of muscle shivering. In this phase, the person concerned looks pale, has cool acrae and warm or hot regions that are located close to the body core (neck, forehead). When the new setpoint temperature is reached, a plateau phase follows, the length of which depends on the disease (second phase). Actually, the plateau phase is characterized by the fact that no activation of heat loss mechanisms can be observed in the subject such as profuse sweating or a massive increase in peripheral skin blood flow or vasodilation. In short, the body is handling the increased temperature as if it were normal. This lack of increased heat loss mechanisms at a core temperature distinctly above normal temperature distinguishes fever from hyperthermia, for example, by physical exertion or strong external heat supply. During the decrease of fever (defervescence), the setpoint temperature is shifted back to a normal temperature around 36.5-37.0 °C. As a result the setpoint temperature lies now distinctly below the actual core body temperature, which as a rule leads to an increased activation of the heat loss mechanisms (evaporation, vasodilation, increased skin blood flow). Due to the rapid vasodilation and the relatively too low blood volume in consequence, a febrile circulatory collapse with warm, moist skin, tachycardia, and low diastolic blood pressure can follow during the critical defervescence phase.

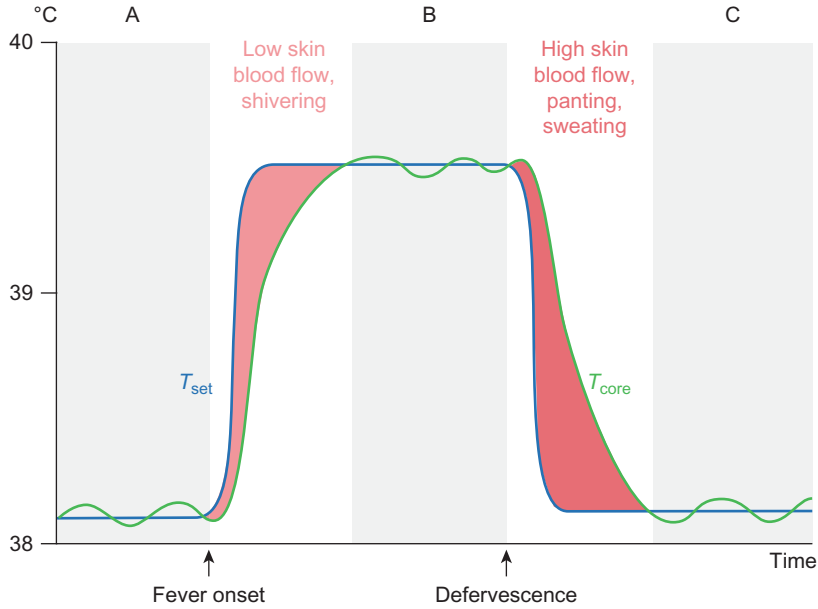

FIGURE 5.24 Typical time course of fever. First of all, an adjustment setpoint (blue line) of the core body temperature is initiated by the anterior hypothalamus (thermoregulatory center) (A). The actual core temperature (green line) follows with a delay (time lag) (increase of fever). In the plateau phase, actual core temperature measured and setpoint value are equal (B). The decrease of fever is initiated by an adjustment of the setpoint value to a lower body temperature, which is again followed by the actual core temperature with a delay (C). The typical physiological accessory symptoms during the increase and decrease of fever are shown in the illustration. *Adapted from Ref. [6].*

5.10 THERMOREGULATION AND CLIMATE

Environmental conditions have a significant influence on the human thermoregulation and the feeling of comfort.

