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REVIEW | *Recovery from Exercise*

Restoration of thermoregulation after exercise

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Kenny GP, McGinn R. Restoration of thermoregulation after exercise. *J Appl Physiol* 122: 933–944, 2017. First published November 23, 2016; doi:10.1152/japplphysiol.00517.2016.—Performing exercise, especially in hot conditions, can heat the body, causing significant increases in internal body temperature. To offset this increase, powerful and highly developed autonomic thermoregulatory responses (i.e., skin blood flow and sweating) are activated to enhance whole body heat loss; a response mediated by temperature-sensitive receptors in both the skin and the internal core regions of the body. Independent of thermal control of heat loss, nonthermal factors can have profound consequences on the body's ability to dissipate heat during exercise. These include the activation of the body's sensory receptors (i.e., baroreceptors, metaboreceptors, mechanoreceptors, etc.) as well as phenotypic factors such as age, sex, acclimation, fitness, and chronic diseases (e.g., diabetes). The influence of these factors extends into recovery such that marked impairments in thermoregulatory function occur, leading to prolonged and sustained elevations in body core temperature. Irrespective of the level of hyperthermia, there is a time-dependent suppression of the body's physiological ability to dissipate heat. This delay in the restoration of postexercise thermoregulation has been associated with disturbances in cardiovascular function which manifest most commonly as postexercise hypotension. This review examines the current knowledge regarding the restoration of thermoregulation postexercise. In addition, the factors that are thought to accelerate or delay the return of body core temperature to resting levels are highlighted with a particular emphasis on strategies to manage heat stress in athletic and/or occupational settings.

heat stress; postexercise; recovery; heat loss; cooling

OVER PAST DECADES there has been extensive work examining the human thermoregulatory response during exercise. However, the evaluation of the physiology of temperature regulation in the recovery period has received relatively less attention. Yet, this period is defined by a unique biological phenomenon, termed the postexercise disturbance in thermal homeostasis, wherein a prolonged state of hyperthermia is observed as a consequence of a time-dependent attenuation in the body's ability to lose heat. Our understanding of the complex mechanism(s) governing postexercise temperature control has advanced extensively in the past decade. Research into this postexercise phenomenon reveals that restoration of thermoregulation following exercise involves a complex interplay of physiological systems and pathways that include the influence of both thermal and nonthermal inputs (those factors which are thought to modify our body's physiological capacity to dissipate heat which can occur independent of changes in the thermal state of the individual). In this brief review, we explore the current evidence available regarding the mechanism(s) underpinning the postexercise disturbance on thermoregulatory function and how different nonthermal factors affect the restoration of thermoregulatory function following exercise.

Thermoregulation During Exercise

Everyday activities require a functioning thermoregulatory system that will activate and suppress the heat-dissipating mechanisms to minimize changes in body core temperature. Changes in temperature can be perceived at various levels of body tissue with the most critical areas being the skin, muscle, and core (i.e., of the rectal, visceral, and esophageal) tissues. During exercise, the increase in metabolic heat production augments the rate at which heat must be dissipated to the environment to prevent dangerous elevations in tissue temperature. Increases in heat loss via cutaneous vasodilation and sweating induced by the activation of the autonomic nervous system facilitate the increases in dry heat exchange (primarily convection and radiation) and evaporative heat loss, respectively. When exercise is performed in the heat, the additional heat gained from the environment must be compensated through a commensurate increase in the rate of heat dissipation. During exercise, the body stores heat as a result of an imbalance between the rate of heat gain and heat loss. Under compensable conditions, heat balance is achieved within

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30–45 min of steady-state exercise, with the greatest rate of body heat storage, and therefore increase in core temperature, occurring in the first 15–20 min (46) (Fig. 1). During the performance of intermittent exercise, a greater increase in evaporative heat loss occurs following the initial exercise such that the amount of heat stored in the successive exercise bouts is reduced by as much as 40–60% under compensable conditions (26, 42, 43, 58, 60). This has been associated with either a priming effect induced by the progressive increase in body core temperature (42) and/or enhanced peripheral and/or central adaptations of thermoeffector activity (25).

There are numerous factors which can enhance or limit the capacity for heat loss of a particular individual during exercise including ambient conditions and clothing along with hydration status, advanced age, reduced fitness, the presence of chronic diseases, and others (46). However, as discussed below, although these conditions may alter the body's capacity to dissipate heat during an exercise-induced heat stress, differences may only be evidenced above a given heat load threshold (i.e., exercise intensity) (88, 89). Moreover, their influence may differ in the postexercise period. As such, it is also important to consider their separate effects in the restoration of postexercise core temperature.

Postexercise Thermoregulatory Control

Upon the cessation of exercise, there is an abrupt, centrally mediated suppression in the heat loss responses which occurs as the rate of metabolic heat production also returns to baseline levels. However, as recent studies demonstrate, this leads to a sustained elevation of muscle and core temperatures above resting levels for as long as 90 min postexercise (42, 47, 55) (Fig. 1, Tables 1 and 2), a response which was observed in a number of earlier studies but not directly addressed (7, 39, 80). Importantly, studies have noted that the heat is not stored uniformly throughout the body core (34) and that a large portion of this extra heat is stored in the active (1, 8, 42, 55, 80) as well as the inactive musculature (42, 55). This postexercise disruption in thermal homeostasis occurs irrespective of the intensity and/or ambient temperature conditions under which

