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COMPREHENSIVE REVIEW

Heat-induced hypervolemia: Does the mode of acclimation matter and what are the implications for performance at Tokyo 2020?

Lorenz S. Kissling[a], Ashley P. Akerman[a,b], and James D. Cotter[a]

[a]The School of Physical Education, Sport and Exercise Sciences, University of Otago, Dunedin, New Zealand; [b]Human and Environmental Physiology Research Unit, School of Human Kinetics, University of Ottawa, Ottawa, Ontario, Canada

ABSTRACT

Tokyo 2020 will likely be the most heat stressful Olympics to date, so preparation to mitigate the effects of humid heat will be essential for performance in several of the 33 sports. One key consideration is heat acclimation (HA); the repeated exposure to heat to elicit physiological and psychophysical adaptations that improve tolerance and exercise performance in the heat. Heat can be imposed in various ways, including exercise in the heat, hot water immersion, or passive exposure to hot air (*e.g.*, sauna). The physical requirements of each sport will determine the impact that the heat has on performance, and the adaptations required from HA to mitigate these effects. This review focuses on one key adaptation, plasma volume expansion (PVE), and how the mode of HA may affect the kinetics of adaptation. PVE constitutes a primary HA-mediated adaptation and contributes to functional adaptations (e.g., lower heart rate and increased heat loss capacity), which may be particularly important in athletes of "sub-elite" cardiorespiratory fitness (*e.g.*, team sports), alongside athletes of prolonged endurance events. This review: i) highlights the ability of exercise in the heat, hot-water immersion, and passive hot air to expand PV, providing the first quantitative assessment of the efficacy of different heating modes; ii) discusses how this may apply to athletes at Tokyo 2020; and iii) provides recommendations regarding the protocol of HA and the prospect for achieving PVE (and the related outcomes).

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Introduction

The Olympic and Paralympic games in Tokyo will likely be the most heat stressful to date, by virtue of the high humidity (~2.5–3 kPa) in conjunction with high dry bulb and associated radiant temperatures (dry bulb temperature ~27–31°C, [1]) [2,3]. The median wet bulb globe temperature (WBGT) in Tokyo during the Olympic period is expected to be higher than any of the previous Olympic games; ~4°C hotter than the Rio de Janeiro Olympics, ~12°C hotter than London, and ~3°C hotter than Beijing [2]. Compared to the previous Tokyo Olympics (1964), the games are being held in the hotter period of July/August (WBGT ~8°C higher than October) to satisfy overseas media interests, but subsequently compromising athlete performance and welfare.

High ambient heat stress impairs performance in a range of athletic events [4–9]. For instance, cycling in a warm environment (32–40°C dry bulb) can reduce power output by 6.5% [10] and repeat sprint ability by 9.5% [4], compared to temperate conditions. This is due, at least in part, to substantial cardiovascular strain during competitive exercise, which is exacerbated in the heat [11]. A humid environment further exacerbates this physiological challenge, as expected in Tokyo, and WBGT under-represents the impairment caused by higher humidity for well-trained male and female athletes alike [12,13], because of the lower maximal evaporative power [1]. Compared to 30°C and 24% relative humidity (RH), an increase to 60% or 80% RH leads to a 14 and 22 min reduction in time to exhaustion, respectively [14]. Even at the most modest of performance decrements, a 6.5% reduction in cycling time trial performance during the Rio De Janeiro Olympics – all else being equal – would have been the difference between gold and 21st position [15].

Spectators and athlete support staff may not be aware that these warm-humid environments have a disproportionately larger impact on the fittest athletes, but relatively less than people resting or working at low intensities. Specifically, (i) dry heat loss mechanisms (convection and radiation) can

CONTACT Ashley P. Akerman aakerman@uottawa.ca

transfer little more than resting rates of heat production in warm environments, such that (ii) the additional transfer needed to sustain exercise requires evaporation, but the high ambient vapor pressure limits the gradient for evaporation and thus evaporative heat loss will become maximal at modest work rates, whilst (iii) any extra speed provides negligible additional convective or evaporative transfer (i.e., velocity-vs-heat loss is an exponential relation). So, whereas endurance performance is most impaired for less fit individuals in non-humid heat stress [16], the opposite may be true in humid heat because of the relatively larger importance of evaporation for elite endurance athletes. The heat stress elicited in the Tokyo Olympics (and Paralympics) will therefore concomitantly reduce the performance of sustained/repeated exercise and increase the risk of exertional heat illness [17]. Plans to move competition into the earlier hours have been proposed [18], but even at midnight the WBGT could be ~30°C [2], which would likely still impair performance and carry a risk of exertional heat illness [17].

The Tokyo 2020 Olympics will incorporate 33 sports, each requiring different physical characteristics for peak performance (*e.g.*, a marathoner versus a fencer). The demand of the sport will influence i) the rate of heat production, ii) the potential to offload heat, and iii) the adaptations that may support performance. Endurance events such as road cycling, marathon, and race walking have a high sustained metabolic heat production, but they typically lose heat relatively effectively due to high sweat rates in conjunction with substantial airflow (not necessarily in Tokyo due to high absolute humidity). In this situation, several adaptations from heat acclimation (HA; *Table 1*), and further acute strategies (*e.g.*, precooling, athlete rotation) could limit physiological strain [19] and aid performance. Sports such as fencing (*i.e.*, skill/power/speed based) have an elevated metabolic heat production, but due to clothing or additional environmental limitations to evaporation, increased sweat rate could exacerbate dehydration and in doing so may reduce the psychomotor or cognitive function [20,21] that is crucial for success. This complexity and myriad contributing factors (to performance success) therefore make "one-size fits all" preparation programs complicated and often invalid by way of their lack of specificity.

Performance in the heat can be improved – or rather the performance decrement relative to temperate conditions and/or other athletes can be attenuated – following HA or acclimatization for well-trained endurance athletes [6,22–26] and elite team-sport athletes [25,27,28]. However, there is currently no published controlled-trial research on HA and performance in elite endurance athletes, despite the incorporation of control groups in HA trials with trained (not elite) participants becoming more common [29–31]. Further, studies that have used control groups have almost exclusively matched absolute rather than relative work, which inherently favors the more stressful condition, i.e., HA. The lack of control groups within the limited studies in elite athletes limits the interpretation and application of such findings and indicates the need for further research. While the complexity of these studies is acknowledged, only when these comparisons are forthcoming, can a valid and empirically driven assessment be made regarding the merits of HA for performance in elite athletes (*discussed below*).

