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SYSTEMATIC REVIEW

The Effects of Heat Adaptation on Physiology, Perception and Exercise Performance in the Heat: A Meta-Analysis

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Abstract

Background Exercise performance and capacity are impaired in hot, compared to temperate, conditions. Heat adaptation (HA) is one intervention commonly adopted to reduce this impairment because it may induce beneficial exercise performance and physiological and perceptual adaptations. A number of investigations have been conducted on HA but, due to large methodological differences, the effectiveness of different HA regimens remain unclear.

Objectives (1) To quantify the effect of different HA regimens on exercise performance and the physiological and perceptual responses to subsequent heat exposure. (2) To offer practical HA recommendations and suggestions for future HA research based upon a systematic and quantitative synthesis of the literature.

Data Source PubMed was searched for original research articles published up to, and including, 16 February 2016 using appropriate first- and second-order search terms.

Study Selection English-language, peer-reviewed, full-text original articles using human participants were reviewed using the four-stage process identified in the PRISMA statement.

Data Extraction Data for the following variables were obtained from the manuscripts by at least two of the authors: participant sex, maximal oxygen consumption and age; HA duration, frequency, modality, temperature and humidity; exercise performance and capacity; core and skin temperature; heart rate, stroke volume, cardiac output, skin blood flow, sweat onset temperature, body mass loss, sweat rate, perception of thirst, volitional fluid consumption, plasma volume changes; sweat concentrations of sodium, chloride and potassium; aldosterone, arginine vasopressin, heat shock proteins (Hsp), ratings of perceived exertion (RPE) and thermal sensation.

Data Grouping Data were divided into three groups based upon the frequency of the HA regimen. Performance and capacity data were also divided into groups based upon the type of HA used.

Data Analyses Hedges' *g* effect sizes and 95 % confidence intervals were calculated. Correlations were run where appropriate.

Results Ninety-six articles were reviewed. The most common duration was 7–14 days and the most common method of HA was the controlled work-rate approach. HA had a moderately beneficial effect on exercise capacity and performance in the heat irrespective of regimen; however, longer regimens were more effective than shorter approaches. HA had a moderate-to-large beneficial effect on lowering core body temperature before and during exercise, maintaining cardiovascular stability, and improving heat-loss pathways. Data are limited but HA may reduce oxygen consumption during subsequent exercise, improve glycogen sparing, increase the power output at lactate threshold, reduce lactate concentrations during exercise, have a trivial effect on increasing extracellular concentrations of Hsp, and improve perceived ratings of exertion and thermal sensation.

Conclusion HA regimens lasting <14 days induce many beneficial physiological and perceptual adaptations to high ambient temperatures, and improve subsequent exercise performance and capacity in the heat; however, the extent of the adaptations is greatest when HA regimens lasting

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longer than 14 days are adopted. Large methodological differences in the HA literature mean that there is still uncertainty regarding the magnitude and time course of potential adaptation for a number of physiological and perceptual variables.

Key Points

HA protocols lasting <7 days (short-term HA) are sufficient to induce adaptations in core body temperature and heart rate, but protocols should last at least 14 days (long-term HA) if sweat response adaptations are desired.

Short-term and medium-term HA interventions can improve exercise capacity and performance, but protocols longer than 14 days (long-term HA) are required to maximise improvements.

Exercise intensity must be monitored to ensure that the thermal impulse is sufficient to ensure that physiological adaptation continues to occur. Due to the response of heart rate (HR) to HA, using HR may be a practical way to ensure an appropriate physiological strain is maintained.

1 Introduction

1.1 General Introduction

Exercise in hot ambient temperatures places the body under a greater physiological strain than exercise of the same intensity in temperate conditions and, as a result, exercise performance is often impaired in the heat [1]. Increased body temperatures have been directly linked to cardiovascular, neuromuscular, metabolic, immunological and perceptual alterations that can cause premature fatigue [2], and thus a number of countermeasures designed to attenuate the rate at which hyperthermia develops have been investigated. Such countermeasures include fluid replacement strategies [3], cooling interventions before or during exercise [4] and heat adaptation (HA). Hydration strategies and cooling may be effective acute interventions [3–5]; but a recent consensus statement suggested that HA is the "most important intervention one can adopt to reduce physiological strain and optimize performance (during training and competition in the heat)" [6].

Short-term HA protocols (≤7 heat exposures (STHA)) can offer a physiological and performance benefit [7]; however, longer regimens are often advocated, and a recent review suggested that protocols involving 8–14 (medium-term HA (MTHA)) heat exposures are more effective than those involving less than 7 [1]. Recently, Guy et al. [1] conducted a brief review of the HA literature using a "representative sample of relevant research papers"; however, the authors stopped short of conducting a full meta-analysis. Over the last century, a variety of HA regimens have been investigated differing in the number of heat exposures, duration of heat exposure, heat stress intervention and participant group, and so the effect of HA on reducing physiological and perceptual stress and/or improving exercise performance has proved difficult to establish, and the optimal approach is still unknown.