5.10.1 Definitions

5.10.1.1 Atmosphere, Climate, Weather, Microclimate

The layer of air held by the gravity of the Earth consists of different gases (nitrogen, oxygen, CO_2, inert gases) as mentioned before. In meteorology, the specific qualities of the atmosphere above a defined region of the Earth are summarized under the term climate. These qualities are decisively determined by the position above the surface of the Earth. An observation period of at least 20 years is prerequisite for the characterization of a climate (altitude, tropic, desert, and polar climate). The recorded meteorological factors are, among others, air temperature, relative air humidity, wind velocity, as well as amount and temperatures of radiation. A season is a term given to the state of the atmosphere over a period of three to four months (seasons spring, summer, autumn, winter). If the state of the atmosphere can be predicted for 48 h, this is referred to as weather

[35]. A special bioclimate for humans is the indoor climate, because, besides the climatic parameters, nonclimatic variables such as protective function (housing) or the thermic resistance of the clothing play a significant role for the feeling of comfort; the latter is also known as microclimate.

5.10.1.2 Climate Indices

The mentioned climatic and nonclimatic factors can be represented individually or as climate indices. For the understanding of the climate index curves, it is important that individual climate factors can be compensated, alleviated, or augmented in their effect on the organism by simultaneous changes of other climatic and/or nonclimatic variables. If, for example, an air temperature that has been perceived as comfortable is increased, no feeling of heat originates with simultaneous augmentation of the air movement. In this case, the increased convective heat losses compensate the effects of the increased air temperature. Climate indices thus give numerical values for the individual climate values leading to a synopsis of an identical effect on humans. Besides room temperature and air velocity, the relative air humidity is depicted as a further important climatic variable. It becomes evident that with low relative humidity (10%) and high wind velocity (3.0 m/s) an actual air temperature of 37 °C is perceived by the organism as 25 °C (comfort). However, a combination of high relative humidity (95%), low wind velocity (0.1 m/s), and a room temperature of 29 °C is perceived as a disagreeable, damp-warm indoor-climate (discomfort). Relevant detailed studies with various climatic combinations have led to the term *effective temperatures*. A distinction is made (i) between normal effective temperature that applies to persons with usual streetwear (clothing) and (ii) basal effective temperature, which applies to persons with unclothed upper part of the body. However, the same room temperature can be perceived quite differently by different persons with the same clothing and activity. Wearing light summer clothing with a relative air humidity of 50% and a wind velocity of 0.1 m/s, most test persons regard an indoor climate of 25-27 °C as comfortable. For some, however, this room climate might already be "too cool". The reason for this subjectively different thermic perception lies in the individually different balance of heat production and heat loss depending, among other things, on numerous factors like age, body size, body composition, and hormone levels [35].

5.11 OUTLOOK: GLOBAL WARMING AND HUMAN HEALTH

The WHO report by Campbell-Lendrum et al. [82] as well as more recently the Intergovernmental Panel on Climate Change (IPCC) [83] stated that climate change is an emerging risk factor for human health due to the fact that almost the entire planet has experienced surface warming (Figure 5.25).

According to Cubasch [85], we have to consider that each of the last three decades in particular has been successively warmer at the Earth's surface than any preceding decade since 1850 (Figure 5.26).

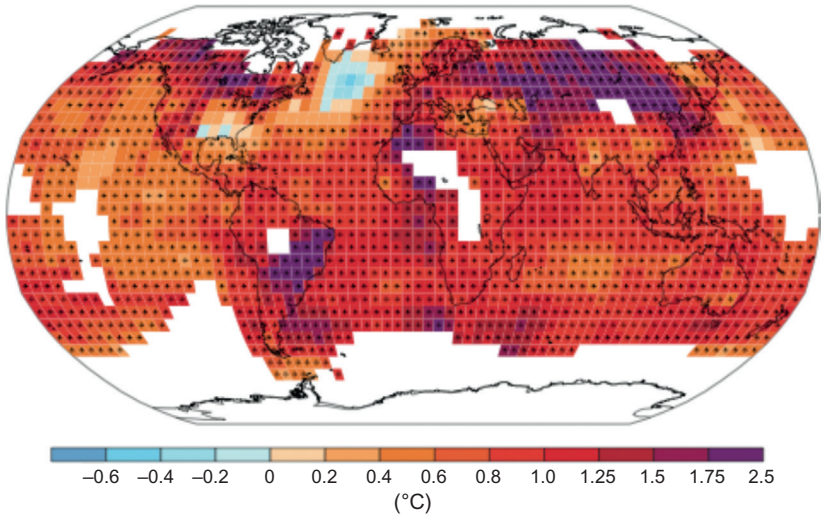

FIGURE 5.25 Observed change in surface temperature 1901-2012 (Stocker, 2013) [98].

