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Effectiveness of an Intermittent Heat Exposure Protocol to Maintain Heat Acclimation

J. Luke Pryor, PhD

University of Connecticut, 2015

Background: Heat acclimation (HA) adaptations are temporary and must be sustained for the continued safety of those periodically exerting themselves in hot conditions. **Purpose**: To assess whether an intermittent exercise-heat exposure protocol can mitigate HA decay 25 days after initial acclimation. **Methods**: Sixteen males (VO_{2max}=54.98±5.5 ml·kg^{-1}·min^{-1}) were pair-matched using physical training duration, VO_{2max}, and body surface area then randomly allocated to a no heat (NHE; n=7) or intermittent exercise-heat exposure (IHE; n=9) group. All participants heat acclimated by completing 10-11 days of low-to-moderate intensity exercise (90-240 min) in hot conditions (40℃, 40%RH). Both groups completed a Pre HA and Post HA heat stress test (HST) consisting of two hours of exercise at 45% VO_{2max} in hot conditions to assess HA. After Post HA, participants completed the HST in either a hot (IHE; 40℃, 37%RH) or thermoneutral environment (NHE; 24℃, 21%RH) every fifth day for 25 days with both groups exercising in the hot condition at day 25 (+25d). Thermoregulatory, cardiovascular, and circulating biomarkers of stress were evaluated. Self-led out-of-lab physical activity duration and intensity (heart rate [HR]) were recorded for 25 days after HA. **Results**: Both groups heat acclimated as post-exercise HR and rectal temperature (T_{re}) were lower and sweat rate higher at Post HA versus Pre HA (all p≤0.05). At +25d, post-exercise HR was attenuated in IHE versus NHE (mean difference [NHE-IHE]=28

bpm (95%CI [8, 48], effect size [ES]=1.41, p=0.01) but sweat rate (0.13 $L \cdot hr^{-1}$, 95%CI [-0.21, 0.46], ES=0.36, p=0.44), skin temperature (0.65℃ (95%CI [-0.17, 1.47], ES=0.85, p=0.11) and T_{re} (0.47℃, 95%CI [-0.24, 1.19], ES=0.68, p=0.18) were similar. Post-exercise cortisol and epinephrine concentrations were higher in NHE versus IHE at +25d (p≤0.046). At +25d, heat adaptation decay was greater in NHE than IHE for T_{re} (87% versus 2.7%), skin temperature (44% versus 18%), and HR (163% versus 53%). Out-of-lab exercise intensity and +25d post-exercise HR were inversely related in IHE (r=-0.89, p=0.017). **Conclusions**: Periodic exercise-heat exposure every five days mitigated rectal temperature decay and cardiovascular strain 25 days after initial HA efforts. Intense exercise in thermoneutral environments in addition to exercise-heat stress after HA aids in minimizing adaptation decay.

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Chapter 1. Review of Literature

Scope of the problem

Hot environments reduce an individual's ability for prolonged exercise. The combination of exercise and heat stress imparts exaggerated strain on the thermoregulatory and cardiovascular systems (32, 56). This is caused by increased skin temperature (T_{sk}) creating a reduced core-to-skin thermal gradient. The vasodilation of skin vascular beds and increased blood flow is needed to transport heat from the core to periphery. The shunted blood flow to the skin creates competition for limited cardiac output between cutaneous and skeletal muscle vascular beds. Cardiac output is sustained (or increased if exercising) through compensatory increases in cardiac frequency. Moreover, exercise in hot conditions leads to losses in total body water and electrolytes due to sweating. Over time hypovolemia occurs, further impairing circulatory and thermoregulatory functions resulting in decreased athletic performance and increased risk of thermal injury if fluids are not replaced (32, 56).

Heat-related injuries, especially exertional heat stroke and myocardial infarct after exercise-heat stress, are serious life-threatening problems in several occupations such as firefighters, military personnel, and athletes of all ages (1, 16, 20, 41, 55). Between 1997 and 2006, heat-related injuries treated in U.S. emergency departments increased 133.5%, accounting for an estimated 54,983 patients (55). Further, in American football, more exertional heat stroke deaths occurred from 2005-09 than any five-year period in the previous 35 years (54). Keeping recreational and occupational athletes safe during physical activity in hot, humid conditions is critically important.

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While strategies have been implemented to mitigate heat-related injury (e.g., work-rest cycles, body cooling, hydration, education), the continued rise in heat-related injuries observed in emergency departments (41, 55) and high school athletes (42) necessitate investigation into physiological adaptations to the heat. Heat acclimation (HA) reduces resting and exercising body temperature, increases sweat rate and sensitivity, and reduces cardiovascular strain (6, 13, 49, 51, 72). Taken together, these physiological adjustments enhance heat dissipation mechanisms that mitigate thermal load, improve exercise-tolerance in the heat, and ultimately contribute in part to the prevention of exertional heat illness (9, 60).