The current meta-analysis will systematically review the HA literature and quantify the effect that HA has on physiological and perceptual responses to heat exposure, and subsequent exercise performance and capacity in a hot environment. Recent review articles have summarised the physiological adaptation to heat [8, 9], physiological adaptation and maladaptation to heat [10], and the epigenetic response to heat exposure [11], but a brief overview of the physiological responses is appropriate here to provide context.

1.2 Human Adaptation to Heat

If homeostasis is regularly disturbed by the repeated presence of heat stress that is sufficient to cause repeated periods of physiological strain [9], morphological, chemical, functional and genetic alterations can occur to reduce the physiological strain experienced during future exposures to heat stress. Thermal adaptation requires a series of sufficiently overloading thermal impulses (i.e. changes in body temperature over time) which exceed an adaptation threshold and facilitates adaptation growth within the accommodation reserve range [9]. If the impulse is insufficient adaptation will either not occur or it will occur suboptimally. The two main forms of heat adaptation are known as heat acclimation, which refers to artificially induced heat adaptation (e.g. using a laboratory-based environmental chamber), and heat acclimatization, which refers to adaptations which occur as a result of natural exposures (e.g. warm-weather training camps or domestic exposure to elevated ambient temperatures) [12].

While changes in body temperature probably receive the most attention in the HA literature, Taylor [9] highlighted how integrated the physiological responses to heat exposure are. As an example, a reduced body temperature during rest and exercise is a key variable associated with successful HA, but reductions in mean body temperature require alterations in the water loss, sweat rate (SR) and/or skin blood flow (SkBF) responses, and alterations in these would not be possible if not for the involvement of the

kidneys and sweat glands operating under the control of the medulla oblongata and hypothalamus, respectively. Sawka et al. [10] suggested that the "four classic markers of heat acclimation" were a lower heart rate (HR), a lower core body temperature (T_{core}), a higher SR and an improved exercise performance in hot conditions but the model produced by Taylor [9] identified seven key variables (HR, stroke volume (SV), sodium loss, urine loss, water loss, SR, SkBF) and another five regulated variables (mean arterial pressure, central venous pressure, blood volume, plasma osmolality and T_{core}) integral to complete heat adaptation [9]. Exercising in the heat places the body under greater physiological strain than exercising at the same intensity in temperate conditions and this extra strain often results in an impaired ability to exercise [13, 14]. Increased thermoregulatory strain has been shown to have many detrimental effects on the human body such as decreasing volitional muscle recruitment [15], increasing body fluid loss, increasing feelings of discomfort and disturbing the linear relationship between HR and oxygen consumption observed in temperate conditions due to increases in SkBF and subsequent reductions in SV [13]. As mentioned, Taylor [9] highlighted that complete heat adaptation requires integrated adaptations, and so an effective HA regimen would be one that positively modulated the related variables, at rest and during exercise, and attenuated the reduction in the ability to exercise in the heat.

1.3 Different Methods of Heat Adaptation (HA)

The effectiveness of any HA regimen appears dependent upon the magnitude and rate [16] of physiological change; however, despite there being a number of articles investigating heat adaptation, there is little consensus on the optimal approach.

It seems prudent to suggest that repeated bouts of identical thermal stress will not induce optimal heat adaptation because the magnitude of physiological strain will be reduced as adaptation occurs. With this in mind, it has been proposed that adaptation may be optimised by presenting the individual with a constant thermal impulse so that as adaptation occurs a physiological overload continues to be presented. This HA approach can be active or passive, and is often called "controlled hyperthermia" or the "isothermal model" and is one of three main HA types—the others being the "controlled work-rate" and "self-regulated" methods. The controlled work-rate model is perhaps the most commonly used method, especially in military settings, because of its ease of use for acclimatising multiple individuals simultaneously; however, the relative thermal load will differ among individuals. The self-regulated model also suffers from problems associated with different levels of thermal load among individuals because it allows individuals to self-select their work rate based upon their own perceived levels of discomfort caused by the heat exposure. It has been suggested that, due to difficulties in quantifying the thermal strain and ensuring a progressive overload, the controlled work-rate and the self-regulated methods may be inferior to the controlled hyperthermia model for optimising adaptation [9].

The response to different HA protocols can be influenced by the method and number of heat exposures used. While a number of articles have attempted to quantify the minimal period of time required to induce physiological adaptation to heat, our understanding is still limited. Sawka et al. [10] suggested that full adaptation in the "four classic markers of heat acclimation" (lower HR, lower T_{core}, higher SR and an improved exercise performance in hot conditions) occurs following 10–14 exposures; however, Wyndham et al. [17] reported that although complete HR and T_{core} reductions occurred within 14 exposures, SR changes did not. Adaptation appears to occur more rapidly in the first week of HA compared to the second [18–20], and it is also suggested that trained individuals may adapt more rapidly than untrained populations [21]. These observations may explain why benefits have been observed following as few as 4 heat exposures in trained athletes [22]. Recently, HA protocols have been categorised into three time-frames—STHA ($\leq$7 exposures), MTHA (8–14 exposures) and long-term HA (LTHA: $\geq$15 exposures) [7]. STHA approaches are attractive to time-short individuals and teams, and can offer a benefit for physiological strain and exercise performance in hot environments [7, 23]; however, longer HA approaches may offer greater benefits [1, 10, 19, 20].