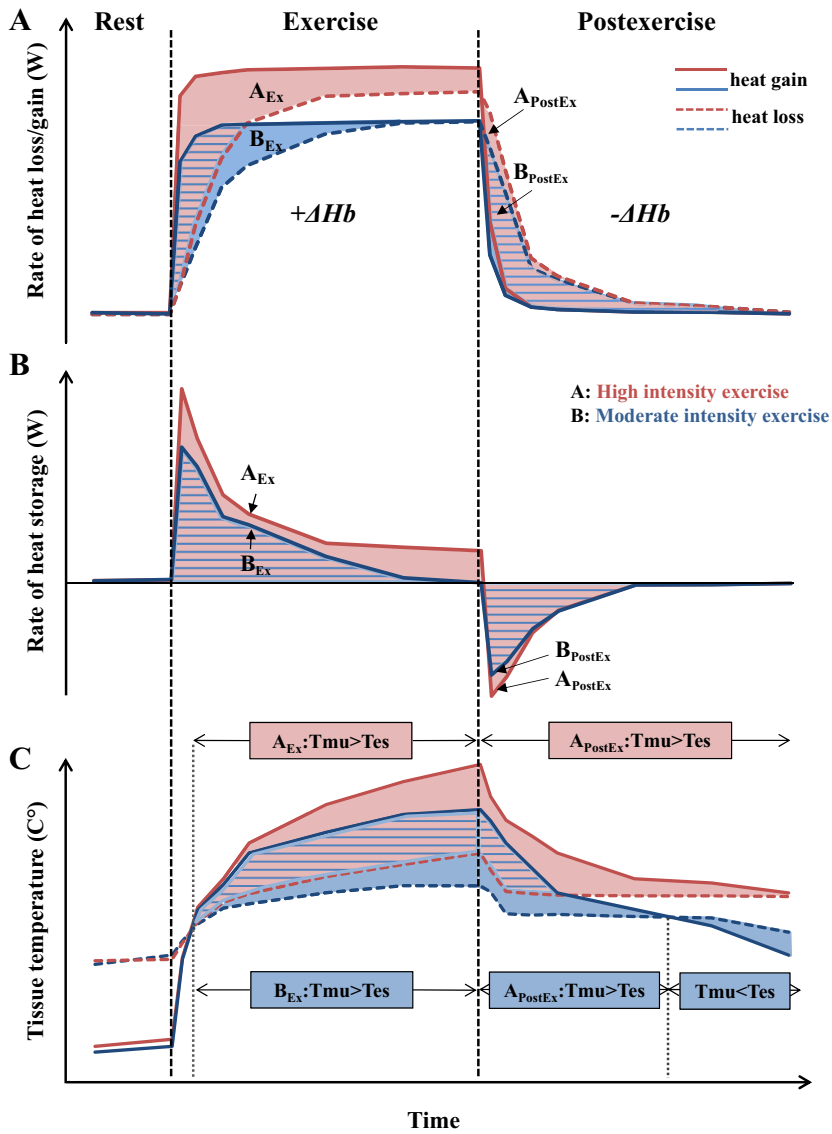

Fig. 1. Schematic figure of the changes in whole body heat exchange as defined by the rate of heat production (top panel *A*; solid line), heat loss (top panel *A*, dashed line) and rate of heat storage (middle panel *B*) as well as core (as defined by esophageal temperature, Tes; dashed lines in panel *C*,) and muscle (Tmu; solid lines in panel *C*) temperatures during (Ex) and following (PostEx) a high (A, red)- and moderate (B, blue)-intensity dynamic exercise bout. Note: shaded area in *A* and *B* represents the amount of heat stored with overlapping areas defined by colored lines. Shaded area in *C* depicts the difference between core and muscle temperatures. ΔHb refers to the change in body heat content.

Table 1. *Esophageal and rectal temperature responses during and following exercise from select studies using varying exercise modes, intensities, duration, and ambient conditions*

Study, (Ref. No.)	Conditions		Core Temperatures, °C				
			Resting	End Ex	Rec 15	Rec 30	End Rec
(7)	Ex: 24°C, 49% RH, cycling at 50% $\dot{V}O_{2max}$ for 45 min; Rec: 24°C, 49% RH (60 min)	ΔTeso		+0.7	+0.4	+0.3	+0.2
(24)	Ex: 42°C, treadmill running at 70% $\dot{V}O_{2max}$ for ~30 min; Rec: 30°C (60 min)	Teso	36.8	39.5	38.0	37.7	37.4
		ΔTeso		+2.7	+1.2	+0.9	+0.6
(26)	Ex: 35°C, 20% RH, cycling at 130 external W (60 min); Rec: 35°C, 20% RH (60 min)	Teso	36.7	37.8	37.4	37.4	37.2
		Trect	37.0	38.3	37.8	37.5	37.4
		ΔTeso/Trect		+1.1/1.3	+0.7/0.8	+0.7/0.5	+0.4/0.4
(28)	Ex: 42°C, 20% RH, cycling at 120 external W for 120 min; Rec: 42°C, 20% RH (90 min)	Teso	36.8	38.8	38.2	38.0	38.0
		ΔTeso		+2.0	+1.4	+1.2	+1.2
(40)	Ex: 22°C, cycling at 70% $\dot{V}O_{2max}$ for 15 min; Rec: 22°C (60 min)	ΔTeso		+1.1	+0.6	+0.4	+0.2
(44)	Ex: 20°C, 50% RH, treadmill running at 45% $\dot{V}O_{2max}$ for 18 min; Rec: 20°C, 50% RH (20 min)	Teso	36.7	37.4	37.2		37.2
		Trect	37.1	37.4	37.4		37.4
		ΔTeso/Trect		+0.7/0.3	+0.5/0.3		+0.5/0.3
(44)	Ex: 24°C, 50% RH, treadmill running at 75% $\dot{V}O_{2max}$ for 18 min; Rec: 24°C, 50% RH (20 min)	Teso	36.6	37.7	37.3		37.3
		Trect	37.1	37.8	38.0		37.9
		ΔTeso/Trect		+1.1/0.7	+0.7/0.9		+0.7/0.8
(44)	Ex: 29°C, 50% RH, treadmill running at 75% $\dot{V}O_{2max}$ (18 min); Rec: 29°C, 50% RH (20 min)	Teso	36.7	38.2	37.4		37.3
		Trect	37.1	38.3	38.2		37.9
		ΔTeso/Trect		+1.5/1.2	+0.7/1.1		+0.6/0.8
(47)	Ex: 25°C; bilateral knee extensions at 60% $\dot{V}O_{2sp}$ for 15 min; Rec: 25°C (90 min)	Teso	36.8	37.3	37.2	37.2	37.0
		ΔTeso		+0.5	+0.4	+0.4	+0.2
(47)	Ex: 25°C; bilateral knee extensions at 80% $\dot{V}O_{2sp}$ for 15 min; Rec: 25°C 90 min)	Teso	36.8	37.6	37.4	37.3	37.3
		ΔTeso		+0.8	+0.6	+0.5	+0.5
(55)	Ex: 30°C, 30% RH, cycling at 70 external watts for 60 min; Rec: 30°C, 30% RH (60 min)	ΔTeso/Trect		+0.8/0.7	+0.4/0.6	+0.3/0.4	+0.2/0.2
(65)	Ex: 35°C, 20% RH, cycling at 85% $\dot{V}O_{2max}$ for 15 min; Rec: 35°C, 20% RH (45 min)	Teso	36.9	38.4	37.6	37.5	37.5
		ΔTeso		+1.5	+0.7	+0.6	+0.6
(1)	Ex: 24°C, 55% RH, cycling at 900 kpm/min for 30 min; Rec: 24°C, 55% RH (60 min)	Teso	37.0	37.7	37.4	37.2	37.0
		Trect	36.9	37.8	37.6	37.2	36.9
		ΔTeso/Trect		+0.7/0.9	+0.4/0.7	+0.2/0.3	0/0
(1)	Ex: 24°C, 55% RH, cycling at 1,200 kpm/min for 30 min; Rec: 24°C, 55% RH (60 min)	Teso	37.0	38.4	37.6	37.3	37.0
		Trect	37.1	38.6	38.0	37.6	37.3
		ΔTeso/Trect		+1.4/1.5	+0.6/1.0	+0.3/0.5	+0/0.2
(1)	Ex: 24°C, 55% RH, cycling at 1,500 kpm/min for 30 min; Rec: 24°C, 55% RH (60 min)	Teso	37.0	39.0	37.6	37.3	37.0
		Trect	37.0	39.0	38.3	37.8	37.4
		ΔTeso/Trect		+2.0/2.0	+0.6/1.3	+0.3/0.8	+0/0.4

