

Impact of Exercise Intensity on Thermoregulation and Body Temperature During Physical Activity

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Abstract: *Thermoregulation is a vital physiological process that maintains core body temperature within narrow limits during rest and physical activity. Exercise induces increases in metabolic heat production, challenging homeostatic mechanisms. This review examines how varying exercise intensities affect thermoregulation, body temperature dynamics, and physiological responses, specifically focusing on cardiovascular adjustments, sweating, heat dissipation, and implications for performance and safety.*

Keywords: Exercise intensity, thermoregulation, body temperature, core temperature

I. INTRODUCTION

During physical activity, the human body produces heat at rates proportional to exercise intensity (González-Alonso, 2012). Thermoregulation, controlled primarily by the hypothalamus, ensures that core body temperature remains within safe limits (Noble & Robertson, 1996). However, high exercise intensities can overwhelm dissipative mechanisms, leading to heat storage and hyperthermia (Cheung & McLellan, 1998). Thermoregulation is a critical physiological mechanism that allows the human body to maintain its core temperature within a narrow range, ensuring homeostasis during rest and physical activity.

During exercise, the rate of metabolic heat production increases in proportion to exercise intensity, placing substantial demands on the body's thermoregulatory systems (González-Alonso, 2012). The hypothalamus functions as the primary control center for thermoregulation, integrating signals from thermal receptors in the skin and core, and coordinating heat dissipation through vasodilation, sweating, and behavioral responses (Noble & Robertson, 1996).

Exercise intensity profoundly affects both the rate of heat production and the body's capacity to maintain thermal balance. Low-intensity exercise, typically below 40% of maximal oxygen consumption ($\text{VO}_{2\text{max}}$), results in modest increases in core temperature, with the cardiovascular and sweat mechanisms effectively compensating for the generated heat (Gleeson & Bishop, 2000).

Moderate-intensity exercise, ranging between 40% and 70% $\text{VO}_{2\text{max}}$, produces a progressive rise in core temperature, requiring increased skin blood flow and sweating to prevent heat storage (Nybo et al., 2014). At high intensities, above 70% $\text{VO}_{2\text{max}}$, metabolic heat production rises exponentially, and compensatory thermoregulatory mechanisms can be challenged, particularly under environmental heat stress, leading to potential hyperthermia and reductions in exercise performance (Cheuvront et al., 2010).

During exercise, the cardiovascular system plays a central role in thermoregulation. Increased cardiac output supports both the metabolic demands of working muscles and the delivery of blood to the skin for heat dissipation (Rowell, 1974). At higher exercise intensities, however, a competition arises between the demands of skeletal muscle perfusion and skin blood flow, which may compromise the body's ability to dissipate heat efficiently (Crandall & González-Alonso, 2010). Sweating is another essential thermoregulatory mechanism that facilitates evaporative heat loss. The rate of sweat production increases with exercise intensity, reflecting the body's attempt to balance heat gain and loss (Gagnon & Kenny, 2012). Excessive sweating, however, can lead to dehydration, reduced plasma volume, and impaired thermoregulation, thereby increasing cardiovascular strain and core temperature during prolonged high-intensity exercise (Sawka et al., 2011).



Environmental factors such as temperature, humidity, and solar radiation significantly influence thermoregulatory responses during exercise. In hot or humid environments, the evaporative cooling capacity of sweat is reduced, accelerating the rise of core temperature even at moderate exercise intensities (Périard et al., 2015). High-intensity exercise in such conditions can precipitate heat-related illnesses, including heat exhaustion and heat stroke, especially in unacclimated individuals or those inadequately hydrated (Casa et al., 2015). Conversely, exercising in cooler environments can enhance heat dissipation and reduce thermal strain, allowing for prolonged high-intensity efforts without excessive increases in core temperature. The interaction between exercise intensity and environmental conditions underscores the importance of contextual factors in predicting thermoregulatory outcomes and performance. Metabolic heat production, the primary source of heat during exercise, increases linearly with work rate but can exhibit exponential effects at near-maximal efforts. Muscle contractions during high-intensity exercise can increase metabolic heat production up to twenty times the resting rate, challenging the efficiency of thermoregulatory mechanisms (Rowell, 1974; Cheung & McLellan, 1998). Core temperature typically rises by 0.5–1°C during moderate exercise, while high-intensity exercise can elevate core temperature by 2–3°C, approaching or exceeding thresholds associated with impaired performance and cognitive function (González-Alonso et al., 1999). The body responds through complex integrated mechanisms, including vasodilation, increased cardiac output, enhanced sweat rate, and behavioral adjustments such as reduced intensity or seeking cooler conditions. These physiological responses are critical to maintaining homeostasis but are inherently limited by the body's heat storage capacity, hydration status, and environmental conditions.