While the number of heat exposures is important, the extent of the adaptation is also dependent upon the intensity and duration of the heat exposure [24]; however, these components of HA regimens have received less attention than the number of exposures. Taylor [9] reported that the benefit of any HA intervention will be dependent upon the magnitude of the thermal impulse and so the duration of heat exposure and/or exercise intensity may be more important than the frequency. Previously, it was suggested that successful HA requires a minimum daily heat exposure of 2 h [10], and although more recently it has been suggested that a minimum of 1 h a day is required [8], it is difficult to find data to support either claim. A wide range of intensities and durations have been used to acclimatise individuals to heat, but, as with frequency, the optimal approach is still unknown.

1.4 Aim of the Meta-Analysis

Although a number of investigations spanning almost a century have been conducted on HA, it is clear that, due to large methodological differences, there is a lot of

uncertainty regarding the magnitude and time course of potential adaptation, along with the effectiveness of different HA protocols. The primary aim of this review is to provide the most thorough and complete review to date regarding the effect of HA on physiological and perceptual responses to heat exposure and on exercise performance and capacity in a hot environment. The secondary aim of the meta-analysis is to attempt to establish the optimal HA regimen and offer practical recommendations for HA delivery and future HA research based upon a systematic review and quantitative synthesis of the literature.

2 Methods

2.1 Literature Search Parameters Used

An initial search using the PubMed database was conducted on 17 June 2014. The first-order search terms used were '*acclimation*', '*acclimatisation*', '*acclimatization*' and '*adaptation*', and these were used in conjunction with the following second-order search terms: '*heat*', '*exercise*', '*performance*', '*capacity*' and '*training*'. The PubMed database search was repeated throughout the review process. The final search was performed on 16 February 2016.

2.2 Inclusion/Exclusion Criteria

Initial filtering restricted searches to full-text articles using human participants published in English-language peer-reviewed journals. Abstracts and unpublished theses were not included. English language manuscripts were selected due to the native language of the authors and it has been reported that language-inclusive meta-analyses do not differ when estimating the effectiveness of an intervention compared to language-exclusive versions [25]. When manuscripts investigated multiple interventions, e.g. HA and hydration status [26] or HA and supplementation [27], the HA data were extracted and the non-HA data were excluded.

We used the four-stage (identification, screening, eligibility and inclusion) process identified in the PRISMA statement [28] to reduce the number of initial search results. Data were extracted for the following variables from these articles: participant sex, maximal oxygen consumption and age; the duration, frequency, modality, ambient temperature and humidity of the HA regimen; exercise performance and capacity; T_{core} and skin temperature (T_{skin}); HR, SV, cardiac output; SkBF, sweat-onset temperature (and site of measurement), body mass (BM) loss, SR; perception of thirst, volitional fluid consumption, plasma volume (PV) changes, sweat concentrations of sodium, chloride and potassium; plasma or serum concentrations of aldosterone, arginine vasopressin, heat shock proteins (Hsp); ratings of perceived exertion (RPE) and thermal sensation.

2.3 Data Grouping

Data were divided into three groups based upon the frequency of the HA regimen using the criteria previously described by Chalmers et al. [7]. The three groups were STHA ($\leq$7 days), MTHA (8–14 days) and LTHA (15+ days). Performance and capacity data were also divided into groups based upon the type of HA used: controlled work rate, isothermal, fixed-duration passive, self-regulated approach and passive isothermal.

2.4 Data Analyses

Mean values, standard deviations (SD) and sample sizes were obtained from each manuscript by at least two of the authors. The same data were extracted from ten of these articles, selected at random, by three of the authors to ensure consistency between investigators. When standard error data were reported these were converted to SD. Where mean values were not stated in text, the data were visually estimated from the figures, by two or three of the authors, when possible. If exact agreement was not reached the data were excluded.

Using Review Manager (Version 5.3 Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014), effect sizes (Hedges' g) and 95 % confidence intervals (CIs) were calculated for each study and the weighted-mean estimate of the effect sizes and associated 95 % CIs were calculated to account for sample size differences. Cohen's classification of effect size magnitude was used for interpretation whereby: $g < 0.19$ = trivial/negligible effect, $g = 0.20$–0.49 = small effect, $g = 0.50$–0.79 = moderate effect and $g > 0.8$ = large effect [29]. The risk of bias was assessed using the low- to high-risk scale. To investigate the extent of any relationship between the participants' age or aerobic capacity, or the duration, frequency and ambient temperature of the HA on the magnitude of change for variables collected in at least 15 manuscripts (resting T_{core}, resting HR, SR, PV, BM loss and performance/capacity data), Spearman's correlation analyses were performed.