Resting, baseline values; Ex, exercise; Rec, recovery; End Ex, values at the end of exercise; Rec 15 and Rec 30, recovery values at the specific time-point during recovery (15 and 30 min into recovery, respectively); RH, relative humidity; $\dot{V}O_{2max}$, maximal aerobic capacity; $\dot{V}O_{2sp}$, maximal aerobic capacity during bilateral knee extensions; Teso, esophageal temperature; Trect, rectal temperature; ΔTeso/Trect, the change in esophageal/rectal temperature relative to baseline levels. Note: for some studies multiple conditions were studied, and these are presented separately.

Table 2. *Muscle temperature responses during and following exercise from select studies using varying exercise modes, intensities, duration, and ambient conditions*

Study, (Ref. No.)	Conditions		Muscle Temperatures, °C				
			Resting	End-Ex	Rec 15	Rec 30	End Rec
(45)	Ex: 25°C, concentric knee extension at 60% $\dot{V}O_{2sp}$ (15 min); Rec: 25°C (90 min)	ΔV.Med		+2.5	+1.8	+1.6	+1.1
		ΔTri.Brach		+1.3	+0.8	+0.7	+0.3
(55)	Ex: 30°C, 30% RH, cycling at 70 external W for 60 min; Rec: 30°C, 30% RH (60 min)	ΔV.Lat		+2.4	+2.0	+1.5	+1.1
		ΔTri.Brach		+2.2	+1.7	+1.5	+0.9
(47)	Ex: 25°C, concentric knee extension at 60+80% $\dot{V}O_{2sp}$ (15 min); Rec: 25°C (90 min)	ΔV.Lat (MI)		+2.5	+1.5	+1.3	+0.9
		ΔV.Lat (HI)		+3.2	+2.2	+2.0	+1.4
(1)	Ex: 24°C, 55% RH, cycling at 900 kpm/min for 30 min; Rec: 24°C, 55% RH (60 min)	Quad	33.8	37.8	37.0	36.0	35.0
		ΔQuad		+4.0	+3.2	+2.2	+1.2
(1)	Ex: 24°C, 55% RH, cycling at 1,200 kpm/min for 30 min; Rec: 24°C, 55% RH (60 min)	Quad	33.8	38.3	37.0	36.0	35.0
		ΔQuad		+4.5	+3.2	+2.2	+1.2
(1)	Ex: 24°C, 55% RH; cycling at 1,500 kpm/min for 30 min; Rec: 24°C, 55% RH (60 min)	Quad	35.0	39.8	38.0	37.2	36.1
		ΔQuad		+4.8	+3.0	+2.2	+1.1

Resting, baseline values; Ex, exercise; Rec, recovery; End Ex, values at the end of exercise; Rec 15 and Rec 30, recovery values at the specific time-point during recovery (15 and 30 min into recovery, respectively); RH, relative humidity; ΔV.Med, change in vastus medialis muscle temperature relative to baseline levels; ΔTri.Brach, changes in triceps brachii muscle temperature relative to baseline levels. ΔV.Lat (MI), changes in vastus lateralis muscle temperature with moderate-intensity exercise relative to baseline levels; ΔV.Lat (HI), changes in vastus lateralis muscle temperature with high-intensity exercise relative to baseline levels; Quad, quadriceps muscle temperature; ΔQuad; change in quadriceps muscle temperature relative to baseline levels. Note: for some studies multiple conditions were studied, and these were presented separately.

exercise is performed (44), albeit these factors will influence the magnitude of the elevation in tissue temperatures and the time required for recovery (Fig. 1). For example, moderate-intensity exercise (70% of maximal aerobic capacity) resulted in a sustained elevated postexercise esophageal temperature of 0.4°C above baseline compared with 0.8°C after higher-intensity exercise (93% of maximal aerobic capacity) (50). Calorimetry-based studies [e.g., Snellen direct calorimeter, a device for making extremely accurate measurements about the heat emitted by the human body (46)] demonstrate that as a consequence of this impairment, only 30–50% of the heat gained during exercise is lost during a recovery of similar duration (based on 1:1 work to rest ratio) (42, 55) (Fig. 2). Moreover, although the amount of heat stored during a successive exercise bout is reduced relative to the initial exercise bouts, this is not paralleled by a concomitant increase in heat loss during recovery (26, 42, 43, 58, 60). Rather, whole body heat loss and thus restoration of body core temperature remains similarly compromised as in the first recovery bout even despite progressively greater increases in body heat storage and therefore core temperature with each successive exercise/recovery cycle.