HA leads to a range of physiological and psychophysical adaptations that improve one's tolerance to heat stress. The general adaptations to HA are most demonstrably cardiovascular (*e.g.*, increased plasma volume (PV) and reduced heart rate (HR)) and thermoregulatory (*e.g.*, sweat rate and core temperature (T_c)). These collectively support improved thermal comfort and may contribute to potential metabolic [32–35], and possibly neuromuscular adaptations [36]. Increased cellular stress protection, and therefore thermotolerance, may also be induced and could be important in athletic performance contexts [37]. PV expansion (PVE) – a key outcome of HA – supports the maintenance of cardiac output via increased stroke volume and reduced HR [38–40], which increases cardiac reserve and could improve $\dot{V}O_{2\ peak}$ (most likely in untrained only) even in temperate conditions [41–44]. These adaptations are likely to contribute, collectively, to improved exercise performance in the heat (*e.g.*, PVE, reduced T_c [45–47]). PVE therefore supports, and may indeed facilitate other functionally important markers of HA (see Figure 1).

PVE is associated with improved exercise performance in hot environments following repeated heat exposure [26,45,47,48], and may help mediate other beneficial outcomes, as shown in Figure 1. The role of

Table 1. Adaptations to heat acclimation and their importance for different sporting contexts.

Sport	Adaptations	Resting/exercising T_c	Heat loss	Thermotolerance	Thermal comfort	Cardiac output
	Prolonged endurance outdoor (e.g., Marathon)	High $\dot{H}$ Limited ability to manage High airflow	High $\dot{H}$ Limited ability to manage High airflow	High $\dot{H}$ Limited ability to manage Prolonged GI ischemia	Low cognitive demand High skin temp High airflow likely reduce discomfort	High CV demand Already highly adapted CV system Benefit late in exercise? E.g., defense of PV
	Intense endurance outdoor (e.g., 10,000 m)	Very high $\dot{H}$ Limited ability to manage High airflow	Very high $\dot{H}$ Limited ability to manage High airflow	Very high $\dot{H}$ Limited ability to manage GI ischemia	Low cognitive demand High Skin Temp High thermal strain contributes to elevated perceived exertion	High CV demand Already highly adapted CV system Limited time to realize benefit of PVE
	Team sport (e.g., football/soccer)	Moderate – high $\dot{H}$ Breaks allow management WU can elevate starting Tc	Moderate – high $\dot{H}$ Breaks allow management Clothing could limit heat dissipation	Moderate – high $\dot{H}$ Prolonged GI ischemia	Moderate – high cognitive demand Breaks allow management	Moderate – high CV demand Breaks allow management Not "aerobically elite"
	Power, strength, skill (e.g., shooting)	Low $\dot{H}$, short physical exertions Opportunity to cool Contribution to performance likely minimal	Low $\dot{H}$, Limited exposure time Opportunity to cool Contribution to performance likely minimal	Low $\dot{H}$, Limited exposure time Opportunity to cool Contribution to performance likely minimal	High cognitive demand Attention, technique, and strategy critical	Low CV demand Time gives opportunity to limit strain Not "aerobically elite"

The traditionally recognised adaptations to heat acclimation (Columns) are broadly considered in the context of the different major sport categories. **The first row (colour designation)** *corresponds to the importance of the respective adaptation for that category of sport. Black, grey, and white correspond to high, moderate, and low importance, respectively. This is a subjective assessment, based partly on information in other rows within that cell.* **The first row of text** *corresponds to the magnitude of strain expected in that sport category.* **The second row** *corresponds to the opportunity for athletes/support staff to offset this strain.* **The third row** *refers to pertinent sport/context specific factors that would alter the magnitude of this strain, or the specific relevance of the adaptive response. For instance, during intense endurance outdoor there will be a high metabolic heat production and thus a large thermal strain, meaning that core temperature, heat loss mechanisms, and thermotolerance will contribute substantially to performance success. Alternatively, cardiovascular load (or cardiac output) and metabolic heat production are not substantially stressed during power/strength/skill-based sports, and thus the adaptations to these systems/effectors would not meaningfully contribute to performance success. 'Aerobically elite' refers to an already highly adapted cardiovascular system. Abbreviations: CHO, carbohydrate; CV, cardiovascular; ⊠, rate of heat production; PVE, plasma volume expansion; Tc, core temperature; WU, warm-up*

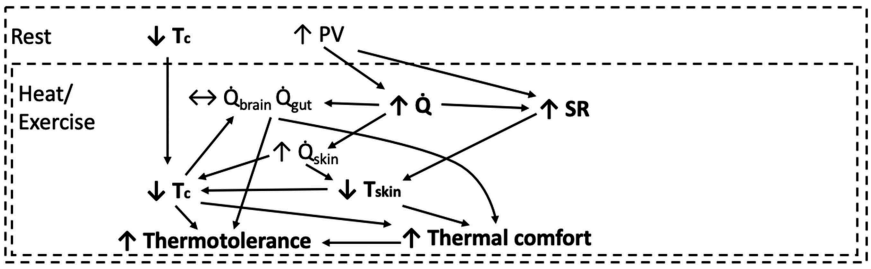

Figure 1. Heat acclimation initiates several adaptations that develop independently and inter-relatedly. Those bolded are mentioned within Table 1. Plasma volume (PV) expansion has several potential roles, albeit these are not definitively demonstrated, esp. for elite endurance athletes. Tc; Core Temperature, $\dot{Q}$; Cardiac Output, $\dot{Q}_{brain}$; Cardiac Output to the Brain, $\dot{Q}_{gut}$; Cardiac Output to the Gut, $\dot{Q}_{skin}$; Cardiac Output to the Skin, SR; Sweat Rate, T_{skin}; Skin Temperature.

heat-induced PVE is difficult to determine, particularly for highly trained endurance athletes, because artificial PVE (via albumin/saline infusion or sodium loading) has shown both no improvement [39,40,49], and positive effects on endurance performance [42,50,51]. Untrained individuals gain cardiovascular and performance benefits from artificial or natural (e.g., HA) PVE in both temperate and hot environments. Athletes do not necessarily [40,52]. They already possess high PV in accordance with their training status and mode of exercise, but this is further expanded by short-term increases in training volume and with HA, so it presumably confers some benefit (e.g., for reasons indicated in Figure 1).

Table 1 highlights some principal limiting factors to exercise performance in the heat, and the associated adaptations observed following HA (as discussed above). The relevance (or not) of these adaptations to specific sporting contexts is seldom considered, despite different sports/events existing in thermally distinct contexts.