Beyond preventing exertional heat illness, HA has gained popularity among scientists and athletes attempting to improve endurance performance in hot and cool environments (17, 18, 64, 67). Regardless of the intended application, the benefits of HA are temporary (9, 28, 60). It follows that for prolonged protection against thermal injury and mitigation of physiological strain during physical activity, HA associated adaptations must be sustained.

Heat acclimation

Heat acclimation occurs in an artificial (heat chamber) setting while heat acclimatization occurs in a natural (outdoor) setting. Both acclimation and acclimatization are effective modalities (8, 68). Acclimatization in humid heat elicits greater T_{sk}, sweat rates, and circulatory adaptations compared to dry heat. These changes are thought to increase skin wettedness and optimize evaporative heat losses, although sufficient scientific support is for this theory is lacking (73). To facilitate sudomotor (e.g., neural activation of sweat glands) and cardiovascular gains, it may be

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beneficial to train in humid heat during the end of acclimation protocols (67). Perhaps most importantly, acclimation/acclimatization should occur in an environment that emulates the competition setting to enable the athlete to experience the exact nature of the exercise-heat stress (9). In addition to exogenous heat stress, endogenous heat production is an important consideration in HA protocols (see below).

Several consecutive days of exercise-heat exposure induce HA resulting in reduced heat storage (66) and body temperature. These important changes are facilitated by increased sweat rate, sweat gland sensitivity, improved skin blood flow, and cardiovascular adjustments that minimize attenuation of stroke volume and heart rate (HR) elevation (47). The combination of these adaptations reduces perceptual, thermal, and physiological strain. Importantly, the risk of exertional heat illness is lessened and heat tolerance and endurance performance in hot and cool environments improved (9, 46).

Researchers have categorized HA into three divisions defined by the duration of exercise-heat exposure. Short-term HA refers to ≤ 7 days, medium-term is defined as 8-14 days and long-term is considered greater than 14 days of consecutive exercise-heat exposure. The daily exercise-heat exposure duration required to achieve thermal habituation is balanced between session duration, intensity, environment, and fitness/training status of the individual undergoing HA. The optimal synergism between these parameters is unknown, but it is accepted that 100 min/day at approximately 40-50% VO_{2max} for 10-14 days will elicit thermal acclimation (60). However, HA has been induced with high intensity short duration (75% VO_{2max}, 30-35 min) exercise-heat stress bouts for 9 days and isothermal techniques for as little as 5 days (27, 40). More

recently, Taylor (75, 76) contends that elevating T_{re} above 38.5℃ for at least 60 minutes during consecutive days is a sufficient heat adaptation stimulus. While many protocols for inducing HA exist, the adaptive impulse for HA remains exercised-heat induced hyperthermia and hypovolemia sufficient to stress thermoregulatory and circulatory effector organs.

Individuals with high VO_{2max} and regularly exercise require fewer exercise-heat exposures to acclimate to the heat. Thus, short-term HA has been recommended for highly trained athletes (10, 28). The reduced time commitment to achieve HA via short-term protocols fits well with the congested training and competition schedules of elite athletes. However, it is important for clinicians and practitioners to consider that complete acclimation is not realized with short-term HA protocols (< 6 days). It is accepted that 75% of HA adaptations occur within 4-6 days of exercise-heat exposure but full acclimation may require up to 14 days (Table 1.1) (9). Weighing the expected benefits and time required to achieve these benefits via each respective HA protocol is warranted on an individual and competition specific basis.

Table 1.1

"Plateau days" of Physiological Adaptations (Point at Which Approximately 95% of the Adaptation Occurs) During Heat Acclimatization

Adaptation	Days of heat acclimatization												
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
Heart rate decrease			—	—										
Plasma volume expansion			—	—										
Rectal temperature decrease				—	—									
Perceived exertion decrease			—	—										
Sweat Na^+ and Cl^- concentration decrease*				—	—	—	—							
Sweat rate increase							—	—	—	—	—	—	—	—
Renal Na^+ and Cl^- concentration decrease			—	—	—	—	—							

*While consuming a diet low in NaCl.

Reprinted from Armstrong and Dziados 1986.

Note. The grayed boxed represents the duration and expected adaptations gained from short-term (< 7 days) heat acclimation.