Thermoregulatory strain has direct implications for exercise performance. Elevated core temperature increases perceived exertion, reduces muscular endurance, and can impair both aerobic and anaerobic capacity (González-Alonso et al., 1999). In endurance sports, maintaining an optimal core temperature is critical for sustaining performance over prolonged periods, whereas in high-intensity interval exercise, the rapid accumulation of heat may necessitate longer recovery intervals to avoid overheating.

Heat acclimation strategies, including repeated exposure to exercise in hot environments, improve thermoregulatory efficiency by increasing plasma volume, sweat rate, and cardiovascular stability, thereby enabling better tolerance of high-intensity workloads (Cheuvront et al., 2010). These adaptations highlight the importance of conditioning, hydration strategies, and pacing in managing thermal stress during exercise.

Individual variability also plays a significant role in thermoregulatory responses to exercise intensity. Factors such as age, sex, body composition, fitness level, and acclimation status influence both the magnitude and efficiency of heat dissipation mechanisms (Gagnon & Kenny, 2012; Kenefick & Cheuvront, 2012). For example, trained athletes generally exhibit enhanced cardiovascular and sweat responses, enabling them to tolerate higher exercise intensities with smaller increases in core temperature. In contrast, untrained or sedentary individuals, as well as older adults, may experience faster rises in core temperature and greater thermoregulatory strain during similar exercise intensities. Sex differences in sweat rate and skin blood flow also contribute to differential thermal responses, emphasizing the need for individualized exercise prescriptions in both recreational and competitive settings.

From a practical standpoint, monitoring core temperature and thermoregulatory markers is crucial for optimizing performance and safety during physical activity. Heart rate, perceived exertion, skin temperature, and sweat rate are commonly used indicators of thermal strain, while emerging technologies such as ingestible temperature sensors and wearable thermometers provide real-time assessments (Nybo et al., 2014). Proper hydration, pacing strategies, and environmental adjustments are essential for mitigating the impact of high exercise intensity on thermoregulation. In addition, understanding the interactions between exercise intensity, environmental conditions, and individual characteristics can inform training protocols, competitive strategies, and workplace safety recommendations.

Exercise intensity has a profound impact on thermoregulation and body temperature during physical activity. Low-intensity exercise elicits minor thermal stress, whereas moderate and high-intensity exercise significantly challenge thermoregulatory mechanisms, particularly under hot or humid conditions. Core temperature rises in proportion to metabolic heat production, necessitating cardiovascular adjustments, sweating, and behavioral strategies to maintain



thermal homeostasis. Environmental factors, individual variability, and hydration status modulate these responses, influencing both performance and safety.

Effective management of thermal strain requires a comprehensive understanding of the physiological principles underlying thermoregulation and the interplay between exercise intensity, environment, and individual characteristics. Future research should focus on optimizing exercise prescription and cooling strategies, as well as exploring wearable technologies for real-time monitoring of thermoregulatory responses to enhance performance and reduce the risk of heat-related illnesses.

II. PHYSIOLOGICAL BASIS OF THERMOREGULATION

Core body temperature (T_c) is regulated through heat production and loss. Metabolic heat from muscular contractions increases with higher workloads (Rowell, 1974). The cardiovascular system redistributes blood flow to the skin to facilitate heat loss through conduction, convection, radiation, and evaporation (Sawka et al., 2011). Sweating plays a key role in heat loss but is affected by environmental conditions (Kenefick & Cheuvront, 2012).