HA protocols typically vary from a few days, to more than 2 weeks, and may involve two fundamentally different phases in progressing from brief to prolonged phenotypes [37]. More prolonged exposures have been undertaken in rodents, but yet to be replicated in humans, thus major gaps in knowledge exist regarding the full spectrum of human heat adaptation; *especially* dose:response relations for multi-system physiological adaptation, and inter- and intra-individual factors contributing to the adaptive process. This is pertinent in the context of PVE, as PV contraction (relative to short-term adaptation) is a hallmark of the transition to a long-term HA phenotype [53]. The dosing of HA in time and mode may influence the pattern and outcome of all adaptation, but this has yet to be determined. Most HA studies also vary from 30 to 120 min per session and are completed either actively (exercise in the heat; ExH) or passively via hot air (*e.g.*, sauna) or hot water immersion (HWI). Combinations of these modes have been reported [45,54–59], but mostly within rather than between sessions. Importantly, the mode of HA (ExH, passive air or HWI) may affect the type and volume of adaptations gained, due to the medium (air or water) or nature of the heat stress (active or passive), however, this notion remains almost unexamined despite its theoretical and practical value.

Beyond the HA protocol *per* se, the rate of adaptation also depends on the variable of interest. For example, it is purported that PVE can plateau within ~5 days (one session per day; volume per session not known) whilst sudomotor and vascular adaptations likely take more than 1 week to months [60–63]. These guidelines are often typically based on average responses, and therefore omit important information related to within-group differences [60,64,65]. Thus, the rate of heat adaptation is still poorly understood, but individual factors such as training status [66], previous heat exposure [67], sex [68], and the mode of HA likely have a role.

The protocol of HA employed, and the nature and extent of adaptation sought, must, therefore, consider the environment in which the athletes will likely compete, and the sport-specific determinants of success. Humid conditions, such as those at the Tokyo 2020 Olympics, could mean that increased PV and thus larger vascular volumes available to support high(er) sweat rates may have no benefit on heat loss due to skin wettedness from a low vapor pressure gradient. However, increased skin blood flow could still be important as it serves both dry and evaporative heat loss mechanisms, and PVE is involved a myriad of related and important adaptations (see Figure 1). Nevertheless, in this circumstance, it is unclear whether HA should be of *primary* importance, or greater focus placed on artificial strategies for reducing thermal strain (e.g., "warm up" and precooling) [69].

The intent for the remainder of the review is to i) highlight the capacity for typically available modes of HA (ExH, HWI, and passive hot air) to expand PV, ii) consider how this may apply to athletes attending Tokyo 2020, and iii) provide recommendations regarding PVE and the protocol of HA used. We feel that this approach (sport-, athlete-, and/or outcome-specific) is likely the most scientifically robust and practically appropriate method for assessing recommendations for HA (and wider training) in elite athletes. Comprehensive literature searches focused primarily on HA studies that included measures of blood or plasma volume, to provide a focussed and novel addition to the HA literature.

Plasma volume

PVE occurs across repetitive exposure to passive [70,71] and active heat exposure [72–74]. This section

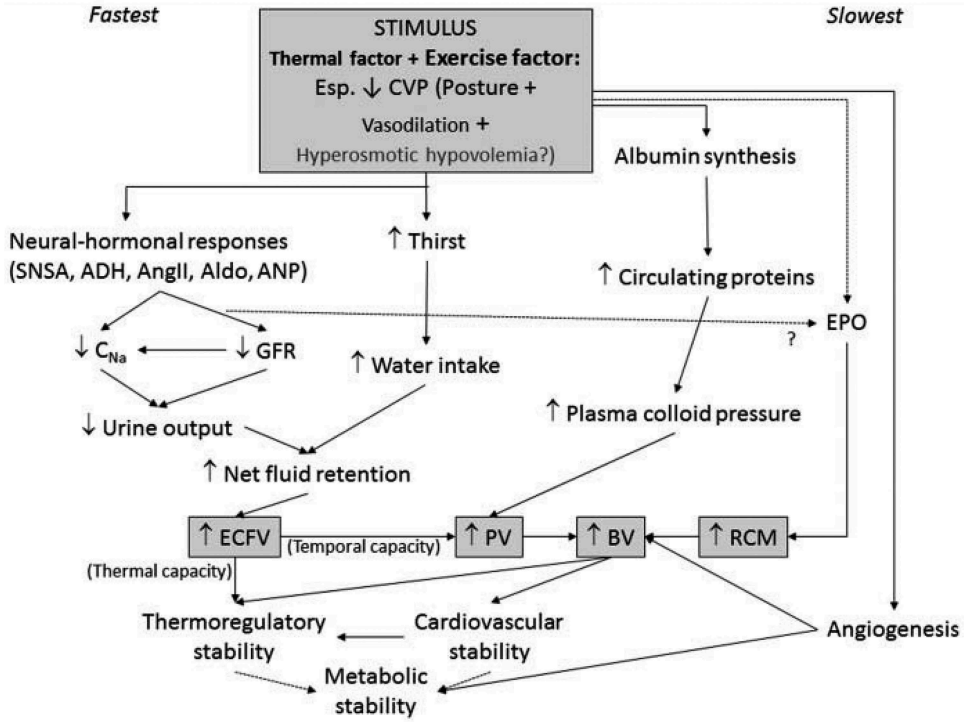

Figure 2. Illustration of the complex and interrelated mechanisms acting on fluid expansion. Used with permission from Akerman [34]. ADH, Anti-diuretic hormone; Aldo, aldosterone; AngII, Angiotensin II; ANP, Atrial natriuretic peptide; BV, Blood volume; C_{Na}, sodium clearance; ECFV, Extracellular fluid volume; EPO, Erythropoietin; GFR, Glomerular filtration rate; PV, plasma volume; RCM, Red cell mass; SNSA, Sympathetic nervous system activity.

provides a brief description of the mechanisms that support PVE. Figure 2 illustrates how PVE can arise from several interrelated mechanisms. These mechanisms, which include fluid-regulating hormones and plasma proteins, are mentioned briefly because they may be important for the different modes of HA. Detailed reviews of PV control are also available elsewhere [75–78].

Fluid conservation can be increased directly through stimulation of the kidneys to retain fluid (via renin-angiotensin-aldosterone system (RAAS)), initiation of thirst (*e.g.*, hypertonicity and anti-diuretic hormone (ADH)), increased fluid movement into the vascular space (*e.g.*, protein translocation and production), and potentially through reductions in arterial blood pressure (ABP) and central venous pressures (that may modulate the above mechanisms).