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The physiological changes characterizing a heat acclimated state is induced at varying rates. In their seminal review, Armstrong and Maresh (9) eloquently describe the expected induction rates for the major HA adaptations which are curvilinear in nature (Figure 1.1). Adaptations of cardiovascular origin are the first to occur, usually within 4-6 days followed by reductions in resting and exercising body temperature, plateauing around 5-8 days. Sweat gland adaptations usually take longer to develop (5-14 days) as does electrolyte conservation at the eccrine gland and kidney. This process is mediated by the secretion of aldosterone in response to repeated bouts of exercise-heat stress that induce hypovolemia and blood sodium perturbation. Several physiological changes beyond the classical heat adaptation responses (e.g., HR, sweat rate, and body temperature) have been reported following HA (Table 1.2).

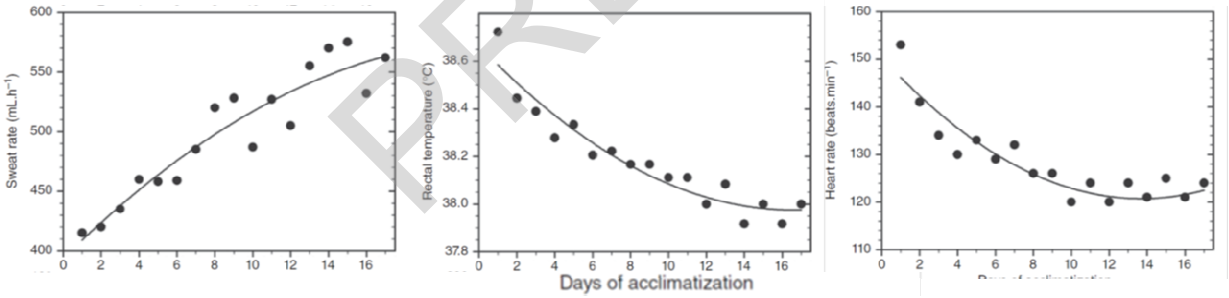

Figure 1.1. Rectal temperature, sweating, and heart rate show rapid initial responses then plateau during successive days of bench stepping (12 steps/min, 4 hours) under 34 C, 80% RH. Data from Wyndham et al. (85).

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Table 1.2. Physiological Adaptations Following Heat Acclimation/Acclimatization

Category	**Increased**	**Decreased**
Thermoregulatory		
	Sweat rate	Sweat electrolyte concentration
	Sweat sensitivity	Resting body temperature
	Skin blood flow	Exercising body temperature
		Exercising skin temperature
		Core-to-skin temperature gradient
Cardiovascular		
	Maximal cardiac output	Sub-maximal heart rate
	Maximal oxygen uptake	
	Plasma volume	
	Total body water	
	Ventricular efficiency	
Metabolic		
		Running economy
		Carbohydrate metabolism
Other		
	Exercise-heat tolerance	Fatigue perception
	Speed at lactate threshold	Effort perception
	Intracellular HSP70	Thermal perception

Table modified from Guy et al. (33)

After HA, on average, one would expect the following degree of adaptation: a post-exercise HR decrease of 16 bpm (range = 10-36), post-exercise body temperature decrease of 0.4℃ (0.17-0.80), post-exercise T_{sk} decrease of 0.68℃ (0.22-1.89), resting body temperature reduction of 0.2℃ (0.01-0.30), and 0.29 $L \cdot hr^{-1}$ (0.00-0.44) increase in

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sweat rate (11, 19, 27, 40, 47, 61, 63, 66, 71, 80). Plasma volume expansion in response to HA appears is variable, increasing 200-500 mL or 3-28% post HA (9, 62, 63). Certainly differences in HA protocol (traditional vs. hyperthermia controlled, number and duration of exercise-heat exposures, frequency, and ambient conditions) and fitness characteristics of the participants affect the magnitude of physiological change derived from HA.

Methodological considerations for inducing heat acclimation

As HA develops, a progressively lower training or adaptive impulse is elicited in traditional constant workload protocols because relative intensity and adaptation are inversely related. It is theorized that this may limit the magnitude of adaptation if precautions are not instituted. For example, increasing exercise duration, intensity, or environmental conditions (76). Authors from international labs advocate that isothermic acclimation protocols are adaptively superior to traditional regimens because exercise intensity is guided by T_{re} responses, not absolute workloads (27, 62, 76). Thus, as adaptation occurs, exercise intensity must increase to achieve similar thermoregulatory strain. However, similar physiological responses between the two techniques were recently observed in a small cohort (n=8) of males with above average aerobic fitness (VO_{2max} range 45-50 $mL \cdot kg^{-1} \cdot min^{-1}$) (30) refuting this superiority claim. Additional evidence is required before definitive conclusions can be drawn, however.