A link to blood pressure regulation. The alteration in postexercise thermoregulation function has been associated with altered baroreflex activity, which also contributes to systemic vasodilation and a pooling of blood in the extremities resulting in reductions in mean arterial pressure (MAP) (31). We will discuss this relationship briefly, although the reader is referred to a recent review for a more comprehensive description (48). The temporal association between the restoration of blood pressure and core temperature to preexercise levels (each within ~2 h) has led to extensive examination of a relationship between blood pressure and core temperature regulation. It has been shown that increases in baroreceptor unloading associated with greater reductions in MAP brought about, for example, by performing higher-intensity exercise result in a more pronounced suppression of skin blood flow (SkBF) and sweating paralleled by a higher core temperature (47, 51, 52) and therefore a longer recovery time. However, reductions in MAP as small as 4 mmHg from resting levels, such as occurs following light-intensity exercise (55% of maximal aerobic capacity), have also been shown to lead to a delay in the restoration of core temperature to resting levels (47, 51, 52).

In the face of a sustained impairment in heat loss responses of SkBF and sweating, the restoration of postexercise core temperature is ultimately dependent upon the magnitude of the residual heat in the previously active musculature (Fig. 1). During dynamic exercise, a significant amount of heat is stored in the active muscle with muscle temperatures reaching values 2–3°C (dependent on exercise duration and intensity as well as ambient conditions) above core temperature (47). However, the pooling of blood in the muscles of the lower limbs postexercise reduces compartmental heat exchange such that heat content of the previously active musculature remains elevated (42, 47, 55) (Fig. 1). Given that the circulatory system is a significant avenue of heat transfer between the body compartments (as defined by the convective exchange of heat between muscle and blood, and blood to the central body core), in the absence of an increase in whole body heat dissipation (i.e., a reversal of

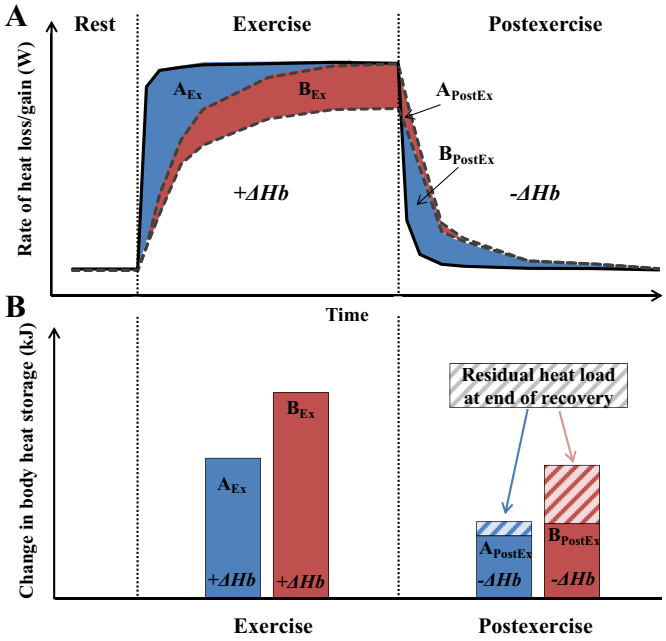

Fig. 2. Schematic illustrating the influence of nonthermal intrinsic factors on the time-dependent changes in the rate of whole body heat loss (dashed line) for a fixed rate of heat production (black solid line). Impairments in heat loss caused by aging, reduced fitness or acclimation, chronic health conditions (e.g., diabetes), etc., result in greater body heat storage during exercise [defined by the area under the curve; $A_{Ex} + B_{Ex}$, panel *A*; and larger bar (B_{Ex}) in panel *B*]. Despite an elevated thermal drive in recovery (of similar duration to exercise), only ~30–50% of the heat gained during exercise is lost, a consequence of the strong nonthermal suppression of postexercise heat loss. As a result, a similar reduction in heat storage (ΔHb) is measured between the two groups (panel *B*, solid section of the bar). However, as a result of the higher amount of heat stored during exercise, individuals with impaired heat loss retain a greater residual heat load (dashed section of the bar) at the end of recovery.

the postexercise attenuation of heat loss), core temperature will remain elevated as long as the heat content of muscle remains higher or equal to that of core which by extension is also dependent on the duration of the postexercise hypotensive effect (47) (Tables 1 and 2).

Conditions which induce greater metabolic and/or environmental heat loads such as higher-intensity exercise and/or exercise performed in the heat and which generate greater increases in muscle heat content and core temperature at end exercise (Fig. 1), can translate into significant postexercise cardiovascular (reductions in blood pressure as great as 20 mmHg) (28) and thermal strain [elevated body core temperatures (>38°C)] (68, 69). The consequence of these responses in the context of an individual's ability to recover from an exercise-induced heat stress can be worsened following the performance of prolonged and/or intense exercise especially in hot environments (68, 69). In situations whereby elevated sweat rates induce a state of dehydration (such as occurs with prolonged exercise especially in the heat and/or when protective clothing is worn), cardiovascular stability can be further compromised exacerbating an already compromised thermoregulatory system (28, 29, 68). Moreover, progressive dehydration associated with exercise can lead to serious complications such as syncope and electrolyte imbalances, with even mild dehydration (~2–4% decrease in body weight) causing impairments in heat loss (29, 81). Under these conditions, whereby changes in SkBF and sweating cannot facilitate a sufficiently high rate of heat loss to offset the large heat gains from the prior exercise bout(s), precipitous increases in core temperature will occur even if rest periods are interspersed between exercise/work bouts as discussed below (69, 86).

In an experimental setting, the negative impact of this postexercise suppression of heat loss can be observed by the rapid reversal of the individual's hyperthermic state when postural (i.e., head-down tilt) (67) or mechanical (application of positive pressure to the lower limbs, passive leg compressions) (43) interventions are employed to reverse the postexercise hypotension (Table 3). When performed, these manipulations have been shown to reverse the attenuation of SkBF and sweating and enhance the compartmental transfer of heat through the translocation of the blood pooled in the previously hot active muscles to the body core region (43, 67). Baroreceptor loading can lead to lower core temperatures by ~0.5°C within 20 min of reversing the postexercise hypotension (65) secondary to increased SkBF (40, 65) and sweating (24, 37, 40). Importantly, the response is dependent upon the level of hyperthermia such that a greater impact on SkBF is observed at higher core temperatures whereas the influence on sweating is most evident at lower levels of hyperthermia (24). Although some studies reported no improvements in sweating (65, 74, 75), other nonthermal factors were shown to enhance the sweating response (i.e., metabo- and osmoreceptors) (74, 75). Ultimately, the increase in SkBF and sweating associated with the reversal of postexercise hypotension enhances whole body heat dissipation facilitating a more rapid recovery of core temperature.