3 Results

3.1 Search Result Overview

Figure 1 shows the process used to select the 96 manuscripts included in this meta-analysis. The 96 articles reviewed involved a total of 1056 participants of whom at least 103 featured in more than one manuscript. Seventy-six (7 %) of the participants were female (STHA: 11 % of participants;

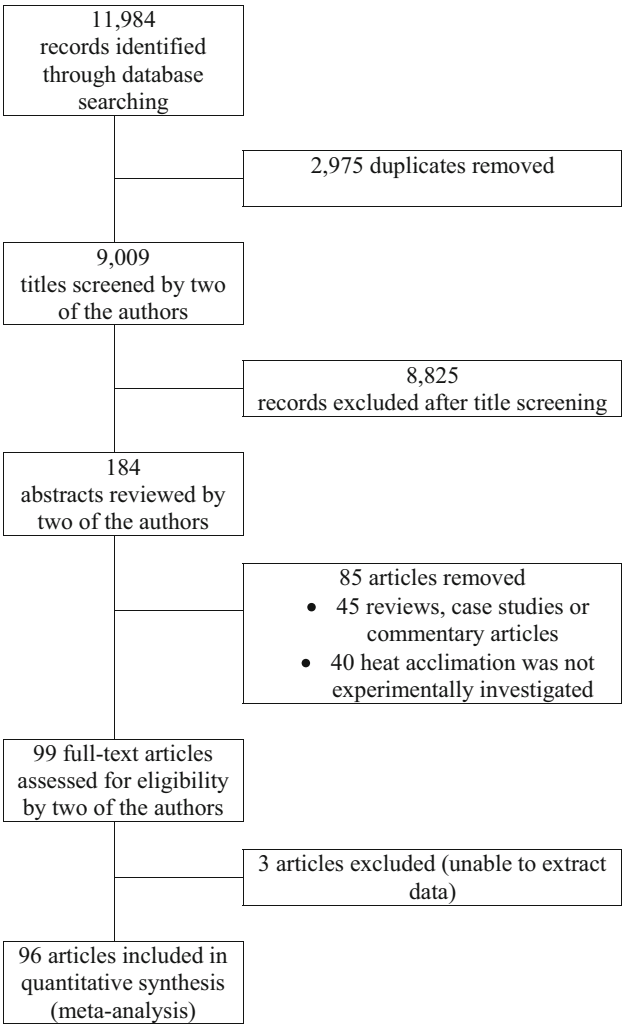

Fig. 1 Overview of the selection process used for this meta-analysis

MTHA: 3 % of participants; LTHA: 25 % of participants). Mean and median data of the HA protocols and participants are presented in Table 1 while detailed summaries of each individual study reviewed are in Tables 2, 3 and 4. The most common method of HA was the controlled work-rate approach ($n = 64$ investigations), followed by the isothermal and fixed-duration passive heat exposure approaches ($n = 11$ and 9, respectively), the self-regulated approach ($n = 7$) and the passive isothermal approach ($n = 6$). Risk of bias assessment showed that HA investigations are at high risk of bias due to a lack of blinding and, in the majority of investigations, the lack of a control group (Fig. 2).

3.2 The Effect of HA on Exercise Performance and Capacity in the Heat

The effect of HA on exercise performance and capacity data are presented in Fig. 3. Thirty-two investigations reported performance ($n = 14$) and/or capacity ($n = 18$) data; however, due to subgroups within the manuscripts, 52 data sets were analysed (performance: 26; capacity: 26). When the data were analysed collectively, HA had a moderately beneficial effect on exercise capacity and performance in the heat irrespective of regimen. Forty-seven of the 52 data sets reported an improvement greater than 1 % and regarding the overall mean and median, improvements were $+15 \pm 22$ % and +6 (0–92) %, respectively. The average percentage improvement reported was greater following LTHA [mean $+22 \pm 29$ %; median +10 (4–65) %; 4/4 data sets reporting a benefit >1 %] and MTHA [mean $+21 \pm 28$ %; median +9 (1–92) %; 24/25 benefit >1 %] than STHA [mean $+7 \pm 8$ %; median +4 (0–33) %; 13/15 benefit >1 %] regimens. There was a weak–moderate correlation between HA duration and HA ambient temperature, and the improvement in exercise performance/capacity observed ($\rho = 0.38$; $p = 0.004$; $\rho = 0.36$; $p = 0.008$, respectively), but no correlation between the improvement in exercise performance/capacity observed and maximal aerobic capacity ($\rho = 0.12$, $p = 0.25$), nor the number of HA days ($\rho = 0.17$, $p = 0.12$).

The size of the improvement observed was greater in capacity tests [mean $+23 \pm 29$ %; median: 8 (0–92) %; 23/26 benefit >1 %] than performance tests [mean $+7 \pm 7$ %; median +4 (0–33) %; 24/27 benefit >1 %].

The magnitude of effect was similar for the controlled work rate HA approach [Hedges' $g = 0.63$ (0.46–0.79); $n = 316$] and the isothermal approach [Hedges' $g = 0.63$ (0.20–1.07); $n = 44$] although both magnitudes of effect were lower than that reported from the investigations that used a self-regulated approach [Hedges' $g = 1.12$ (0.78–1.45); $n = 86$]. There were no performance or capacity data from passive isothermal investigations and only one investigation that reported performance data following fixed-duration passive HA [Hedges' $g = 0.40$ (−0.49 to 1.28)] [30].