Body temperature measurement equipment and access to artificial indoor conditions pose limitations to the individuals without access to this equipment. Alternatively, Periard et al. (64) suggested using HR to measure relative intensity and acclimation progress. One wonders how effective guiding exercise intensity with HR

would be during exercise-heat stress given the propensity of dehydration and possibility of cardiac drift (53). Alternatively, it has been demonstrated that highly trained runners (8) and team sport athletes (68) can acclimate to the heat outdoors (acclimatization) without affecting team directed training regimens. Importantly, HA induction and decay responses have been shown to vary between individuals (28, 67, 69) and a trial and error period that occurs months before competition is prudent to assess physiological responses.

Relationship between physical training and heat acclimation induction

Endurance training evokes a wide array of cardiovascular, pulmonary, and metabolic adaptations that elevate maximal oxygen consumption (VO_{2max}). Additionally, during acute exercise, large quantities of thermal energy are created, increasing body temperature proportionate to intensity. As a consequence, many of the physiological adaptations elicited following high-intensity endurance training are similar to thermally adapted individuals, suggesting a partial positive cross acclimation or adaptation (38, 76). Similar physiological improvements include lower resting HR and core temperature, expanded blood volume, greater sweat rate and sensitivity, and greater exercise-heat tolerance (76). Many have observed that highly trained athletes appear partially, but not fully, heat acclimated (10, 60, 65). The optimal balance between exercise intensity, duration, and frequency in temperate environments to facilitate HA remains largely unknown.

VO_{2max} has been shown to account for approximately 42-46% of variance in the rate of acclimation measured as the time to rectal temperature (T_{re}) plateau, an accepted measure of a heat acclimated state (59). However, it is not VO_{2max} *per se*, that

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is important, but the physiological changes during the acquisition of higher cardiorespiratory fitness that promotes the quasi-acclimated state. For physical activity to contribute to heat adaptation it appears that increased internal and cutaneous temperature with hypovolemia must occur during exercise. Prospective studies using swimmers (12) and sweatless training (35) established the notion that exercise without a sustained increase in body temperature resulted in insufficient stimulus for HA. Although regular exercise elicits a partial HA state, complete adaptation to the heat requires 5-14 consecutive days of moderate to intense exercise (30-120 minutes) in hot ambient conditions sufficient to elevate body (core and skin) temperature and stress fluid homeostasis.

Heat acclimation decay

Decay of HA induced benefits remains to be fully characterized. Studies examining HA decay are sparse compared to induction. The early HA decay studies of the 1940-60's were rife with inadequacies but pioneering in that they showed the retention of classic HA adaptations varies between individuals and between environments (hot-dry vs. hot-humid) (60). Common shortcomings of these studies include very small sample sizes, incomplete acclimation, or inappropriate measures (3, 60, 84). More recent investigations examining the decline of the heat acclimated phenotype have better characterized the process, but not without disagreement (11, 19, 26, 27, 61, 66, 70, 71, 74, 80, 81). Generally, the classical acclimation criteria of reduced T_{re} and HR persist for approximately 7-21 days (9, 60, 76), although reports of adaptations persisting longer (up to 26 days) are available but few (19, 80). Several questions regarding HA decay, and how to extend acclimation benefits remain. How

much exercise-heat exposure is required to sustain HA adaptations? How long can we expect to extend these physiological adjustments before re-acclimating? After HA, does exercise have to occur in a hot environment to prolong HA adaptations? Is maintaining a fully acclimated state required to reap the prophylactic and ergogenic benefits of HA during exercise-heat stress?

Our current body of HA decay knowledge can provide insight and direction into exploring these important questions. Heat adaptations that occur the earliest also decay the fastest (9, 28, 60). Cardiovascular improvements are gained and lost exponentially in a few days, with and without exercise-heat exposure, respectively (9). Body temperature and sweat rate/sensitivity take longer to develop but also demonstrate a slower decay rate (60).

Taylor (76) purported HA induction and decay is not linear, but cyclical in nature, with decay lasting longer than the time required for acquisition. One of the first descriptions of HA decay was by Dreosti (21) who characterized the physiological losses in indigenous miners after prolonged removal from working in a hot mine, stating, “these workers showed virtually the same rise in body temperature as did natives with no mining experience.” From this initial observation it was clear that adjustments to the heat exist on a continuum and can be modified by exercise-heat exposure.

Although the timeframe of HA decay is generally accepted, there is great inter-individual variability. Individual differences in heat tolerance and acclimation, physical fitness and activity, and the duration, intensity, and frequency of exercise-heat exposures are cited as factors contributing to HA induction and decay variability (3, 11, 19, 26, 27, 61, 66, 70, 71, 74, 80, 81, 84). Although the spread of physiological