With an extended natural recovery, the restoration of normal thermoregulatory function can occur as the postexercise hypotension naturally resolves. Following moderate-intensity exercise (15 min of running at 70% of maximal aerobic capacity), this was observed within ~90 min following cessation of exercise (49). Thus thermoregulatory restoration after exercise may take 4–5 times longer than the duration of the exercise bout and is dependent upon the restoration of baseline mean arterial pressure. However, under conditions in which an individual may experience a very high state of hyperthermia, delaying the management of an individual's elevated state of hyperthermia may lead to deleterious consequences on physiological function (i.e., heat-induced syncope or collapse exacerbated by the postexercise hypotension, other heat-related injuries, etc.). Irrespective of the nature of the physical effort that caused the elevated state of hyperthermia, interventions which enhance whole body heat dissipation (including postural adjustments such as lying supine or lower leg compression) should be employed to accelerate restoration of normal thermoregulatory function.

Nonthermal Modulation of Postexercise Heat Loss

Influence of metaboreceptors and osmoreceptors. Although the disruption in postexercise thermoregulatory control has been largely ascribed to a nonthermal baroreceptor-mediated influence associated with postexercise hypotension, other nonthermal factors associated with metabo- and osmoreceptor activation have been shown to modulate the restoration of thermoregulatory control following exercise. During a bout of exercise, metabolites are released in large amounts which can

Table 3. *Esophageal and rectal temperature responses during and following exercise from select studies employing different recovery modes and/or recovery interventions*

Study, (Ref. No.)	Conditions		Core Temperatures, °C				
			Resting	End Ex	Rec 15	Rec 30	End Rec
(24)	Ex: 42°C, treadmill running at 70% $\dot{V}O_{2max}$ for ~30 min; Rec: Inactive, 30°C (60 min)	Teso	36.7	39.5	38.2	37.7	37.5
		ΔTeso		+2.8	+1.5	+1.0	+0.8
(24)	Ex: 42°C, treadmill running at 70% $\dot{V}O_{2max}$ for ~30 min; Rec: Passive, 30°C (60 min)	Teso	36.7	39.5	38.0	37.4	37.2
		ΔTeso		+2.8	+1.3	+0.7	+0.5
(24)	Ex: 42°C; treadmill running at 70% $\dot{V}O_{2max}$ for ~30 min; Rec: Active, 30°C (60 min)	Teso	36.7	39.5	38.0	37.5	37.3
		ΔTeso		+2.8	+1.3	+0.8	+0.6
(40)	Ex: 22°C, cycling at 70% $\dot{V}O_{2max}$ for 15 min; Rec: no pressure, 22°C (60 min)	ΔTeso		+1.2	+0.6	+0.4	+0.2
(40)	Ex: 22°C, cycling at 70% $\dot{V}O_{2max}$ for 15 min; Rec: LBPP, 22°C (60 min)	ΔTeso		+1.2	+0.6	+0.1	−0.3
(65)	Ex: 35°C, 20% RH, cycling at 85% $\dot{V}O_{2max}$ for 15 min; Rec: no pressure 35°C (45 min)	Teso	36.9	38.4	37.6	37.5	37.5
		ΔTeso		+1.5	+0.7	+0.6	+0.6
(65)	Ex: 35°C, 20% RH, cycling at 85% $\dot{V}O_{2max}$ for 15 min; Rec: LBPP, 35°C (45 min)	Teso	36.9	38.4	37.6	37.3	37.1
		ΔTeso		+1.5	+0.7	+0.4	+0.2
(67)	Ex: 24°C, 45% RH, cycling at 75% $\dot{V}O_{2max}$ for 15 min; Rec: upright seated, 24°C (60 min)	Teso	36.8	38.1	37.4	37.4	37.1
		ΔTeso		+1.3	+0.6	+0.6	+0.3
(67)	Ex: 24°C, 45% RH; cycling at 75% $\dot{V}O_{2max}$ for 15 min; Rec: head-down tilt, 24°C (60 min)	Teso	36.8	38.1	37.1	37.0	36.8
		ΔTeso		+1.3	+0.3	+0.2	0.0

Resting, baseline values; End Ex, values at the end of exercise; Rec 15 and Rec 30, recovery values at the specific time-point during recovery (15 and 30 min into recovery, respectively); RH, relative humidity; $\dot{V}O_{2max}$, maximal aerobic capacity; Teso, esophageal temperature; ΔTeso, the change in esophageal temperature relative to baseline levels; Inactive, resting recovery. Passive, legs taken passively through the full range of motion to mimic cycling; Active, loadless pedaling; LBPP, lower-body positive pressure applied starting at 15 min of recovery; head-down tilt, subjects laying supine tilted with the head down at 15° from the horizontal for the entire recovery period. Note: for some studies multiple conditions were studied, and these are presented separately.

become concentrated within the active muscle. This produces an activation of the metaboreceptors which increases blood pressure to better perfuse the tissues and dilute the concentration of metabolites. Although metaboreceptors have been well studied during both passive heat stress and exercise, only recently were they examined during the postexercise period (5a, 11a, 81a). The activation of metaboreceptors induced by the occlusion of the upper arm following a brief 1-min isometric handgrip exercise, performed at 20 min into recovery following high-intensity running, delayed the reduction of sweating toward baseline levels relative to a control condition with no forearm occlusion (66). Curiously, it was also reported in this study that a metaboreflex response was induced with forearm occlusion without prior isometric handgrip exercise. Therefore, it seems likely that high-intensity exercise has substantial leftover metabolites that remained in a concentration sufficient to activate the metaboreflex and transiently improve heat loss in recovery.