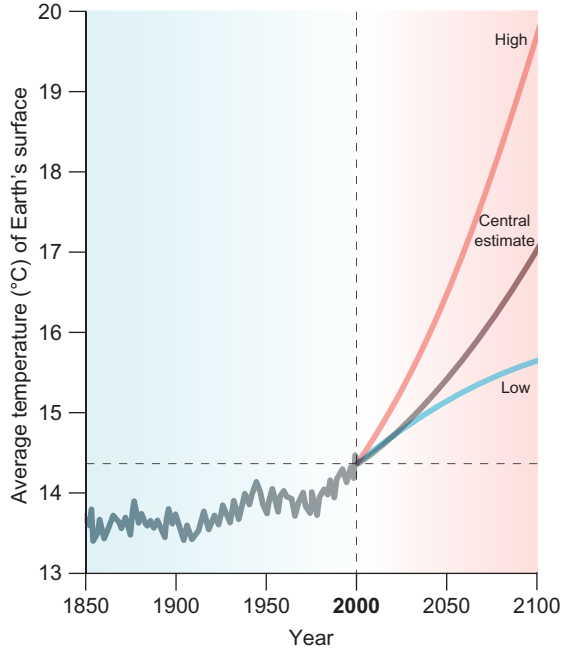

FIGURE 5.26 Global temperature record since instrumental recording began in 1860 and projection to 2100 according to the IPCC (Stocker, 2013).

In addition to that, the Northern Hemisphere during the time span of 1983-2012 likely experienced the warmest 30-year period of the last 1400 years. Also, the globally averaged combined land and ocean surface temperature data as calculated by a linear trend showed a warming of 0.85 °C over the period 1880-2012. When multiple independently produced datasets exist, the total increase between the average of the 1850-1900 period and the 2003-2012 period is 0.78 °C. The continental-scale surface temperature reconstructions show multi-decadal periods during the Medieval Climate Anomaly (year 950-1250) that were in some regions as warm as in the late twentieth century. These regional warm periods did not occur as coherently across regions as the warming in the late twentieth century (Figure 5.27). Changes in many extreme weather and climate events have been observed since about 1950. Finally, it is likely that the number of these heat waves has increased in large parts of Europe, Asia, and Australia.

Global warming, along with the growing incidence of extreme climatic conditions and the rising population densities in metropolitan areas lead to considerable increases in risk for human health [83,84]. Furthermore, this could lead to a substantial reduction in economical productivity [85]. Thus, there should be increasing interest from the scientific and political communities to explore, understand, and answer the emerging questions of the impact of global warming and heat waves on the health of the different populations around the world (rural and urban) and its impact on economy, with the ultimate aim to develop multifactorial preventive strategies for reducing climate-related issues such as morbidity and mortality [86,87]. For example, several studies have shown that