PVE occurs predominantly through stimulation of fluid conserving hormones and accruing albumin in the intravascular space. Principal fluid conserving hormones are the RAAS and ADH. The RAAS initiates with the release of renin, which then stimulates the adrenal cortex to release angiotensinogen, which is converted to angiotensin I, and angiotensin II. Angiotensin II is a potent vasoconstrictor, that limits the renal loss of sodium, increases water absorption, promotes thirst [79,80], and stimulates aldosterone production [81]. Aldosterone limits the excretion of sodium and preserves the sodium/water balance, total sodium content, and retains water through changes in osmotic pressures [82]. ADH is released from the posterior pituitary gland into the bloodstream, inducing water retention through the kidneys, vasoconstriction of arterioles, and stimulating thirst [83,84].

Plasma proteins, in particular albumin, are also important in restoring and determining blood volume through changes in intravascular colloid osmotic pressures [38,85]. Albumin accounts for ~60–80% of the colloid osmotic pressure within the intravascular space and accrues in response to fluid regulatory stress, pulling water into the intravascular space and thus expanding blood volume [38,86]. The three main mechanisms responsible for increased intravascular albumin include increased lymphatic flow, reduced trans-capillary escape rate and increased albumin synthesis. Decreased albumin degradation after exercise may also play a role, but the slow rate of degradation (~3%/day) ensures that the net effect on

intravascular albumin content is probably trivial compared with the other mechanisms (*i.e.*, reduced degradation has likely a small effect on the total albumin lost) [87]. Given that the movement of 1 g of albumin into the intravascular space takes with it ~18 mL of water [85,88], PVE can, therefore, occur through albumin relocation or production. A reduction in ABP after exercise, referred to as post-exercise hypotension, has been shown to contribute to PVE through increasing albumin translocation into the intravascular space [89]. Post-exercise hypotension augments colloid osmotic pressure, and in turn increases fluid absorption [89]. However, post-exercise hypotension and PVE have some independence and do not necessarily occur together [90]; likely only a modest post-exercise hypotension is required to support fluid retention [91].

Given that disturbances to volume regulation stimulate the adaptations to these systems, dehydration may be an additional stressor to support adaptation. Specifically, dehydration could increase some fluid-regulating hormones (*e.g.,* aldosterone [91]), which may support fluid retention and PVE. Garrett [92] demonstrated the potential positive effect of dehydrated versus euhydrated HA that has been further supported by Berland [93] (conference abstract), and Akerman [91] who demonstrated a positive effect of dehydration and heat versus heat alone on 24 h PVE. However, three other studies have shown no added effect of dehydration versus euhydration for adaptation [30,94,95], and we are unaware of any studies showing impaired adaptation. These inconclusive findings relating to the effect of dehydration on adaptation could be due to inter-individual responses to dehydration per se [34], HA, or the capacity for PVE. Alternatively, hydration state during HA bouts may have little impact on the net outcome at least for healthy young adults (discussed elsewhere; [34])

Methods of heat acclimation

This section summarizes how different modes of HA may differentially influence PVE via the mechanisms described above.

Passive air versus passive hot-water immersion

No study, to our knowledge, has investigated the adaptive response of passive hot air versus HWI. The difference in mediums may play a role on fluid-regulating hormones, albumin, and ABP. For example, fluid-regulating hormones are likely stimulated during both conditions due to a reduction in PV (see Figure 2) and an increase in T_c. However, ADH release may be inhibited during HWI due to the hydrostatic pressure of water increasing central venous pressure, atrial stretch, and atrial natriuretic peptide release, which inhibits ADH [96–98]. Hypotension during recovery may be important in PVE (see above), and occur in either condition but likely depends on the T_c, skin temperature, or vessel stress (i.e., shear stress), as they all affect local vascular conductance. Water immersion has also been shown to reduce sympathetic activation, which supports conductance and potentially hypotension, albumin translocation from the lymphatic systems, and PVE in recovery [99,100]. Albumin synthesis is stimulated partly by increased temperature, and given that HWI conducts thermal energy and inhibits evaporation to a greater extent than passive hot air, one could expect larger albumin-mediated PVE in hot water. Whether these adaptive responses are different when matched for thermal strain has yet to be determined. On the other hand, the lymphatic flow of albumin depends partly on decreased central venous pressure, which may not occur when immersed in water (i.e., with increased central venous pressure). Thus, the comparisons between methods of heat exposure for this specific variable (PVE), is complex given the multiple integrative pathways involved in fluid regulation. Direct comparisons between modes of heating, therefore, remain hypothetical and further investigation is warranted.

Passive versus active

The comparison between passive and active modes of heat exposure is complex, and only one study, to our knowledge, has presented the adaptive differences between exercise (in a temperate environment) and passive hot air [70]. The metabolic heat production associated with exercise increases heat production, which, when combined with a high ambient temperature (and/or humidity), increases T_c more so than temperate exercise alone. In comparison to passive heat exposure, the combination of hot air and metabolic heat production elicits a faster rise in T_c than hot air alone. However, the thermal impulse associated with hot water,

rather than hot air, may be larger than exercise in a temperate environment [101], but the comparison to exercise in hot air is undetermined. Exercise could provide a greater stimulus for centrally- and peripherally mediated vasodilation, and thereby support lower ABP in recovery, but whether the magnitude of this stimulus is similar to passive exposure appears to be unknown. Further research is required to fully understand the differences between these methods of HA during and after the heat stress itself, especially when thermal strain is matched.

Figure 3 below highlights the different modes of heat stress that are regularly employed for HA, and the different dosing parameters that typically accompany them (*e.g.*, intensity and duration) [61]. Some studies have utilized combined-mode HA, but none appear to have compared modes of HA on PVE, or any other adaptive response. To our knowledge, only Convertino [70] have compared exercise in temperate conditions to resting in hot air (between- rather than within-participants), and demonstrated significantly larger PVE following exercise.

In *lieu* of direct comparisons, between-study comparisons are possible. Considerable heterogeneity between studies makes this process difficult, however available data related to PVE and heat exposure methods are presented in Figure 4 below. While Figure 4A does not account for the considerable variability between studies (i.e., is not a meta-analysis), it provides the first quantitative assessment of PV responses to each of the four common modes of applying heat stress. Figure 4B extends these generalized data, by standardizing for duration and number of sessions to more appropriately compare modes of HA. Exercise is also presented in Figure 4 to highlight the role that endogenous factors alone have on PVE, and provide a valid comparison for HA-induced changes in PV. Included studies [22,25,28,30,31,57,70,72,73,85,88–92,94,102–124] were standardized for duration using a simple linear 60-min protocol (*i.e.*, PV changes were divided by hours of exposure), notwithstanding that a linear correction may be, and is likely, inaccurate. This process is therefore warranted to provide some parity between protocols, but also theoretically appropriate given lack of sufficient information to inform the characteristics of a non-linear relation between exercise/heat dose and magnitude of PVE.