As occurs during prolonged exercise, particularly in the heat, high levels of sweat output can lead to dehydration which typically is reported as a decrease in blood volume (hypovolemia) and increase in blood osmolality (hyperosmolality) (29, 81). Hyperosmolality induced by hypertonic saline infusion has consistently been shown to induce a pronounced modulation of SkBF during heat stress (3, 62); however, exercise-induced hyperosmolality ranging from ~8 to 13 mosmol/kg of solvent relative to preexercise levels was recently shown to have no effect on postexercise SkBF (74). Of note, it is possible that the impact of hyperosmolality on SkBF is not a graded response per se, but rather the threshold level for osmoreceptor-mediated control of SkBF was surpassed in each of the study's conditions. It is generally accepted that hyperosmolality has a perturbing effect on the sweating response during passive or exercise-induced heat stress such that the central onset threshold for heat loss is increased (3, 62). This is a result of the body working to minimize fluid losses. Moreover, a recent study demonstrated that hyperosmolality induced an attenuation of postexercise sweating, albeit with regional differences such that areas of high sweat output (i.e., the upper back) showed the greatest decrease in sweating compared with the forearm (74). These findings demonstrate that the maintenance of hydration status with respect to euvolemia and isos-

molality is important in ensuring the restoration of normal thermoregulation following an exercise-induced heat stress. In particular, in a state of osmotic stress or dehydration, the restoration of thermoregulation can be delayed which would be worsened with dehydration associated with additional exercise bouts.

Postexercise alteration in autonomic nervous system function. Exercise-induced hyperthermia can elicit marked impairments in autonomic nervous system (ANS) function as assessed by both heart rate variability (HRV, the time difference between repeated heart beats) and baroreflex sensitivity (BRS, the amount of change in heart rate attributable to changes in systolic blood pressure), which persist for a prolonged period following cessation of exercise. Specifically, exercise and increased core temperature both independently alter the ratio of sympathetic to parasympathetic activity (2, 19). Even in highly endurance-trained individuals the perturbation to ANS function can last >80 min following an exercise-induced heat stress, although functional parameters returned to baseline levels within 24 h (2). In support of these findings, a recent report showed that recovery of HRV, and therefore ANS function, was temporally associated with the restoration of core temperature to preexercise levels (19). Importantly, these findings were verified such that even when participants were cooled rapidly by immersion in 2°C water (~6–11 min for rapid cooling vs. 54–76 min for natural recovery) following an exercise-induced heat stress (eliciting an end-exercise core temperature of ≥40°C), the recovery of HRV to preexercise levels coincided with the restoration of baseline core temperature values (19). In addition, cold water immersion following exercise has been shown to have greater benefit to postexercise cardiac autonomic regulation when compared with both active and resting recovery modes (4).

Influence of the Phenotypic Profile on Postexercise Heat Loss

In parallel to the influence of sensory receptor activation, nonthermal factors associated with the individual's phenotypic profile such as sex, age, fitness, chronic disease, acclimation status, and others can play an unequivocal role in the regulation of thermoregulatory function during and following exercise. Although there have been extensive studies examining the influence of these factors on thermoregulatory function during exercise, our understanding of their effects on the restoration of thermoregulatory function following exercise remains limited. The following section briefly examines how these factors interplay to modulate the recovery of body core temperature postexercise.

Sex- and age-related effects. Sex-related differences in thermoregulatory function have been largely ascribed to differences in physical characteristics and aerobic fitness (32). However, recent evidence indicates that females demonstrate a reduced whole body sweating response during exercise, a response which extends beyond differences in physical characteristics (27) and which may be attributed to peripheral modulation of sweat gland function (23). These sex-related differences have been shown to extend into the postexercise recovery period such that females demonstrated a more rapid attenuation of heat loss responses paralleled by a more enduring postexercise elevation in muscle and core temperatures (45). This is thought to be related to changes in baroreceptor sensitivity in the postexercise period which manifest as a greater level of postexercise hypotension in females (45). This ultimately leads to a longer time required for females to restore thermoregulatory control following a bout of exercise.

Aging is another key modulator of the body's physiological capacity to dissipate heat with adults as young as 40 yr of age demonstrating marked impairments in heat loss (57). In addition, the impact of age may be more apparent during the exercise bout in women compared with the postexercise period (13). These impairments in heat loss are evidenced even in the very early stages of exercise (i.e., first 10 min) (58, 60). In terms of the restoration of core temperature, these age-related differences have recently been shown to further extend the time required to reestablish normal core temperature. However, this is due solely to the greater amount of heat stored during exercise and not a more rapid and/or pronounced suppression of heat loss in the recovery period (57, 59, 60). For example, a recent study showed that older adults (60–70 yr) stored ~63% more heat than their younger (20–30 yr) counterparts over the course of four successive 15-min moderate-intensity exercise bouts (each separated by 15 min recovery). Despite this, the older group exhibited a similar rapid decay in whole body heat loss (60). Moreover, the postexercise suppression of heat loss responses remained intact with the successive exercise bouts despite a progressively greater residual heat load measured at end exercise; a heat load which was substantially more elevated in the older adults (60).

Aerobic fitness and training. Regular aerobic training improves thermoregulatory function, leading to improvements in heat dissipation during exercise (5, 22, 88), a response which recently has been shown to result in a correspondingly greater rate of core temperature recovery (88). For example, in middle-aged adults, higher levels of aerobic fitness (as defined by aerobic capacity, $\dot{V}O_{2peak}$) were associated with an enhanced whole body sweating response during exercise and paralleled by a concomitant reduction in body heat storage and therefore body core temperature. However, this was not paralleled by improvements in the rate of heat dissipation during recovery which further highlights the strong influence of nonthermal intrinsic factors in the modulation of postexercise thermoregulation. Indeed, restoration of body core temperature was more rapid in the adults with higher fitness due only to an enhanced capacity to dissipate heat during exercise (88).

In contrast to long-term endurance training, the influence of short-term exercise training on thermoregulatory function has yielded mixed findings with some studies reporting greater heat loss responses of SkBF and sweating, paralleled by reductions in core temperature (35, 79), whereas others reported no effect (83, 85). One study reported comparable improvements in local heat loss (i.e., earlier onset threshold for SkBF and sweating) and core temperature (lower resting and end-exercise values) responses following an 8-wk exercise training program, although this was not paralleled by improvements in whole body heat loss during and following moderate-intensity exercise in warm ambient conditions (87). However, as recently reported the lack of an improvement in the body's ability to dissipate heat should be interpreted with caution as differences may only be evident beyond a given heat load (88).