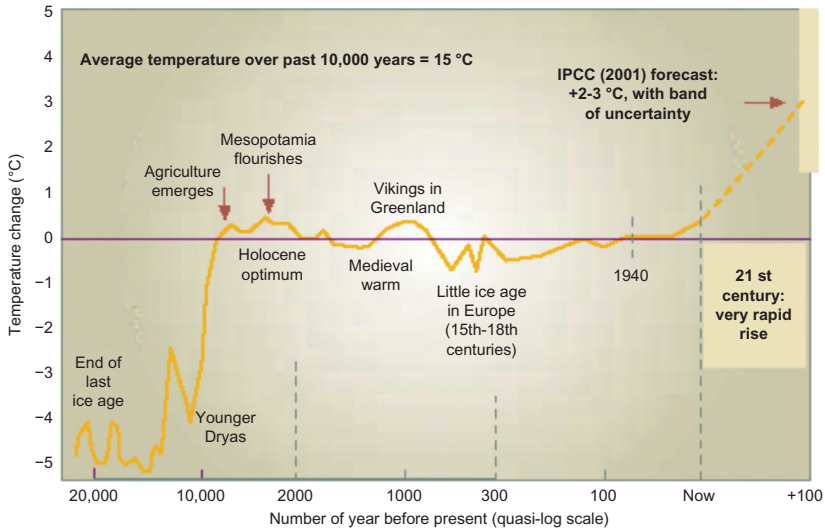

FIGURE 5.27 Average global temperature changes in the last 20,000 years.

increased rates of morbidity and mortality during heat waves can be found especially in risk groups such as children in their first year of life, the elderly, bedridden patients, drug addicts, alcoholics, diabetics, immobile and homeless persons [67,68,88]. Preventive measures demand an awareness of the problems, not only in the population, but also in many responsible governmental institutions, and especially in the group of medical professionals and social services [8]. More than 70,000 additional deaths occurred in Europe during the summer of 2003 [84]. Major distortions occurred in the age distribution of the deaths (older population), but no harvesting effect was observed in the months following August 2003 [84,86,87]. Thus, warming constitutes a new health threat in an aged Europe that may be difficult to detect at the country level. Depending on its size, for example, centralizing the count of daily deaths on an operational geographical scale seems to be a clear priority to Public Health Service in Europe. Furthermore, a change in lifestyle is required in the group of people mentioned above, including dietary measures, sufficient fluid intake, and regulation of room climate. To gain a better understanding of heat waves on human health, a new generation of web-based, autonomous, noninvasive, light, and easy-to-handle methodologies have to be developed for collecting significant physiologic information on the impact of heat stress on humans in rural and urban areas compared with people in metropolitan areas. Such technology could be used to build a database that will support the understanding of the relationship between extreme climate conditions during sleep, rest, and work and the potential health risks. Moreover, this data could be used to develop a computer model as an early warning system for predicting urban population- and region-specific human health risks of future extreme climate condition situations, and provide architectural assistance and support, for example, in future city developments to optimize the protection of the population from climate hazards [83]. Especially, in recent years, heat waves were found to commence earlier in the year and to last longer, locally accompanied by heavy rain and varying levels of humidity. In the near future, we have to face even worse global weather conditions with increasing global temperatures [83]. The three different kinds of estimations concerning the global temperature development for the twenty-first century given in Figure 5.26 exceed by far the human comfort interval and must be looked upon as an extreme strain, because the body's heat-defensive mechanisms have to be permanently active. Full recovery during sleep is almost impossible under heat wave conditions, because night temperatures are kept high, so that the cardiovascular system is permanently stressed during day and night, causing significant increases in morbidity and mortality. Furthermore, since 2007 more people are living in cities than in rural areas, which means that in the near future these extreme climates will hit a population that is growing older, has impaired levels of fitness, and suffers increasingly from diseases such as diabetes, cardiovascular deconditioning, and dementia. Another severe factor is the exponentially increasing level of obesity and adiposity in the population. In addition, due to, for example, cardiovascular diseases, patients are forced to