Figure 4B therefore improves the validity of the comparisons between different modes, but there are still remaining sources of variability that could significantly impact PVE and its regulatory mechanisms. For instance, HA protocols likely vary with respect to temperature, humidity, exercise intensity (or T_c rise), participant training status, previous heat exposure, and participant ethnicity. The roles that some of these factors have on HA are highlighted elsewhere [60,125]. Therefore, Figure 4 comprises an initial assessment of responses to different HA modes, in the hope that future research will clarify factors associated with the variability in PVE – and its overall importance for elite performance. Figure 4 will be

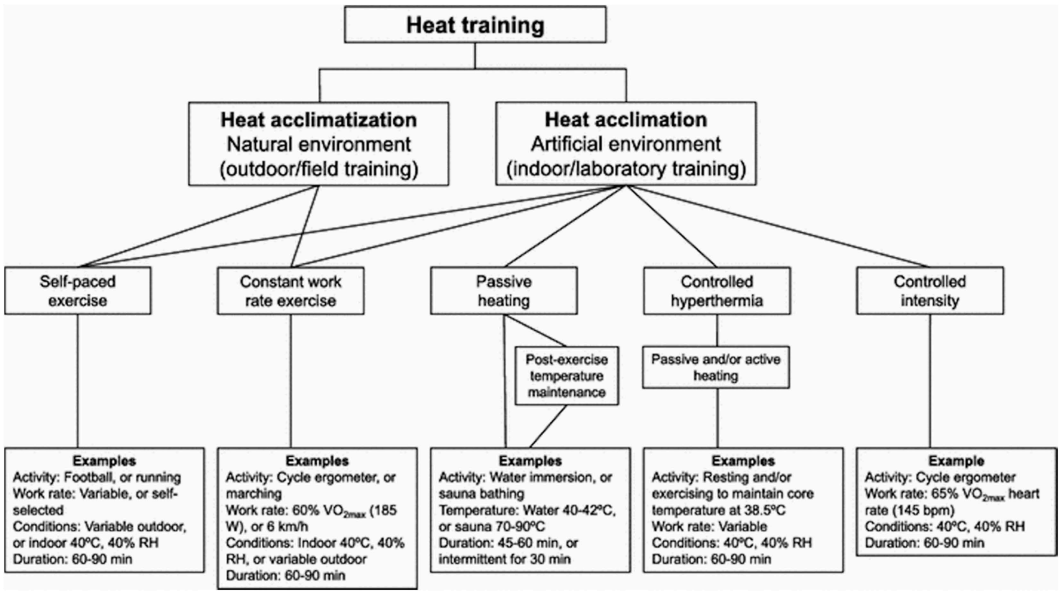

Figure 3. Schematic overview of methods for heat acclimation and heat acclimatization, with examples. RH; relative humidity, $\dot{V}O_{2\ max}$; maximal oxygen utilization. Used with permission from Daanen [125].

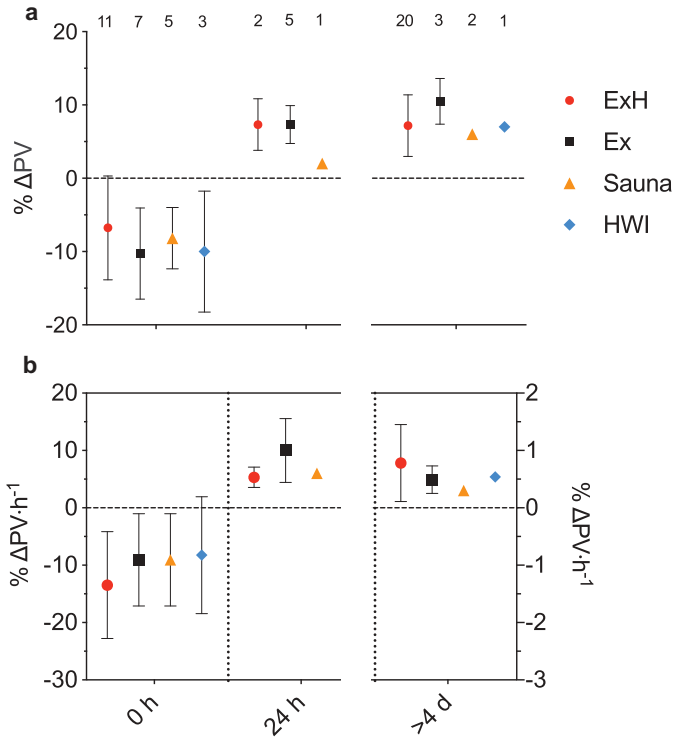

Figure 4. (A) Plasma volume (PV) changes from baseline, for exercise in heat (ExH), exercise in temperate air (Ex), passive hot-air exposure (Sauna), and hot-water immersion (HWI). PV changes are demonstrated for three periods of interest: within/immediately postexposure (0 h), at ~24 h post (24 h) and after a heat acclimation protocol of at least 4 sessions (≥4 d). (B) PV changes accounting for duration and number of sessions. The average increase in PV is compared when (linearly) controlled for total time under stress. It is likely that a non-linear relation exists, but in lieu of sufficient information to inform this relation, a linear response is appropriate. Numbers above the Figure indicate the quantity of studies represented at each datapoint. Conditions are missing when no data are available. Data presented are mean and SD.

referred to below in its relation to PVE and each mode of HA.

Comparison of heat acclimation modes

PVE can occur through multiple mechanisms, and is likely stimulated within every mode of HA mentioned within this review. The focus below is to highlight, for each mode of HA; (i) their observed effect on PVE, (ii) the mechanisms that (may) support the fluid retention, and (iii) their relation to (elite) athletic performance.

Passive hot air

Context

Passive hot air, such as sauna, is a common HA protocol and accessible to many athletes. The high temperature (*e.g.*, 50–90°C) promotes increased skin temperature and as a result, elevated thermal discomfort [126–128]. Due to the discomfort and thermal uncompensability of most saunas, studies often limit the exposure to ~30 min, or have intermittent breaks [129–132]; likely limiting the volume (~impulse) of thermal strain. These exposures to hot air can occur after exercise training [45,57], which can be time efficient and prolong the strain (both thermal and cardiovascular) from the preceding exercise session. HA via passive hot air seems unlikely to negatively affect subsequent training sessions, however, full body exposure to relatively hot environments (32°C) has been shown to impair glycogen resynthesis [133]. Thus, while the passive nature of this mode of heating will limit any excessive muscular fatigue associated with ExH (see section 4.3), the effect on recovery is still uncertain.

