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Abstract
Thermoregulatory function, that is, heat dissipative responses such as skin blood flow (SkBF) and sweating to an increased body temperature, is critical during physical work or exercise in warm and hot conditions and during hyperthermia. Thermoregulatory function is associated with individual somatotype, fitness level, normal aging, and physiological status and diseases. Individuals with type 2 diabetes have decreased thermoregulatory responses compared with healthy counterparts, characterized by decreased SkBF and sweating. The decreased SkBF and sweating would be associated with the reduction in nitric oxide bioavailability and endothelial functions in skin vasculatures, also with central mechanisms, and so on. Aerobic exercise training and/or acclimation to the heat improve heat dissipative responses in healthy subjects. The effects of exercise training in type 2 diabetics on glycemic control are well established while it remains unclear that high levels of aerobic fitness or exercise training in diabetics improve thermoregulatory function during heat stress.

Keywords: thermoregulation, sweat rate, skin blood flow, plasma volume, aging, diabetes

1. Introduction
Individuals are more likely to become exhausted and to develop heat-related illnesses when physical work or sporting activities are performed for a prolonged time in warm and hot conditions or direct sunlight compared to in cooler conditions. Thermoregulation is one of the most important physiological functions for when individuals are exposed to extreme hot environments. The incidence of heat-related illnesses is particularly great if physical work or
exercise is performed at higher intensities and in higher ambient temperatures (T_a) and relative humidity (RH) [1]. In addition, the incidence is greater in persons who are dehydrated, who are not acclimated to hot environments, or who have low levels of physical fitness and daily activity even if they are healthy [1]. More importantly, the incidence is greater in individuals with obesity, diabetes mellitus, and cardiovascular diseases, due to impaired thermoregulatory responses [1, 2]. It is also greater in individuals with attenuated thermoregulatory responses, such as those with skin grafts, spinal cord injuries, and multiple sclerosis [3–5]. Moreover, an extreme hot environment can be dangerous for the elderly (even those who are healthy) because of the normal aging process [1, 2]. Over the last two decades, the morbidity and mortality of heat-related illnesses has rapidly increased in Japan because of the rapidly aging population and global warming [6]. This chapter first discusses thermoregulation during work and exercise in warm and hot conditions and the possible physiological effects of humoral and other important factors on thermoregulation. Second, the effects of diseases such as diabetes on thermoregulatory responses are discussed.

2. Thermoregulation in warm and hot conditions

Core body temperature is determined by the heat equilibrium between heat gain and loss [1]. Typically, core body temperature is elevated when we face continuous whole-body work and exercise. This is because only approximately 20% of the energy produced in contracting muscles is used for muscle contraction; the remaining 80% is converted to heat energy, and therefore exercise causes an increase in muscle temperature. The heat is distributed to the body by the circulation and increases body temperatures. Therefore, greater exercise intensities are associated with greater heat production during exercise. However, although core body temperature elevates rapidly within several minutes of starting exercise, heat dissipation mechanisms are activated sufficiently to balance heat production and eventually the increase in core body temperature reaches a steady state. The cutaneous vasculature and sweat glands are thermoregulatory effectors of the skin and promote heat loss by increasing skin blood flow (SkBF) via cutaneous vasodilation and by sweating, respectively [1]. The heat generated in contracting muscles is transferred, because of circulation of blood, to the skin surface; thus, skin surface temperature is elevated by the increased SkBF. The heat transferred to the skin surface is released to the surrounding air according to the thermal gradient between the skin and the air (non-evaporative heat dissipation). Therefore, in a cool environment, non-evaporative heat dissipation is effective; however, in warm environments, when T_a is higher than skin temperatures (~30°C), it becomes ineffective or even negative and acts as a source of heat gain. Conversely, sweating enhances heat loss via sweat evaporation from the skin surface to the air (evaporative heat dissipation). Evaporative heat dissipation is determined according to the gradient of water vapor between the skin surface and the surrounding air. Therefore, it is available regardless of T_a and is the main and critical heat loss mechanism when T_a exceeds ~30°C; however, it is attenuated in a humid environment. Consequently, when heat gain exceeds heat loss during high-intensity work and/or exercise in a hot and humid environment, core body temperature increases rapidly. Therefore, these conditions increase susceptibility to heat-related illnesses.
Physiologically, SkBF and sweating are regulated by body core and skin temperatures (thermal factors) and also other (non-thermal) factors [1]. Body core temperature is monitored continuously by central thermoreceptors. On the other hand, skin temperatures are monitored continuously by the peripheral thermoreceptors. These receptors send received thermal information through an afferent pathway to the thermoregulatory center in the preoptic/anterior hypothalamus [1]. When the hypothalamic thermoregulatory center determines that core and skin temperatures are elevated compared to the set point, SkBF and sweating are increased through the reflex efferent pathways of sympathetic nerve systems [1]. In humans, SkBF and sweating are neutrally controlled via two distinct skin sympathetic nerves. A sympathetic adrenergic vasoconstrictor system is one of the neural control mechanisms for SkBF and the other is a separate sympathetic cholinergic vasodilator system [1]. Sweating is controlled by sympathetic cholinergic nerves [1]. In addition, non-thermal factors modify the thermal reflex, including central commands, mechano- and metabo-reflexes, arterial and cardiopulmonary baroreflexes, blood volume and osmolality, and mental stimuli [1]. During exercise, they exert different effects on SkBF and sweating responses [1]. Typically, SkBF at the same levels of core and skin temperatures is lower during exercise than resting conditions, and this restriction is greater at higher exercise intensities. Conversely, sweating at the same levels of core and skin temperatures is likely to be higher during exercise than resting conditions.