III. EXERCISE INTENSITY AND HEAT PRODUCTION

Exercise intensity is typically quantified as a percentage of maximal oxygen consumption (% VO_2max) or metabolic equivalents (METs). Higher intensity increases metabolic rate, thus elevating heat production:

1. **Low intensity ($\leq 40\% \text{VO}_2\text{max}$):** Minimal increase in core temperature; thermoregulation is efficient (Gleeson & Bishop, 2000).
2. **Moderate intensity ($40\text{--}70\% \text{VO}_2\text{max}$):** Progressive increase in T_c ; compensatory mechanisms maintain thermal balance (Nybo et al., 2014).
3. **High intensity ($>70\% \text{VO}_2\text{max}$):** Exponential rise in heat production; increased risk of heat storage and hyperthermia (Cheuvront et al., 2010).

IV. THERMOREGULATORY RESPONSES TO INTENSITIES

Exercise intensity has a profound influence on thermoregulatory responses, as the human body must constantly maintain homeostasis in the face of increased metabolic heat production. Thermoregulation is primarily controlled by the hypothalamus, which integrates signals from central and peripheral thermoreceptors to coordinate heat production and heat loss mechanisms (Noble & Robertson, 1996). As exercise intensity increases, skeletal muscle activity elevates metabolic rate, generating a substantial amount of heat that must be dissipated to maintain core temperature within safe limits (Rowell, 1974).

Low-intensity exercise, typically defined as activities below 40% of maximal oxygen uptake (VO_2max), produces minimal additional heat, and the body's thermoregulatory systems effectively maintain core temperature through modest increases in skin blood flow and sweat rate (Gleeson & Bishop, 2000). Cardiovascular adjustments during low-intensity exercise are minimal, as the circulatory system can simultaneously support active muscles and the skin for heat dissipation without compromising perfusion. Evaporative heat loss through sweating is also modest in this intensity range, and performance is largely unaffected by thermoregulatory demands.

Moderate-intensity exercise, ranging from 40% to 70% VO_2max , leads to a progressive increase in metabolic heat production, which triggers more robust thermoregulatory responses. The cardiovascular system adapts by increasing cardiac output and redistributing blood flow to the skin, facilitating convective and radiative heat loss (Crandall & González-Alonso, 2010). Simultaneously, sweat rates increase to enhance evaporative cooling, which is the primary mechanism of heat dissipation in humans during exercise in temperate to warm conditions (Sawka et al., 2011). These responses are generally sufficient to prevent dangerous elevations in core temperature under normal environmental conditions; however, performance can begin to be affected if the thermal load approaches the limits of heat dissipation capacity. Core temperature rises gradually with moderate-intensity exercise, and the body responds by increasing heart rate, respiratory rate, and sweating, all of which represent compensatory mechanisms to prevent hyperthermia (Nybo et



al., 2014). Dehydration, even in moderate exercise, can reduce plasma volume and limit the effectiveness of these thermoregulatory adjustments, highlighting the critical role of hydration in maintaining thermal balance.

High-intensity exercise, exceeding 70% VO_2max , presents a significant challenge to thermoregulation due to the exponential increase in heat production. Muscular metabolism at this intensity generates substantial amounts of heat that can exceed the body's capacity to dissipate it, particularly under hot or humid environmental conditions (Cheuvront et al., 2010). The cardiovascular system is under considerable strain, as it must simultaneously deliver oxygenated blood to active muscles while diverting sufficient blood flow to the skin for heat dissipation.

This competition can compromise either muscular performance or cooling efficiency, resulting in progressive rises in core temperature (González-Alonso et al., 1999). Sweating reaches maximal rates, but its effectiveness may be reduced in humid environments, leading to an accumulation of heat within the body. Consequently, high-intensity exercise is associated with significant cardiovascular strain, elevated perceived exertion, and reduced endurance performance (Cheung & McLellan, 1998). The risk of heat-related illnesses, such as heat exhaustion and heat stroke, becomes considerable if thermoregulatory mechanisms are overwhelmed, particularly when dehydration is present or acclimatization to heat is incomplete (Casa et al., 2015).