3.3 The Effect of HA on Core Body and Skin Temperatures

Effect size data for T_{core} and T_{skin} are presented in Fig. 3. There was a moderate-to-large beneficial effect of HA on reducing resting (-0.18 ± 0.14 °C, $n = 407$), mean (-0.31 ± 0.31 °C, $n = 140$) and comparable time-point (-0.34 ± 0.24 °C, $n = 181$) T_{core}. The effect on resting values was comparable across measurement sites, although the effect size was greatest for oesophageal and gastrointestinal (rectal: -0.18 ± 0.15 °C, $n = 262$; oesophageal: -0.22 ± 0.09 °C, $n = 70$; tympanic: -0.10 ± 0.10 °C, $n = 36$; gastrointestinal: -0.12 ± 0.11 °C, $n = 21$). Effect sizes and the magnitudes of change were similar for STHA (-0.17 ± 0.12 °C, $n = 144$) and MTHA (-0.17 ± 0.14 °C, $n = 252$) protocols.

Table 1 A summary of the participants and heat adaptation regimens used in the 96 manuscripts reviewed

	Manuscripts	*N*	Maximal oxygen uptake[a] (mL/kg/min)	Age [b] (years)	Number of HA sessions[c]	Days between HA sessions[d]	Duration of HA session[e] (min)	Total duration of HA[f] (min)	Temperature of HA session[g] (°C)	Humidity of HA session[h] (%)
All	96	1056	50 ± 10	26 ± 8	9 ± 4	0 ± 0	105 ± 62	1005 ± 729	40 ± 6	40 ± 19
		(953[i])	50 (21–67)	24 (13–65)	10 (4–24)	0 (0–2)	90 (27–300)	822 (150–4080)	40 (25–55)	40 (13–100)
STHA	30	324	50 ± 10	27 ± 9	6 ± 1	0 ± 0	78 ± 52	433 ± 279	40 ± 5	42 ± 23
			50 (21–65)	25 (20–65)	6 (4–7)	0 (0–2)	60 (30–270)	360 (150–1350)	40 (30–50)	30 (18–100)
MTHA	61	643	51 ± 8	25 ± 7	10 ± 2	0 ± 0	119 ± 64	1186 ± 669	40 ± 6	37 ± 17
			51 (25–67)	23 (13–61)	10 (8–14)	0 (0–1)	90 (27–300)	945 (270–3600)	40 (28–55)	40 (13–87)
LTHA	9	102	37 ± 16	29 ± 11	17 ± 3	0 ± 1	104 ± 52	1756 ± 867	38 ± 7	47 ± 20
			37 (21–54)	25 (22–57)	17 (15–24)	0 (0–2)	90 (53–240)	1500 (970–4080)	37 (25–50)	50 (15–70)

Data are presented as mean ± standard deviation (top row) and median (range) (bottom row)

All data except for *N* are calculated after the removal of any duplicated data to better reflect the range of approaches taken. Number of participants and articles do not equal STHA + MTHA + LTHA due to sub-groups within some papers

HA heat adaptation, *STHA* short-term heat adaptation, *MTHA* medium-term heat adaptation, *LTHA* long-term heat adaptation

[a] Data reported in 67/96 manuscripts

[b] Data reported in 83/96 manuscripts

[c] Data reported in 95/96 manuscripts

[d] Data reported in 94/96 manuscripts

[e] Data reported in 95/96 manuscripts

[f] Data reported in 95/96 manuscripts

[g] Data reported in 96/96 manuscripts

[h] Data reported in 77/96 manuscripts

[i] *N* after the removal of duplicated participants caused by a number of investigations reporting data in multiple papers

There was only one LTHA data set (−0.32 °C, $n = 11$) [31]. There were no statistically significant correlations between any of the variables and the change in resting T_{core} ($\rho = -0.21$ to 0.04).

Overall, resting T_{skin} was unaffected by HA [mean 0.03 ± 0.31 °C; median 0.0 (−0.3 to 1) °C, $n = 179$]. Despite a large degree of variation between studies, there was a small negative effect size for STHA [mean −0.01 ± 0.46 °C; median −0.2 (−0.3 to 1.0) °C, $n = 73$] but neither MTHA [mean 0.05 ± 0.21 °C; median 0.0 (−0.2 to 4) °C, $n = 95$] nor the single LTHA investigation [31] reported a change (0.0 °C, $n = 11$). Despite the lack of effect at rest, there was a large effect of HA on reducing mean T_{skin} during exercise (−0.57 ± 0.49 °C, $n = 67$).

3.4 The Effect of HA on Cardiovascular Stability

HA had a moderate effect on lowering resting HR (−6 ± 5 beats/min, $n = 257$), but a large effect on reducing mean (−12 ± 9 beats/min, $n = 166$) and comparative time-point (−16 ± 6 beats/min, $n = 118$) HR. The effect of STHA (−5 ± 1 beats/min, $n = 60$) and MTHA (−5 ± 5 beats/min, $n = 156$) on lowering resting HR was similar, but data from the two LTHA investigations suggest that the effects are greater following LTHA (−12 ± 3 beats/min, $n = 35$). Despite this, there were no statistically significant correlations between any of the variables and the change in resting HR ($\rho = -0.38$ to 0.02) (Fig. 3).