take medications that constrain the body's defensive mechanisms against heat. The aging, currently about 30-40% of the population in German cities, has to be considered as a risk group [84]. These people are frequently not considering themselves at risk, and consequently do not take any precautions and are mostly unable to maintain appropriate behavior to extreme climates such as adequate fluid intake. This consideration is of utmost importance, because the behavior of humans determines 90% of their thermoregulation. As outlined before, the body's own defensive mechanisms are of high importance, although they cannot compensate for the substantial consequences owed to deficits in behavior. While the statistical link between heat stress and health risks has been well documented, a better understanding of the relationship between climate conditions and health risks is clearly needed. Therefore, from a technological perspective, to achieve this goal specifically, new methodologies have to be available that can simultaneously record, for example, core and body shell temperatures, heart rate, activity and resting periods, sleep stages, as well as environmental conditions (ambient temperature, humidity, and atmospheric pressure), some of them already described in chapter 2. Furthermore, research studies have to be initiated (i) in a larger and longer scale, (ii) in different age groups, (iii) in healthy and unhealthy subjects, (iv) under various environmental conditions (seasons), and (v) under different physical activities. Once the feasibility of such technology is confirmed, it could be used to provide the basis for the development of a prediction system for determining the impact of heat stress on humans. This would promote, for example, the generation of human heat stress data in representative samples in diverse urban areas during different environmental conditions, and provide the basis to link research on climate change and health to other research disciplines such as meteorology, informatics, architecture, and engineering. These collaborative efforts could be used to develop computer models for predicting urban population- and region-specific human health risks of future extreme climate conditions, and to provide continuous support regarding preventive strategies for reducing climate-related morbidity and mortality. Furthermore, the development of individual mobile health risk monitoring systems could be used to assist local, regional, and global rural/urban infrastructure and architectural development planning [85].

These new methodologies and datasets could be used to assess in the impact of global warming on occupational health issues. According to the Kjellstrom study on "Global Assessment of the Health Impacts of Climate Change" [89], it is quite clear that occupational heat stress is already a significant problem in East Asia, South Asia, Southeast Asia, Central America, tropical Latin America, North Africa, East Africa, West Africa, and the Middle East. The study estimates that the global number of occupational heat stress fatalities due to climate change (additional workplace deaths) may amount to 12,000-30,000 cases in 2030 and 26,000-54,000 in 2050 [86,87,89]. For nonfatal heat stroke cases (in addition to cases in 1975), study calculations indicate 35,000-65,000 in 2030 and 40,000-73,000 in 2050. According to the study, there could also

be more than 20 million heat exhaustion cases globally in 2030 due to climate change and possibly 40 million cases in 2050 (each case assumed to be seriously affected for 1 day). This would lead to a loss of work capacity globally of 1.0-1.7% (depending on climate model used) in 2030 and 1.7-2.4% in 2050. As these authors state, this may look like small changes, but in the worst affected regions (South Asia and West Africa), the estimated annual work capacity losses at population level are at least twice as high, not to mention that a healthy workplace climate can also be considered as a component of human rights to health. They also calculated the direct economic impact from sustained reductions in work output and gave an interesting example. If the economic growth per person of an economic unit (country, province, locality, company) is assumed to be 4% per year, in a 30-year period the income per person would increase from $2000 per year to $6500. If this growth was undermined by a 1% or 2% annual productivity loss, the resulting income after 30 years would be $4900 and $3500 respectively, which indicates losses of 36% or 67% of the additional economic growth. In this way, the climate change impact on work productivity clearly undermines the efforts to achieve the economic improvement targets in the Millennium Development Goals [87,89]. If the work capacity losses are expressed as Disability Adjusted Work Years lost (= lost years of fully healthy life in the age range 15-64 years) called DAWYs, the impact is even more apprehensible: The "health" losses in the hottest regions are similar to those caused by all cases of tuberculosis or all injuries in this age range. In their most interesting study, Kjellstrom et al. concluded that increasing occupational heat stress due to climate change is a very significant health and welfare challenge in tropical and other hot parts of the world [87,89,90].

In conclusion, (i) there are several proofs that the planet is warming; (ii) this is mainly due to human activities; (iii) this trend will continue for several decades or longer [91]; so that (iv) weather and climate will exert an increasing major influence on human health by such means as heat waves, floods, and storms, and of more indirect influences (v) on the distribution and transmission intensity of infectious diseases, as well as on the availability of freshwater and food [91–93]; and (vi) climate change has now been recognized as a global dimension issue that will increasingly affect human health and well-being on a wide scale.