Mechanisms of PVE

Passive exposure to hot air is an effective means to increase the release of fluid conserving hormones.

aldosterone rises with passive hot air exposure, peaking during recovery and remaining elevated for at least 2 h [134]. Concurrently, ADH and renin are both increased during exposure and remain elevated in recovery [135]. Changes in osmolality may directly affect ADH release, therefore a high sweat rate could support increased osmolality through water loss [135]. However, ADH concentration is significantly lower in passive hot air than in exercise, even when T_c is matched [70]. Therefore, exposure to passive hot air promotes a fluid retaining response, but it may be less effective than exercise *per se*.

Albumin content is an important determinant of PVE and likely increases after exposure to passive hot air. Convertino [70] demonstrated elevated albumin concentration within the vascular space after a single exposure, however the magnitude of this change was similar to the reduction in PV, meaning that the increased albumin could be due solely to increased concentration. However, as mentioned above, an increase in albumin concentration will increase colloid osmotic pressure and fluid movement into the vascular space. Further, Convertino [70] demonstrated a significant increase in albumin content after 4 and 8 days of exercise training, but no significant change with repeated exposure to passive hot air. Likely, the metabolic requirements of exercise, beyond thermal factors *per se*, may support increased albumin synthesis [70] and this may mediate the increased albumin synthesis associated with exercise alone [87,136].

The existing literature indicates that on average HA via passive hot air induces PVE (see Figure 4, and [137]). Convertino [70] demonstrated a 5% PVE in eight healthy males across 8 days (2 h/session) of passive hot humid air exposure (42°C; 93% RH); findings that are supported by others [138–140]. Further, post-exercise hot air exposure leads to an average PVE of between 7% Scoon [45] and 18% Stanley [57], and occurs even in endurance-trained participants ($\dot{V}O_{2\ max}$: ~60 mL/min/kg [57]). Although the combination of exercise followed by hot-air exposure does not delineate the adaptive potential of hot-air exposure alone, these studies provide ecologically valid methods of HA and thus are important to highlight (similar reasoning to exercise before HWI; as discussed below).

Exercise performance

The only study to investigate exercise performance after repeated passive hot-air exposures (without prior exercise) demonstrated some benefit. Tyka [129] demonstrated that compared to a no heat control, 12 passive hot-air exposures of 3 × 15 min (separated by 4–6 min cool showers) increased total work by ~6% during an incremental test to exhaustion in 33°C, 50% RH in untrained males ($\dot{V}O_{2\ max}$:~46 mL/min/kg). Other findings come from hot-air exposures following regular exercise training sessions [45,57]. For example, in a crossover study, Scoon [45] demonstrated that running time to exhaustion improved by 32%, equating to a 1.9% improvement in 5-km time trial performance in temperate conditions after 12–13 sessions of post-exercise dry sauna bathing, relative to that after an equivalent period of control training. Conversely, Stanley [57] demonstrated no change in cycling peak power output when the test was performed 1 week after HA. The one-week delay before testing could contribute to this response. The positive association between PVE and performance benefit has been highlighted by Scoon [45] and others [26,47], and thus should be considered an important feature of the athletic heat acclimated phenotype.

Summary and recommendations

Passive hot-air is seemingly effective as a mode of HA in that it leads to a small but variable increase in PV (5–8% [70,139]; see Figure 4), which may be increased when completed immediately after exercise. HA with post-exercise hot-air exposure may also improve endurance performance, and this may be associated with the magnitude of PVE [26,45,47], however causation is not currently established. Passive hot-air exposure may be useful after exercise sessions where T_c is already elevated, when training quality is to be maintained, or in athletes such as skill/power-based (Table 1) who may benefit from being exposed to hot environments, *e.g.*, to practice the skill in conditions in which they will compete. However, as mentioned above, exposure to hot environments could impair glycogen resynthesis and thus muscle recovery after exercise [133], which may be an issue to consider during HA if metabolic recovery is an important factor.

Passive hot-water immersion

Context

Spa baths/pools are commonly available in many westernized countries, but are seldom investigated as a mode of HA. Heat is transferred more readily (conduction and convection) in water, while evaporation is limited, which collectively supports a faster rate of heat gain versus passive hot air exposure (as discussed in section 3). HWI has been used before and after exercise training [54–56,58,59], and like post-exercise hot-air exposure, this allows for prolonged thermal and cardiovascular strain and is a time-efficient alternative to HWI alone. Post-exercise HWI may also support muscle recovery for subsequent training sessions [141], as the local application of heat (*e.g.*, lower-limb immersion) has been shown to reduce delayed onset muscle soreness [142]. Thus, local application of heat stress may enhance the endurance phenotype through a range of pathways [35]. For instance, local heat exposure (applied with heat pads) can also support glycogen resynthesis [143] and this could occur with local HWI (e.g., lower-limb immersion), but not full body exposure (as discussed above) [133]. Further, HWI alone or after exercise likely avoids any impact on the quality of exercise in subsequent training sessions, and could, therefore, be considered a practically useful mode of heat exposure.

Mechanisms of PVE

As mentioned above, water immersion increases the release of atrial natriuretic peptide [144] which has an inhibitory effect on fluid retaining hormones [145–149], and it is also a diuretic and vasodilator. In HWI of 38°C, ADH was increased by 210 min of exposure but the rise in aldosterone did not occur at 30 or 210 min of immersion, but was evident 30 min into recovery [122]. Thermoneutral water immersion likely has an inhibitory effect on fluid conserving hormones (as mentioned above), however, this may be due to the often associated PVE during thermoneutral water immersion and its role on increasing central venous pressure, and atrial natriuretic peptide [150,151]. However, during HWI there may be a small increase in fluid conserving hormones [122], but further research is required in regard to this response and its relevance to PVE in recovery.

Limited research has investigated plasma albumin response to HWI. Hope [122] demonstrated a 7.8% or 3.3 g increase in albumin after 4 h immersed in 38.5°C saltwater. This increase has the potential to expand PV by ~60 mL [88], or ~2% [152]. However, the increase in albumin concentration is likely related to the loss of water via sweat, or immersion-associated diuresis [153]. Effects of HWI on the rate of albumin synthesis are currently unknown, so future studies might investigate its synthesis and content after HWI versus exercise to determine the roles of metabolic versus thermal factors.

As Figure 4 illustrates, repeated bouts of HWI cause PVE, but this has been examined in only one study as a singular mode of heating [71]. Bonner [71] demonstrated a 7% PVE after 13* 1-h sessions of HA in a 41°C bath located in a 40°C room. Albumin content was not reported, but a PVE of ~3% occurred following post-exercise HWI for 6 days (40 min cycling and ~40 min HWI) [54,55,59]. While this may indicate that the addition of exercise before HWI doesn't facilitate further PVE, these combined-mode studies typically involve smaller volumes of HA and cooler air temperatures than other studies [71].