The effects of HA on resting systolic (−1.4 ± 3.0 mmHg, $n = 61$), diastolic (−2.9 ± 3.4 mmHg, $n = 61$) and mean (−1.0 ± 1.8 mmHg, $n = 28$) arterial blood pressures were negligible, moderate and small, respectively. HA had a small effect on increasing SV recorded during exercise (+12 ± 16 %, $n = 36$), a small effect on resting cardiac output (+0.3 L/min, $n = 7$) [32], and a trivial effect on mean cardiac output (+0.4 L/min, $n = 12$) [33].

3.5 The Effect of HA on Sweat Responses and Markers of Fluid Balance

HA markedly reduced the T_{core} at which the sweat response commences with the mean overall onset temperature being −0.28 ± 0.21 °C lower following HA ($n = 142$). The reduction was greater, but more variable, when rectal temperature was used (−0.42 ± 0.42 °C, $n = 43$, Hedges' $g = -1.48$ (−1.99 to −0.97) than when mean body temperature (−0.22 ± 0.12 °C, $n = 72$, Hedges' $g = -0.81$ (−1.16 to −0.46) or oesophageal temperatures were recorded (−0.28 ± 0.12 °C, $n = 27$, Hedges' $g = -1.03$ (−1.61 to −0.44). HA had a small, but variable, effect on BM

Table 2 A summary of the investigations that adopted a short-term heat adaptation (≤7 days) approach reviewed in this meta-analysis

References	HA activity	HA frequency and duration	Days between	T_{amb} (°C)	RH (%)	Performance/ capacity (% Δ)	T_{core} resting/ mean (°C Δ)	T_{skin} resting/ mean (°C Δ)	HR resting/mean (b min^{-1} Δ)	Sweat rate (% Δ)	BM loss (% Δ)	Resting PV (% Δ)
Anderson and Kenney [136]	Walking or cycling @ 30 % VO_{2max}	5 × 30 min	0	48	–	–	–/–	–/–	–/–	–	–	–
	Walking or cycling @ 30 % VO_{2max}	5 × 30 min	0	48	–	–	–/–	–/–	–/–	–	–	–
Aoyagi et al. [42]	Walking/running @ 45–55 % VO_{2max}	6 × 60 min	0	40	30	0	−0.2/–0.2	–/−0.2	–/−8	–	5	–
Armstrong et al. [104]	Intermittent running @ ~68 % VO_{2max}	6 × 56 min	0	41	39	–	–/–	–/–	–/–	–	–	2
Brade et al. [43]	Intermittent cycling @ 80 % VO_{2max}. 8–12 sets	5 × 38 min	1	35	60	4	−0.2/–	−0.3/–	–/–	–	0	–
Brazaitis and Skurvydas [67]	Water immersion	7 × 45 min	1	44	–	–	−0.3/–	–/–	−5/–	–	40	–
Buono et al. [68]	Walking @ 4.8 km/h, 3 % grade, and cycling @ 75 W	7 × 100 min	0	35	75	–	−0.3/–	–/–	–/–	–	–	–
Febbraio et al. [69]	Cycling @ 50 % VO_{2max}	7 × 90 min	0	40	20	–	−0.4/–	–/–	–/−11	–	–	–
Fujii et al. [70]	Cycling @ 50 % VO_{2max}; 4 × 20 min with 10 min rest	6 × 120 min	0	37	50	–	−0.2/–	−0.2/–	−7/–	–	–	2
Fujii et al. [71]	Cycling @ 50 % VO_{2max}; 4 × 20 min with 10 min rest	6 × 120 min	0	37	50	–	−0.2/–	–/–	–/–	–	–	3
Garrett et al. [45]	Cycling. Controlled hyperthermia (target T_{core} = 38.5 °C)	5 × 90 min	0	40	60	1	−0.1/–0.1	–/–	–/−1	–	244	5
Garrett et al. [44]	Cycling. Controlled hyperthermia (target T_{core} = 38.5 °C)	5 × 90 min	0	40	60	15	−0.1/–	–/–	–/–	–	–	0
Gonzalez et al. [138]	Cycling @ 25–30 % VO_{2max}	6 × 30 min	0	40	–	–	–/–	–/–	–/–	–	–	–
	Cycling @ 25–30 % VO_{2max}	6 × 30 min	0	40	–	–	–/–	–/–	–/–	–	–	–
Houmard et al. [24]	Walking/running @ 50 % VO_{2max}	7 × 60 min	0	40	27	–	–/–	–/–	–/–	4	–	–
	Running @ 75 % VO_{2max}	7 × 35 min	0	40	27	–	–/–	–/–	–/–	4	–	–
Kenefick et al. [46]	Walking @ 1.56 m/s, 4 % gradient	5 × 100 min	0	45	20	7	–/–	–/–	–/–7	–	–	–
Kuennen et al. [27]	Walking (target T_{core} > 39 °C)	7 × 110 min	0	47	20	–	0/–	−0.2/–	−5/–	–	–	–
Neal et al. [35]	Cycling. Controlled hyperthermia (target T_{core} = 38.5 °C)	5 × 90 min	0	40	50	–	−0.2/–0.2	−0.1/–0.1	–/−7	12	7	–
Peter and Wyndham [139]	Bench stepping @ ~0.9 L/min	5 × 270 min	0	33	90	–	–/–	–/–	–/–	−8	–	–
Petersen et al. [47]	High-intensity cycling (8 × 20 s sprints with 10 s rest)	4 × 38 min	0	30	60	0	0/–	1.0/–	–/–	-8	–	–
Piwonka and Robinson [140]	Treadmill walking @ 1.56 m/s, 5 %	4 × 85 min	0	50	–	–	–/–	–/–	–/–	–	–	–
Poirier et al.[19]	Cycling at 50 % VO_{2max}	7 × 90 min	0	40	20	–	–/–	–/–	–/–	–	–	–