When levels of dehydration worsen, the risk of heat-related illnesses is much higher during work or exercise in a hot environment [7]. Rothstein and Towbin [8] described for the first time the effects of dehydration on thermoregulation in humans. They reported that an ~0.3°C increase in rectal temperature is caused by every 1% body weight loss by sweating in breaks between exercises when soldiers march in the desert. This is because, as non-thermal factors, dehydration-induced hypovolemia and hyperosmolality limit the response to enhance SkBF and sweating to increased body temperature. Decreased body fluid volume and increased osmolality change dramatically during exercise even in a cool environment. Indeed, plasma volume (PV) is decreased with an increased intensity of exercise. In maximal exercise, the decrease in PV is reached by approximately 300–500 mL (8–15%). Increased capillary fluid filtration from the intravascular to the extravascular spaces due to an increase in blood pressure and peripheral vasodilation during exercise causes the PV change. Additionally, metabolites accumulated in the intracellular fluid of contracting muscles such as lactic acid increases free-water shift from the plasma to the intracellular fluid through the interstitium based on the osmotic gradient [9]. It is estimated to be ~1 L of the volume shifted to the contracting muscles at maximal exercise. About half of this water is from PV [10]. In addition, plasma osmolality ($P_{\text{osm}}$) is increased from ~285 mosmol/kgH$_2$O at rest to over 300 mosmol/kgH$_2$O at maximal exercise due to accumulated metabolites. These humoral changes are not caused by dehydration due to sweating; rather, they are associated with the exercise itself. Because they are observed within several minutes after the start of exercise, in addition, when exercise is performed in warm and hot conditions, hypohydration due to the decrease in PV and hyperosmolality due to the increase in $P_{\text{osm}}$ during exercise is exacerbated because of increased SkBF and sweating for thermoregulation. Enhanced SkBF induces a reduction in venous return to the heart due to pooling of excessive blood in dilated peripheral vasculature of the skin [11]. Additionally, ~10% of the sweat volume is lost from plasma fluids, and $P_{\text{osm}}$ is increased according to sweat rate because sweat is hypotonic compared to body fluids. These humoral changes enhance the load on the circulation and act as limiting factors for thermoregulation.
Nadel et al. [12] first showed that isotonic hypovolemia (change in PV, −700 mL; body weight, −2.7%) achieved by diuretic administration before exercise induced upward shift of the esophageal temperature (T es) threshold for the onset of cutaneous vasodilation by 0.4°C during exercise at 55% of maximal oxygen consumption rate (VO2max) in a hot environment (T a, 35°C). It also reduced the SkBF at peak value by about 50% compared to control condition. Besides, Mack et al. [13] reported that an acute reduction in venous return to the heart induced by application of −40 mmHg lower body negative pressure (LBNP) decreased SkBF and elevated T es during exercise at an intensity corresponding to a heart rate of 125 beats/min in a warm environment (T a, 28°C). In contrast, all techniques to acutely increase venous return during exercise, including isotonic hypervolemia with saline infusion [11], postural change from upright to supine position [14, 15], head-out water immersion [16], or continuous negative pressure breathing [17], enhanced the response to increase SkBF to an increased core body temperature during exercise. From these observations, the response to increase SkBF to an increased core body temperature during exercise is attenuated by dehydration-induced hypovolemia via the cardiopulmonary baroreflex. Recently, Ogawa et al. [18] reported that the efferent sympathetic signals to skin vasculature, that is, the skin sympathetic nerve activity (SSNA), component synchronized with the cardiac cycle was decreased by postural change from supine to 30° head-up tilt during hyperthermia (T es increased ~0.7°C) with passive heating and that the responses were correlated with the decrease in cutaneous vasodilation. With these data, they suggested that the SSNA component synchronized with the cardiac cycle was likely to contribute to the suppression of cutaneous vasodilatation.

Previous studies have suggested that the response to increase sweat rate during exercise is also attenuated by hypovolemia. Fortney et al. [19] presented that the ability to enhance sweating of the chest and forearm to an increase in T es but not the T es threshold at the start of sweating during cycle ergometer exercise (65–70% VO2max) in a warm environment (T a, 30°C; RH, 40%), was attenuated by isotonic hypovolemia (9% reduction in plasma volume induced by diuretics) prior to exercise. Additionally, Mack et al. [13] showed that LBNP at the level of −40 mmHg during exercise dampened the sweat rate and SkBF to increased T es. Dodt et al. [20] also observed decreased skin sympathetic nerve activity following LBNP in passively heated subjects. In contrast, Kamijo et al. [21] reported that isotonic hypovolemia induced by diuretic (−10%) before exercise dampened increases in SkBF, similar to the results of Nadel et al. [12]. However, it did not dampen the increase in sweat rate during exercise at 60% VO2max in a warm environment (T a, 30°C; RH, 45%). Thus, the effects of hypovolemia on sweat rate are still debatable.

Plasma hyperosmolality suppresses the response to increase SkBF and sweating during exercise. Fortney et al. [22] showed that 10 mosmol/kgH2O increases in P osm achieved by hypertonic saline infusion before exercise caused an increased T es thresholds for cutaneous vasodilation and sweating by 0.2°C. In addition, the sensitivity to increase SkBF in response to increased T es decreased during exercise at 70% VO2max in a warm environment (T a, 30°C; RH, 40%). Additionally, onset of SkBF and sweat rate responses to increased core body temperature is delayed by plasma hyperosmolality. Takamata et al. [23] showed a linear upward shift of the T es thresholds with several levels of increase in P osm by hypertonic saline infusion. They suggested with these data that T es thresholds for cutaneous vasodilation and sweating during passive heat stress (lower-leg water immersion, 42°C) at rest
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