Environmental conditions play a critical role in modulating thermoregulatory responses across exercise intensities. In hot or humid conditions, evaporative cooling becomes less effective due to the reduced gradient for sweat evaporation, and core temperature rises more rapidly at all intensities (Périard et al., 2015). Even low- and moderate-intensity exercise in such environments can result in significant thermal strain if the body is unable to dissipate heat effectively. Conversely, in cooler environments, heat loss through conduction, convection, and radiation is more efficient, reducing cardiovascular and sweating demands. Clothing, airflow, and solar radiation exposure further influence the effectiveness of thermoregulatory mechanisms, particularly at higher exercise intensities (Kenefick & Cheuvront, 2012). The interplay between exercise intensity and environmental conditions underscores the importance of individualized strategies for heat management, including pacing, hydration, and acclimatization protocols.

The role of sweating as a thermoregulatory effector is highly dependent on exercise intensity. At low intensities, sweat production is minimal, and minor increases in skin blood flow suffice to maintain thermal balance. As intensity rises, sweat rate increases linearly with metabolic heat production, providing an essential mechanism for heat dissipation through evaporation (Gagnon & Kenny, 2012). High-intensity exercise results in maximal sweat rates, which can lead to substantial fluid losses and, if not adequately replaced, a decrease in plasma volume. Reduced plasma volume compromises skin blood flow, heat transfer, and cardiovascular function, creating a feedback loop that can exacerbate hyperthermia and reduce exercise performance. Consequently, hydration status, acclimatization, and the ability to replace fluids during prolonged high-intensity activity are critical determinants of thermoregulatory efficiency and safety.

In addition to cardiovascular and sweat responses, other physiological adaptations influence thermoregulation during repeated high-intensity exercise. Heat acclimation, for example, improves sweating efficiency, increases plasma volume, and reduces cardiovascular strain at a given workload (Cheuvront et al., 2010). Endurance training also enhances thermoregulatory capacity by improving cardiovascular efficiency, increasing cutaneous vasodilation, and delaying the onset of hyperthermia (González-Alonso, 2012). Conversely, untrained individuals may experience rapid rises in core temperature at moderate to high intensities, highlighting the interindividual variability in thermoregulatory responses. Age, sex, fitness level, and genetic factors further influence how the body responds to the thermal load imposed by exercise (Nybo et al., 2014).

Overall, thermoregulatory responses to exercise are intensity-dependent and involve integrated cardiovascular, sweat, and central nervous system mechanisms. Low-intensity exercise produces minimal thermal stress, moderate intensity elicits progressive physiological adaptations, and high-intensity activity imposes substantial heat load and cardiovascular strain, especially under challenging environmental conditions. Effective thermoregulation relies on a balance between heat production and heat dissipation, which is influenced by hydration, acclimatization, fitness, and environmental factors. Understanding these responses is crucial for optimizing performance, preventing heat-related



illnesses, and designing training programs that consider both exercise intensity and environmental conditions. The integration of these mechanisms illustrates the complex interplay between metabolic heat production, thermoregulatory effectors, and environmental demands, highlighting the importance of tailored strategies for safe and effective physical activity across varying intensities.

V. CARDIOVASCULAR ADJUSTMENTS

With increased exercise intensity, cardiac output and skin blood flow increase to support heat transfer to the periphery (Rowell, 1974). However, intense workloads can lead to competition between working muscles and skin for blood flow, compromising heat dissipation (Crandall & González-Alonso, 2010).