Table 2 continued

References	HA activity	HA frequency and duration	Days between	T_{amb} (°C)	RH (%)	Performance/ capacity (% Δ)	T_{core} resting/ mean (°C Δ)	T_{skin} resting/ mean (°C Δ)	HR resting/mean (b min^{-1} Δ)	Sweat rate (% Δ)	BM loss (% Δ)	Resting PV (% Δ)
Racinais et al. [76]	Variable football-specific training	6 × 120 min	0	41	21	–	−0.1/–	−0.3/–	−5/–	–	27	2
Racinais et al. [20]	Cycle training (unspecified)	5 × 240 min	0	34	18	10	–/−0.1	–/–	–/-3	–	−3	–
Shvartz et al. [108]	Walking @ 3.5 mph, 0 % gradient	4 × 50 min	0	40	20	–	–/–	–/–	–/–	9	–	–
	Walking @ 3.5 mph, 0 % gradient (wearing full rubber suit)	4 × 50 min	0	40	100	–	–/–	–/–	–/–	9	–	–
Shvartz et al. [109]	Bench stepping @ 15 steps/min	6 × 90 min	0	50	–	–	–/–	–/–	–/–	3	–	–
Shvartz et al. [109]	Bench stepping @ 15 steps/min	6 × 90 min	0	37	20	–	–/–	–/–	–/–	−8	–	–
Sunderland et al. [22]	Intermittent sprint exercise	4 × 38 min	2	30	24	33	0/–	–/–	–/–	−15	273	−2
Watkins et al. [84]	Cycling at 75 % VO_{2max}	7 × 30 min	0	40	27	–	−0.2/–	0/–	–/–	23	–	–
Willmott et al. [64]	Cycling at 50 % VO_{2max}	4 × 45 min	0	35	60	3	–/-0.1	–/–	–/-10	17	–	–
	Cycling at 50 % VO_{2max}	4 × 2 × 45 min	0	35	60	4	–/−0.1	–/–	–/−6	10	–	–
Yamazaki and Hamasaki [85]	Cycling at 50 % VO_{2max}	6 × 80 min	0	36	50	–	−0.1/–	–/–	−5/–	–	–	–
Zurawlew et al. [30]	Water immersion	6 × 40 min	0	40	–	5	−0.3/–	–/–	–/–	0	–	3

HA heat adaptation, T_{amb} ambient temperature, *RH* relative humidity, T_{core} core body temperature, T_{skin} skin temperature, *HR* heart rate, *BM* body mass, *PV* plasma volume, VO_{2max} maximal oxygen consumption

Table 3 A summary of the investigations that adopted a medium-term heat adaptation (8–14 days) approach reviewed in this meta-analysis