Heat acclimation. Heat acclimation provides an important thermoregulatory advantage to those individuals, who by the

nature of the activity they perform, are exposed to high heat stress conditions. Heat acclimation enhances heat dissipation (i.e., through increases in SkBF and sweating), reduces core and skin temperatures, and enhances fluid balance and cardiovascular control during exercise in the heat (61, 72, 84). However, the improvements in thermoregulatory function are not similarly observed in recovery (76). Interestingly, this pattern of response closely parallels that observed with age- and fitness-related differences in thermoregulation during and following exercise as discussed above. Although a recent study (76) showed an 11% increase in whole body heat loss at very high heat loads (400 W/m^2, which was paralleled by a 34% reduction in body heat storage) following 14 days of dry heat acclimation, the postexercise attenuation in heat loss remained intact with values similar to preacclimation levels. As a consequence, the change in body heat storage during the recovery phase was comparable between the pre- and postacclimation period. It is important to note, however, that this study was designed specifically to examine improvements in the body's physiological capacity to dissipate heat during exercise. Thus further research is required to advance our understanding of the potential role that heat acclimation may play in mediating the influence of nonthermal intrinsic factors on the restoration of postexercise heat loss responses and body core temperature. Regardless, these early findings once again highlight the strong suppressive influence that the nonthermal factors elicit in the regulation of postexercise body core temperature and which occur irrespective of an individual's level of heat acclimation. In this context, although a heat-acclimated person may have the ability to perform in hotter conditions due to their enhanced ability to dissipate heat, it is important to consider the fact that restoration of core temperature will be similarly impacted by the sustained suppression of heat loss responses.

Diabetes mellitus. Chronic diseases which are associated with aging such as Type 2 diabetes have been shown to exacerbate the age-related attenuation in heat loss during exercise, although the same level of impairment in thermoregulatory function in the postexercise period remains (54). Therefore, individuals with diabetes exhibit a greater level of hyperthermia at the end of exercise and a similar rate of postexercise heat loss as their healthy counterparts, which translates into a prolonged delay in the reestablishment of thermoregulatory function. Even young habitually active adults with Type 1 diabetes experience a similar pattern of response (64). Readers are referred to a recent detailed review on this topic (53).

The influence of phenotypic factors on heat dissipation: when do they matter? Although these aforementioned phenotypic factors have been shown to play an important role in modulating thermoregulatory function and therefore the restoration of body core temperature following exercise, their effects may not always be present. A growing number of reports show that differences may only be evident above a specific heat load threshold defined by the sum of exercise (metabolic) and environmental heat load. For example, age-related impairments in heat loss are initially evident in males at a heat load of $\geq$400 W (88) whereas for females this threshold is reduced (i.e., $\geq$300 W) (89). These heat loads are approximately equivalent to performing either low-intensity intermittent exercise in hot conditions or moderate-intensity exercise in warm conditions. As such, age-related differences in thermoregulatory function resulting in a delayed restoration of core temperature recovery would only be evident above these thresholds (Fig. 2). Below this threshold, young to older adults (18 to 70 yr) with comparable levels of fitness would demonstrate a similar thermoregulatory capacity and therefore similar pattern of response during and following exercise (88, 89).

As for the age-related impairments described above, fitness-related differences are also heat load dependent (88). A recent report showed that relative to their age-matched untrained counterparts, middle-aged endurance trained males exhibited a comparable capacity to dissipate heat during low-intensity exercise in the heat (an equivalent heat load of 300 W) which was paralleled by a similar time-dependent restoration of thermoregulatory function (as defined by body core temperature response). However, marked differences were observed above this exercise condition, with the magnitude of difference becoming more pronounced with increasing heat loads (89). Taken together, these relatively new observations clearly demonstrate that the lack of an observed difference between groups or individuals should not necessarily lead one to conclude that differences do not exist. Rather, the physiological stimuli employed (in this case the heat load) must be sufficiently elevated such that they exceed the body's ability to adequately respond to the thermal stress. This is an important consideration not only in our understanding of thermoregulation during exercise, but also as it relates to the restoration of body core temperature following exercise.

Managing the Risk of Heat Strain

The use of work/rest allocations. The recovery from exercise represents an important period in athletic, occupational, and military-relevant activities. In this context, the impact of altered thermoregulatory function has broad implications in how the risk of heat strain is managed. As outlined above, the manner in which exercise (i.e., the allocation of work/rest cycles) is performed can dramatically influence heat distribution within the body and thereby the recovery of core temperature. So long as the total time for both exercise and rest is the same, the cumulative increase in body heat content will be the same (26). However, reducing or lengthening the recovery period can have important consequences on the amount of heat that is stored in the body, thereby affecting the restoration of muscle and core temperatures following exercise (Fig. 1). Ultimately, this can influence performance and/or work capacity (15, 90).

The use of intermittent exercise/work protocols [e.g., the American Conference of Governmental and Industrial Hygiene's (ACGIH) Threshold Limit Values (TLV)] are recommended to protect individuals against dangerous increases in core temperature during exercise/physical work performed in adverse (hot and/or humid) environmental conditions. The benefit of exercise-recovery cycles is that it allows for a decrease in the time-weighted average rate of metabolic heat production for a given task. When combined with a sufficient recovery that is adjusted for environmental conditions, well-hydrated individuals should be able to maintain work performance in hot environments whereby stable core temperatures are achieved. However, as recent work demonstrates, existing guidelines such as those associated with the TLVs may not adequately protect even young hydrated individuals from dangerous increases in hyperthermia (69). Specifically, it was

shown that moderate-intensity work in hot environments performed within the guidelines of the TLVs led to variable core temperature responses between both participants and conditions. Moreover, the TLVs did not result in stable core temperature levels that would be required to prevent dangerous increases in core temperature during extended work periods. Taken together, these findings solidify the need to better understand the influence of the manner in which recovery periods are imposed during prolonged work under different conditions (i.e., exercise intensity, environmental conditions) as differences in dynamic transfer of heat within the body can impact the time-course recovery of body core temperature.