VI. EVAPORATIVE COOLING AND SWEAT RESPONSE

Sweating rate grows with exercise intensity to promote evaporative cooling (Gagnon & Kenny, 2012). However, dehydration due to excessive sweating reduces plasma volume and impairs thermoregulation (Sawka et al., 2011). Thermoregulation is a critical physiological process that maintains core body temperature within narrow limits during physical activity, particularly when metabolic heat production increases with exercise intensity (González-Alonso, 2012). Among the various mechanisms of heat dissipation, evaporative cooling through sweating is considered the most effective means of removing excess heat during prolonged or high-intensity exercise, especially under conditions of elevated environmental temperature and humidity (Sawka et al., 2011).

The production of sweat is mediated by eccrine glands, which are densely distributed across the body, and the rate of sweat secretion is influenced by both the internal heat load and external environmental stressors (Kenefick & Cheuvront, 2012). As exercise intensity increases, the body's metabolic rate rises proportionally, resulting in greater heat production, and consequently, the hypothalamic thermoregulatory center stimulates an augmented sweat response to prevent hyperthermia (Cheung & McLellan, 1998; Rowell, 1974).

During low-intensity exercise ($\leq 40\%$ VO_2max), the rate of metabolic heat production is modest, and sweat rates remain relatively low, allowing for effective heat dissipation primarily through radiation and convection (Gleeson & Bishop, 2000). However, as exercise intensity progresses to moderate levels ($40\text{--}70\%$ VO_2max), core temperature begins to rise more significantly, and the evaporative heat loss system is increasingly recruited to maintain thermal homeostasis (Nybo et al., 2014).

The rate of sweating typically increases linearly with rising core temperature, and the onset of sweating occurs earlier in trained individuals due to heat acclimation and improved thermoregulatory efficiency (Gagnon & Kenny, 2012). At high-intensity exercise ($>70\%$ VO_2max), the metabolic heat load rises exponentially, placing substantial demands on evaporative cooling. In such scenarios, sweat rates can reach 1–2 liters per hour or more in humans, depending on environmental conditions, hydration status, and individual physiological characteristics (Sawka et al., 2011; Cheuvront et al., 2010).

The efficacy of evaporative cooling is influenced by both internal and external factors. Internally, factors such as hydration status, acclimatization, fitness level, and body composition play key roles. Adequate hydration maintains plasma volume, allowing for sustained sweat production and cardiovascular function during heat stress (Kenefick & Cheuvront, 2012). Dehydration, however, reduces plasma volume and skin blood flow, impairing sweat secretion and heat dissipation, which increases the risk of hyperthermia and exercise-associated heat illness (Casa et al., 2015). Acclimatization to heat also improves sweat response; chronically heat-acclimated individuals exhibit earlier onset of sweating, higher sweat rates, and more dilute sweat, which optimizes both fluid and electrolyte balance while enhancing evaporative cooling (Périard et al., 2015).

Externally, environmental factors such as temperature, humidity, wind speed, and radiant heat significantly influence the effectiveness of sweating as a cooling mechanism. High humidity reduces the vapor pressure gradient between the skin and the environment, limiting evaporation and forcing the body to rely more heavily on cardiovascular adjustments for heat dissipation (Crandall & González-Alonso, 2010). Conversely, dry and windy conditions enhance evaporation,



allowing for greater heat loss even at higher exercise intensities. This interaction between exercise intensity and environmental stress underlines the critical role of evaporative cooling in maintaining thermal balance during physical activity.

Sweat composition also varies with exercise intensity and environmental adaptation. At lower intensities, sweat tends to have higher electrolyte concentration relative to volume, whereas at high-intensity exercise, particularly in heat-acclimated individuals, sweat becomes more dilute due to improved reabsorption of sodium in the sweat glands, reducing the risk of electrolyte imbalance (Kenefick & Cheuvront, 2012). This adaptation is particularly important during prolonged endurance exercise, where excessive electrolyte loss can lead to hyponatremia, muscle cramps, and impaired neuromuscular function. Moreover, the total body surface area available for sweating and the density of sweat glands in key regions, such as the torso and limbs, contribute to individual differences in evaporative cooling capacity (Gagnon & Kenny, 2012).