Exercise performance

Exercise performance has been investigated after repeated post-exercise HWI but not HWI alone. Post-exercise HWI has led to ~4% improvement in time trial performance in 33°C heat (but not in 18°C), with concurrent reductions in physiological and subjective strain (e.g., T_c, HR, rating of perceived exertion, skin temperature) during sub-maximal exercise [54]. PVE occurring in response to HWI alone likely facilitates the improvements in performance [26,45,47]. Further, a preliminary report demonstrated that 10 days of HWI (40°C) in conjunction with cycling for 70 min per session can increase intermittent walking time by 9 min during a heat stress test in impermeable clothing [56]. Further research should investigate (i) the effect of HWI as a standalone mode of HA for performance in the heat, (ii) how this compares to other modes of HA, (iii) the dependance (or not) of HA-mediated performance improvement on exercising and resting PVE [47], especially in elite athletes.

Summary and recommendations

The role of HWI, as a mode of HA, in facilitating PVE is still relatively unknown. The only study to our knowledge to demonstrate PVE with HWI alone elicited a 7% PVE, however further research is required to substantiate this relation. Practically, HWI provides a time-efficient and accessible mode relative to passive hot-air exposure, and a less muscle-dependent mode relative to ExH. Available research indicates the effectiveness of HWI (either alone or combined with exercise) in regard to PVE (see Figure 4 and [54,55,59,71]), however its role in elite athletes in relation to PVE or performance has not been reported. HWI may be useful in those needing to maintain subsequent training quality and those with limited equipment availability (*e.g.,* heat chamber). Further, HWI may – in some instances – replace cold water immersion as a recovery tool and also provide physiological adaptations that could support improved exercise performance in the heat.

Exercise in heat

Context

ExH is the most common mode of HA and is a potent mediator of numerous physiological adaptations, but its comparative effectiveness to passive modes of HA are scarce (discussed above). ExH requires access to environments similar to that anticipated during the critical performance, or somewhat specialized equipment to simulate the environment (*e.g.,* heat chamber). However, relatively crude equipment could be used (*e.g.,* room with heater and vapor resistant clothing to increase humidity) to facilitate conditions more comparable to those in the heat [154]. Wearing additional clothing during normal training sessions may have no positive effect on heat adaptation [155], but it may provide an easy and cheap alternative should more specialized access not be possible.

ExH increases T_c and skin temperature, and therefore gradually augments cardiovascular, fluid regulatory, and metabolic strain. The heat stress is primarily metabolic, with the environment either impairing its offload or providing further gain. The most aerobically conditioned individuals may tolerate the workload required to rapidly raise T_c (+1.5°C in 20–30 min), however this may not be the case in those with lower aerobic power (*e.g.,* team sport athletes). Whether ExH should be incorporated into elite training programs prior to competition has yet to be determined; this is likely dependent on the specific goals of the training period, the magnitude of deterioration in performance expected, and individual responses to training in the heat (see Table 2). The consideration of whether (or in what circumstances) HA *should* be implemented prior to elite performance, rather than merely whether it *could* be, is often neglected.

Mechanisms of PVE

HA via ExH elicits PVE between ~4–18%, i.e., substantial variation has been reported (see Figure 4 and [73,92,94,103,106,107,113,114]). The combination of stressors likely facilitate these outcomes via large increases in fluid retaining hormones [70,106] that support PVE during recovery. Of note, aldosterone concentration increases during ExH [25,73,92,94,102,105,106,110,156,157], and typically returns to baseline within 12 h of exposure [102], compared with ~3 h after temperate exercise [85,117]. The magnitude of these responses is also mediated by external factors. For instance, in heat-acclimated males the ExH mediated release of ADH is potentiated by higher exercise intensity and hypohydration [158]. Thus, ExH substantially increases the stimulus for fluid-retention and likely to a greater extent than exercise or passive HA alone (see Figure 4), however further studies comparing passive and active HA to exercise alone are required to confirm this hypothesis.

Similar to HWI, atrial natriuretic peptide concentration increases with exercise, either in the heat [106,159] or in temperate environments [160,161]. The increase in atrial natriuretic peptide is likely due to increased HR (repetitive atrial stretch), catecholamines, and blood temperature [159,162]. However, the role of atrial natriuretic peptides on inhibition of PVE appears to be trivial in the context of a maximally stimulated fluid-retaining response. ExH also increases albumin content and concentration [70,72,106,113]. The initial increase may be due to a slower albumin transcapillary escape rate and higher lymphatic flow, but further increases after 24 h are more likely due to increased protein synthesis [70,163]. Plasma albumin content is increased across HA regimes of 8–19 days, and by an extent that explains nearly all of the PVE [106,107,113,164].

Table 2. Factors to consider when determining the protocol of heat acclimation.

Heat acclimation (HA) modes		Exercise in the heat	HWI	HWI after exercise	Passive hot air	Passive hot air after exercise
Considerations	**Adaptive potential**	Likely largest stimulus for adaptation (for complexity, systems, impulse)	Large thermal stimulus		Difficult to achieve air temperature sufficient to drive Tc strongly (unless sauna), but warm Tsk may aid cardiovascular effects	
		Validity in elite, i.e., already adapted?	Little evidence	Prolong stimulus or alter adaptive stimulus from preceding exercise?		Prolong stimulus or alter adaptive stimulus from preceding exercise?
	Quality and quantity of regular training	Absolute intensity of training reduced beyond first ~15–20 minutes. Factor in to total training volume	Likely little effect due to passive nature	Maintain regular training	Likely little effect due to passive nature	Can maintain regular training
		Vs. regular training? When/how to periodise?	Effect on training quality when engaging in HA and training on same day?	Effect on quality of subsequent training? Impact previous adaptive stimulus?	Effect on training quality when engaging in HA and training on same day?	Effect on quality of subsequent training? Impact previous adaptive stimulus?
	Time efficiency	Large heat production	Strong conduction of heat so potent thermal stimulus	Can combine with regular training	Weaker thermal stimulus (unless sauna), so increased duration required	Can combine with regular training
		Vs. regular training? Especially considering likely financial and other burdens (e.g., environmental)		Relative benefit vs. other recovery modes (e.g., sleep/ nutrition/cold)		Relative benefit vs. other recovery modes (e.g., sleep/ nutrition/cold)
	Recovery	May cause muscular fatigue	Could promote muscle recovery	Could promote muscle recovery	Little evidence	Potential for impaired glycogen resynthesis
				Alter adaptive stimulus from preceding exercise?	Potential for impaired glycogen resynthesis	Alter adaptive stimulus from preceding exercise?