5.12 SUMMARY

Man is counted among the endothermic organisms (mammals, birds) whose body temperature (36-40 °C) lies distinctly above the average temperature of their living environment. This high temperature gradient can be maintained only if heat development and heat loss are in balance. This heat balance is enabled by a high basal energy rate (tachymetabolism), insulating layers for the reduction of heat losses (subcutaneous fat tissue, hair coat), as well as complex mechanisms of temperature regulation (e.g., blood flow regulation, sweating). This enables endothermic organisms to keep their body temperature constant during

a wide range of different states of activity and under wide ranges of different varying environmental conditions. Ectothermic organisms (reptiles, fishes), however, have a metabolism three to four times lower (bradymetabolism) and are less adaptive to changes in environmental temperatures. The high body temperatures of endothermic creatures (animals, animate beings) permit an active, all-year-round way of life, largely independent of environmental conditions. The temperature within the body, however, varies at different anatomical sites and depends on the respective metabolic activity of the organs (heat production). In the body core (brain, heart, liver) the tissue temperature is higher than in the periphery (legs, arms) under resting conditions. Furthermore, the subjective perception of temperature differs individually. It is influenced not only by air temperature, but also by air humidity and wind velocity. Temperature that is perceived as neither too hot nor too cold is called the indifference temperature. Under these conditions, heat production and heat loss are in balance.

Different transport mechanisms are available for heat transport from the body core to the periphery (internal heat transport) and from the periphery to the environment (external heat transport). The internal heat transport is affected mainly by means of blood circulation in a convective manner (convection = heat transport by means of a moving medium). Internal heat is also transported from the vascularized subcutaneous tissue to the body surface via vasoconstriction and vasodilatation. This is apparent especially in the extremities and acrae (e.g., hands, fingers, feet, ears) where the internal heat flow to the periphery can be dramatically increased or decreased. External heat transport in humans is enabled additionally by means of evaporation (evaporation of sweat) and radiation (long-wave, infrared radiation). Under resting conditions and ambient indifference temperature (27-31 °C), heat loss by radiation prevails. With physical work and/or warm environmental conditions, the organism depends more and more on evaporative heat loss. Physical and chemical processes provoking heat production or loss are regulated by the hypothalamus (preoptic area) by comparing the afferent information with an intrinsic reference value. This reference value is generated in the hypothalamus and shows cyclic changes throughout the course of the day (circadian rhythm). Hyperthermia (body core temperature >37.5 °C) is characterized by a disproportion between heat loss and heat production. However, the reference value, the so-called core temperature setpoint, which is created in the preoptic area of the hypothalamus, is unchanged in hyper- and hypothermia. During an increased metabolism, for example, strenuous physical exercise or heavy external heat gain (sauna), body core temperatures may rise rapidly to >40 °C. As a consequence, heat cramps, heat collapse, heat exhaustion, and even a life-threatening heat stroke may result. Hypothermia exists when the body temperature lies at or <35.5 °C. In cold water (5-10 °C), this value can be reached after just 10-20 min. Elderly people with a reduced metabolism, and infants (unfavorable surface-volume relationship) are particularly prone to suffering from hyper- or hypothermias. During a classic fever, in contrast to hyper- and hypothermia, the core temperature setpoint

is up-regulated and mediated by exogenous and endogenous pyrogenes. A rising fever leads to a cold sensation and—among other features—is associated with increased heat production via muscle shivering. With declining fever, an adjustment back to normal core temperature takes place, which leads to promotion of heat loss mechanisms such as sweating and increased blood circulation of the skin. If heat losses exceed the production of heat in the organism for a longer period of time, the body core temperature continuously decreases.

With global warming, it can be foreseen that there will be a growing incidence of extreme climatic conditions. This will lead to considerable health problems and a substantial reduction in economical productivity. Thus, there should be increasing interest from the scientific and political communities to explore, understand, and answer the emerging questions of the impact of global warming on the public health of different populations around the world (rural and urban) and its impact on economies, with the ultimate aim to develop multifactorial preventive strategies for reducing climate-related issues such as morbidity and mortality.

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