The cardiovascular system works synergistically with the sweat response to optimize heat loss. During high-intensity exercise, cutaneous vasodilation increases skin blood flow, transporting heat from the core to the skin surface, where evaporation from sweat becomes the primary cooling mechanism (Rowell, 1974; González-Alonso, 2012). However, there exists a physiological trade-off at high intensities; both working muscles and skin compete for blood flow, and when cardiovascular capacity is exceeded, the efficiency of evaporative cooling diminishes, potentially resulting in increased core temperature and reduced exercise performance (Crandall & González-Alonso, 2010). Consequently, the balance between sweat-mediated cooling and cardiovascular support is a critical determinant of thermoregulatory effectiveness under intense exercise conditions.

Understanding the dynamics of sweat response and evaporative cooling is vital for designing effective strategies to mitigate heat strain and optimize performance. Hydration protocols, heat acclimatization, and individualized pacing strategies are practical interventions aimed at maintaining core temperature within safe limits while sustaining high-intensity exercise (Cheuvront et al., 2010). Additionally, clothing and fabric selection can enhance sweat evaporation by promoting air circulation and minimizing moisture retention, further supporting thermoregulation in athletic and occupational settings. Emerging wearable technologies that monitor sweat rate, electrolyte loss, and skin temperature provide real-time feedback, enabling athletes to modulate intensity and hydration strategies to maintain thermal homeostasis (Périard et al., 2015).

Evaporative cooling through sweating is a cornerstone of thermoregulation during physical activity, with its effectiveness closely tied to exercise intensity, hydration status, environmental conditions, and individual physiological adaptations. Low-intensity exercise imposes minimal thermal strain and allows for efficient thermoregulatory balance, while moderate and high-intensity exercise significantly increases metabolic heat production, necessitating enhanced sweat rates and cardiovascular adjustments to prevent hyperthermia (Sawka et al., 2011; González-Alonso, 2012).

Dehydration, inadequate heat acclimatization, and high ambient humidity can impair evaporative cooling, increasing core temperature, reducing performance, and elevating the risk of heat-related illnesses (Casa et al., 2015; Cheung & McLellan, 1998). Therefore, understanding the mechanisms, determinants, and limitations of sweat-mediated evaporative cooling is critical for optimizing safety, endurance, and performance during physical activity under varying intensities and environmental conditions.

VII. ENVIRONMENTAL INTERACTIONS

Heat stress is exacerbated in hot and humid conditions, where evaporative cooling is limited, and core temperature rises more rapidly (Périard et al., 2015). High intensity under such conditions can lead to heat exhaustion or heat stroke if compensatory capacity is exceeded.



VIII. PERFORMANCE AND SAFETY IMPLICATIONS

Thermoregulatory strain reduces endurance performance, increases perceived exertion, and elevates cardiovascular strain (González-Alonso et al., 1999). Heat illnesses are a significant risk during prolonged high intensity exercise, especially without acclimation or adequate hydration (Casa et al., 2015).

Table 1: Exercise Intensity and Thermoregulatory Responses

Exercise Intensity	Metabolic Heat Production	Core Temp Change	Thermoregulatory Response	Performance Impact
Low ($\leq 40\%$ VO_2max)	Low	Small rise	Efficient heat loss	Minimal
Moderate (40–70% VO_2max)	Moderate	Moderate rise	Increased sweat & blood flow	Slight impairment
High ($>70\%$ VO_2max)	High	Large rise	Elevated sweat, cardiovascular strain	Significant impairment
Heat/Humid Conditions	Very high effective heat load	Rapid rise	Reduced evaporative cooling	Severe impairment, risk of heat illness

IX. CONCLUSION

Exercise intensity has a profound impact on thermoregulation and body temperature dynamics. As intensity increases, metabolic heat production rises exponentially, placing greater demands on cardiovascular and sweat mechanisms. The ability to dissipate heat effectively is crucial to maintaining performance and preventing heat-related disorders. Future research should continue exploring individualized thermoregulatory responses, especially in varying environmental conditions and in populations with compromised heat tolerance.

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