The considerations a practitioner must take into account when designing and implementing a heat acclimation program are highlighted with respect to the most common modes of heat acclimation. The first row (color designation) corresponds to the evidence available to suggest implementing the mode of heat acclimation, in the context of the given training/athletic consideration. Black, gray, and white correspond to highly useful, moderately useful, or not useful, respectively. The assessments are made based on the information in the rows below, and the evidence presented in Figure 4 and throughout the review. The first row highlights the positive and/or negative elements of the given heat acclimation mode for the given consideration, respectively. The second row provides notes for future research considerations. The table is intended as a brief practical guide and summary of evidence, as some caveats exist: i) The importance of the above adaptations are based on the physical requirements of the sport and the physiological adaptations that would enhance performance within hot and humid environments; ii) the training foci during this period may likely change and the individual situation must be considered; and iii) the effect each mode of heat acclimation has on the training foci (e.g., exercise in the heat impairing quality of the following training session) has not currently been investigated and the current perspectives are based on preliminary understandings of these modes; v) the mode of heat acclimation used will depend on factors additional to those mentioned above (e.g., time, equipment) [170], but mentioned throughout the review. Tc, core temperature.

As mentioned previously, metabolic factors associated with exercise are important for the increased albumin content [70] through increased synthesis [87,136].

Exercise performance

ExH as a mode of HA, can improve endurance performance in the heat compared to a temperate control [22,23,25] but this is not always the case when compared to pre HA [28]. Similarly, intermittent sprint performance has been shown to be improved versus pre HA [24], but also have no effect when compared to a temperate control [25]. To effectively demonstrate the impact of HA on performance, control conditions must provide an ecologically valid comparison. Historically, the few studies including control conditions have matched for external workload [23,25,103] but cannot account for strain (*e.g.,* HR, rating of perceived exertion, glycogen depletion) – although this is not always the case [29–31], these studies do not include elite endurance athletes. In meta-analytical approaches, longer HA protocols are associated with larger effects on performance in the heat [165], however the interpretation of these findings is problematic given high heterogeneity and high risk of bias associated with such studies. Understanding how this mode of HA affects elite athletes of different sports requires further research to properly inform an empirical basis for its use in this population.

Summary and recommendations

ExH is likely a potent mode of HA for PVE and other heat-related adaptations [70], however its superiority over other forms of HA requires further investigation. Collating available evidence indicates that it has the potential to elicit larger PVE than other modes (see Figure 4), but this is a preliminary analysis that must be substantiated. ExH is also likely associated with the most muscular fatigue, which could impair concurrent training sessions in a period where training quality may be vital (e.g., taper). Any benefits of ExH are likely due to the combined metabolic and external heat stress, which may be particularly important for those who are not aerobically trained (e.g., skill/power-based), and the familiarization to exercising in an environment they will compete in (e.g., runners, cyclists or triathletes). However, to limit the effect of fatigue within a large-volume HA protocol, ExH could be interspersed with passive heat to maintain training quality and adaptive potential. Alternatively, concurrent heat mitigation [166] and recovery strategies [69,167,168] may be employed alongside HA, however there is currently limited evidence to support these approaches for elite athletes.

Overall summary and conclusion

No study has compared the adaptive differences between ExH, HWI or passive-hot air (i.e., the most common modes of HA), thus the ability to directly compare conditions is difficult. Figure 4 summarizes the existing literature and allows for some insights into the difference, or lack of, in adaptive potential between these modes of HA. The above modes, as highlighted in Figure 4, have the potential to promote PVE, however the current literature indicates no clear advantage of either mode when the volume of HA is taken into account (Figure 4B). Thus, further research is required to compare these different modes of HA on PVE and other adaptations of HA.

When deciding on a HA protocol, other factors, aside from the adaptive potential, are likely to be important. Table 2 highlights our recommendations for HA, *in the context of PVE*, and illustrates some of the potential considerations for researchers, coaches, and athletes. These recommendations are based on i) the adaptations in PV likely required to support endurance performance, or any sport with a substantial cardiovascular load (also see Table 1), and ii) factors that are important to consider during this period of training. Table 2 provides an overview of several factors to consider when determining the mode of HA, but these are not exhaustive. Some clear caveats are highlighted, although many of these considerations are not novel with respect to the HA literature (see [165]). In particular, we highlight the limited research on HA in elite athletes, the lack of control groups/conditions, and expected rates (i.e., volume of HA) of adaptation being based on the average responses [60]; these will differ due to individual factors (e.g., aerobic fitness [66], sex [68], previous heat exposure [67]), and likely the mode of HA used. The recommendations of Table 2 (and throughout the manuscript), alongside other relevant reviews and perspectives [46,60,125,137,165,166,169,170], can help inform a practitioner, providing a framework for understanding how to design a preparation strategy that is specific to the requirements (and external demands) on the athlete.

Abbreviations

WBGT	Wet Bulb Globe Temperature
RH	Relative Humidity
°C	Degrees Celcius
HA	Heat Acclimation
PV	Plasma Volume
PVE	Plasma Volume Expansion
HR	Heart Rate
T_c	Core Temperature
$\dot{V}O_{2\ peak}$	Peak Oxygen Consumption
H	Rate of Heat Production.
ExHAldo	Exercise in the Heat Aldosterone
RAAS	Renin Angiotensin Aldosterone System
HWI	Hot Water Immersion
ADH	Anti-Diuretic Hormone
AngII	Angiotensin II
ANP	Atrial Natriuretic Peptide
BV	Blood Volume
C_{Na}	Sodium Clearance
ECFV	Extracellular Fluid Volume
EPO	Erythropoietin
GFR	Glomerular Filtration Rate
RCM	Red Cell Mass
SNSA	Sympathetic Nervous System Activity

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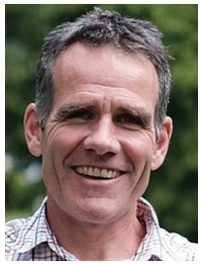
Notes on contributors

Lorenz S. Kissling is a recent Master of Physical Education graduate from the University of Otago. Lorenz is currently based at High-Performance Sport New Zealand as an Assistant Performance Physiologist. Lorenz's main research interests are in thermoregulation and athletic performance.

Ashley P. Akerman is a Postdoctoral Research Fellow at the Human and Environmental Physiology Research Unit at the University of Ottawa. Ashley completed his PhD and previous postdoctoral positions at the University of Otago, and his research interests are the determinants of cardiovascular and autonomic responses to exercise, heat, and orthostatic stress.



James D. Cotter is a Professor of Exercise and Environmental Physiology at the University of Otago. Jim's main research interests are in the separate and interactive effects of stressors within exercise (especially heat, dehydration, and orthostatic), acutely and chronically.

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