Hydration state. Dehydration is common among athletes (30) and workers (68) who are engaged in prolonged periods of exercise, and as discussed earlier, is an important factor affecting body core temperature recovery. Dehydration can markedly impair cardiovascular function (29) and lead to more pronounced elevations in core temperature (28) which is particularly emphasized in the heat when the requirements for evaporation are maximized. In the absence of interventions to enhance whole body heat loss (see below), normal resting temperatures are eventually reestablished after an extended period ($\geq$60 min), a response mediated largely through passive heat loss mechanisms (46). Although this may appear to provide some physiological advantage in preserving body fluid balance (due to the postexercise attenuation of sweating) from the perspective of a single exercise bout followed by an extended period of inactive recovery, it likely provides little to no advantage to those individuals who do not have the luxury of extended recovery before their next work bout. An incomplete restoration of their thermoregulatory system leads to progressively greater levels of hyperthermia and a compensatory greater increase in whole body sweating with successive exercise bouts (26, 42), the latter response leading to an enhanced rate of dehydration. Thus, implementing appropriate management strategies aimed at maintaining near normal hydration status can play a pivotal role in maintaining elevated rates of heat dissipation during exercise and facilitating a more rapid restoration of core temperature during recovery (28). Combined, these responses will circumvent potential dangerous increases in core temperature in individuals who must perform prolonged periods of physical activity especially in the heat.

The use of cooling strategies. Recognition of the importance of recovery on performance has generated significant interest in the effectiveness of local or whole body cooling protocols (water immersion, application of ice, cold air exposure, etc.) to return the body to its preexercise resting state. Whereas increasing muscle and efferent nerve temperatures are known to have beneficial Q_{10} effects, leading to improved performance during brief bouts of exercise (i.e., instantaneous power activities such as sprint cycling performance) (16, 17), sustained increases in muscle temperature [such as occurs in the postexercise period (42, 47, 55)] can lead to a decrement in performance and/or earlier onset of fatigue (15, 90). This has in part been attributed to an increased rate of cross-bridge cycling during exercise (12) and/or a decline in central nervous system activation of the exercising muscles especially when exercise is performed in hot humid conditions whereby marked elevations in core temperature can occur (90).

Although local and whole body cooling has been employed to provide thermoregulatory benefits (i.e., reducing body heat storage) when performed before, during, and/or after high-intensity interval exercise (11, 71), it has also been shown to enhance recovery of muscle function following endurance exercise (36), intermittent sprint exercise (78) as well as during and between (i.e., multi-training days) short-term intensive exercise (e.g., resistance exercise) (94). Some studies have reported increased local muscle reflexes, muscle excitability and short-term release of neurotransmitters from the central nervous system, a response attributed to enhanced motor neuron output of the active muscles (33, 73, 91, 92) even when the cooling of the periphery does not involve the active muscle directly (33, 56, 73). Further, reducing muscle temperature in the early stages of recovery can reduce muscle tissue damage (70) including a reduction in muscle edema, pain, and the accumulation of metabolites and improving muscle performance during subsequent submaximal work efforts in resistance exercise training (78), high-intensity interval training (6), and endurance activities (63) which may be of benefit in improving training adaptations and thermoregulatory function over the long term. However, it is important to note that overcooling of the active muscle can also lead to marked reductions in performance (e.g., power output) (11, 12) via a Q_{10} effect of ATP hydrolysis (18). Thus the cooling strategy employed [e.g., type, duration, surface area covered (local vs. whole-body), etc.] must be carefully considered as positive benefits on performance and/or thermoregulatory function may not warrant such an approach.

Currently, cold water immersion ($<$8°C) is the gold standard for rapidly reducing core temperature (10) especially after prolonged endurance activities performed in the heat. By creating such dramatic temperature gradients between the skin and the water, cooling rates as high as 0.35°C/min result (77). In fact, recent evidence indicates that water at 2°C can eliminate the impact of biophysical characteristics (21) as well as a 40-min delay in treatment (20) on core temperature recovery. Ultimately, the use of cold water immersion is optimal for emergency situations with dangerous levels of hyperthermia but may not always be a practical solution due to the necessary equipment. In situations where cold water immersion is not a realistic option to promote the restoration of thermoregulation, there are some other interventions along with simply removing the individual from hot ambient conditions that have been assessed. For instance, it was hypothesized that a passive (i.e., no physical effort, but the limbs are taken through the normal range of motion) or active recovery mode might be more beneficial for heat loss when compared with inactive recovery (Table 3). Some early studies demonstrated this to be effective in elevating the rate of heat loss by increasing sweating and possibly SkBF (9, 41, 82, 93). This was thought to be modulated by a mechano and/or baroreceptor-mediated increase in heat loss; however, Jay and colleagues (38) concluded that the impact on heat loss was entirely explained by the increases in metabolic heat production associated with passive and active recovery modes. A more promising intervention during the postexercise period is the manipulation of blood pooling and thereby blood pressure. Specifically, studies have shown that reversing the postexercise hypotensive response by positive pressure to the lower limbs (43) or head down tilt (40) has led to a more rapid recovery of core temperature secondary to

increased heat loss. These interventions are arguably more practical as they involve simple modifications such as placing compression stockings on the calves or lying the individual supine with their legs raised. Regardless, in situations whereby body core temperature may achieve potentially dangerous levels (i.e., ≥39.5°C), cooling techniques employing water immersion, application of wet towels, etc., that would promote a rapid decay in body core temperature should be employed (10).

Future Directions

Despite our growing knowledge of thermoregulatory responses in the postexercise period, there remains a paucity of information related to the mechanisms by which nonthermal factors modulate heat loss and core temperature responses. Future work is therefore needed to understand the putative transmitters and cotransmitters involved in the mediation of thermoeffector activity. Moreover, much of our limited understanding of the restoration of thermoregulation following exercise relates to relatively short exercise periods. However, athletes, workers, and military personnel are commonly exposed to successive days of exercise/physical work in the heat. Advancing our understanding of the physiological consequences of repeated exposures in the context of fatigue, sleep deprivation/quality, diet, fluid balance, and others on the restoration of thermoregulation is therefore an important avenue of research.

Summary

The regulation of postexercise thermoregulation involves a complex interaction of multiple factors of thermal and nonthermal origin. The impact of altered thermoregulatory function has broad implications in how exercise is performed in athletic, occupational, and military-relevant situations. Attention must be given to understanding how these different factors may affect the restoration of postexercise thermoregulatory function, which would include adjusting exercise-induced heat loads and/or duration of rest periods between exercise bouts to account for differences in postexercise whole body heat loss and therefore core temperatures.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

G.P.K. and R.M. prepared figures; G.P.K. and R.M. drafted manuscript; G.P.K. and R.M. edited and revised manuscript; G.P.K. and R.M. approved final version of manuscript.

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