References	HA activity	HA frequency and duration	Days between	T_{amb} (°C)	RH (%)	Performance/ capacity (% Δ)	T_{core} resting/ mean (°C Δ)	T_{skin} resting/ mean (°C Δ)	HR resting/ mean (b min^{-1} Δ)	Sweat rate (% Δ)	BM loss (%) Δ	Resting PV (% Δ)
Aoyagi et al. [42]	Walking/running @ 45–55 % VO_{2max}	12 × 60 min	0	40	30	3	–/−0.4	–/–	–/−19	–	−7	–
Armstrong and Kenney [120]	Walking or cycling @ 40 % VO_{2max}	9 × 105 min	1	45	27	–	–/–	–/–	–/–	–	–	–
	Walking or cycling @ 40 % VO_{2max}	9 × 105 min	1	45	27	–	–/–	–/–	–/–	–	–	–
Beaudin et al. [65]	Resting wearing vapour barrier suit (target T_{core} = 38.5–39 °C)	10 × 120 min	0	50	20	–	−0.1/–	0.4/–	–/–	–	–	–
Bonner et al. [66]	Hot water immersion (target T_{core} = 38.5 °C)	13 × 60 min	0	41	–	–	0/–	–/–	0/–	69	12	3
Burk et al. [48]	Walking at 55–60 % VO_{2max}	10 × 100 min	0	42	18	83	−0.2/–	0/–	−5/–	–	–	–
Castle et al. [49]	Cycling 50 % VO_{2max}	10 × 60 min	0	33	52	2	−0.4/–	–/–	–/–	–	0	–
Chang and Gonzalez [141]	Walking @ 1.34 m/s, 0 % gradient	14 × 100 min	0	49	20	–		–/–	–/–	–	–	–
Cheung and McLellan [26]	Walking 3.5 km/h, 0 % gradient (high fitness group)	10 × 60 min	0	40	30	1	−0.1/–	−0.2/–	−4/–	15	15	–
	Walking 3.5 km/h, 0 % gradient (moderate fitness group)	10 × 60 min	0	40	30	5	0/–	0/–	−1/–	14	14	–
Cheuvront et al. [50]	Walking @ 1.56 m/s, 4 % gradient	10 × 100 min	0	45	20	9	–/–	–/–	–/–	6	18	–
Chinevere et al. [115]	Walking @ 1.56 m/s, 4 % gradient	10 × 100 min	0	45	20	–	–/–	–/–	–/–	–	–	–
Flouris et al. [18]	Cycling @ 50 % VO_{2max}	14 × 90 min	0	40	20	–	−0.3/–0.3	–/–	−5/−15	–	–	–
Gibson et al. [72]	Cycling. Controlled hyperthermia (target T_{core} = 39.5 °C)	10 × 90 min	–	40	41	–	−0.5/–	–/–	−18/–	48	–	15
Henane and Bittel [73]	Passive hyperthermia (target $T_{tympanic}$ = 38.2 °C)	9 × 90 min	0	45	24	–	−0.2/–	–/–	–/–		–	–
Henane and Valatx [110]	Passive hyperthermia (target $T_{tympanic}$ = 38.2 °C)	9 × 60 min	0	55	–	–	–/–	–/–	–/–	61	–	–
Hom et al. [142]	Walking @ 50 % VO_{2max}	11 × 90 min	0	33	50	–	–/–	–/–	–/–	–	–	–
Kanikowska et al. [74]	Passive hyperthermia (target $T_{tympanic}$ = 37.5 °C)	9 × 100 min	0	40	50	–	−0.3/–	–/–	−3/–	–	–	–

Table 3 continued

References	HA activity	HA frequency and duration	Days between	T_{amb} (°C)	RH (%)	Performance/ capacity (% Δ)	T_{core} resting/ mean (°C Δ)	T_{skin} resting/ mean (°C Δ)	HR resting/ mean (b min^{-1} Δ)	Sweat rate (% Δ)	BM loss (%) Δ	Resting PV (% Δ)
King et al. [51]	Cycling @ 55 % VO_{2max}	8 × 90 min	0	40	31	3–14	–/–	–/–	–/−16	–	–	–
Kirby and Convertino [111]	Cycling. Controlled hyperthermia (target T_{core} = 39.5 °C)	10 × 120 min	0	40	45	–	–/–	–/–	–/–	14	–	13
Lee et al. [32]	Cycling @ 50 % VO_{2max}	10 × 60 min	0	40	25	5	−0.3/–0.5	–/–	−11/–14	–	88	6
Lorenzo et al. [33]	Cycling @ 50 % VO_{2max}	10 × 100 min	0	40	30	8	–/–	–/–	–/–	–	22	2
Lorenzo et al. [143]	Cycling @ 50 % VO_{2max}	10 × 100 min	0	40	30	–	–/–	–/–	–/–	–	–	–
Lorenzo and Minson [34]	Cycling @ 50 % VO_{2max}	10 × 100 min	0	40	30	–	–/–	–/–	–/–	–	–	–
Magalhaes et al. [52]	Walking. Controlled hyperthermia (target T_{core} increase of 1 °C)	11 × 60 min	0	40	45	4	−0.1/0	–/−0.3	−6/−1	10	–	–
Magalhaes et al. [38]	Walking. Controlled hyperthermia (target T_{core} increase of 1 °C)	11 × 60 min	0	40	45	12	−0.3/–	−0.1/–	–/–	–	–	–
Magalhaes et al. [53]	Cycling @ 50 % VO_{2max}	9 × 60 min	0	40	32	2	−0.2/–	–/–	−9/–	2	–	–
Maher et al. [54]	Submaximal cycling (upright)	8 × 90 min	0	49	–	4	–/–	–/–	–/–	48	–	–
	Submaximal cycling (supine)	8 × 90 min	0	49	–	3	–/–	–/–	–/–	51	–	–
Martinez et al. [144]	Walking @ 40 % VO_{2max}	8 × 90 min	1	35	40	–	–/–	–/–	–/–	–	–	–
Maruyama et al. [75]	Rest (am)	10 × 240 min	0	40	40	–	−0.0/–	–/–	−1/–	–	–	–
	Rest (pm)	10 × 240 min	0	40	40	–	−0.2/–	–/–	−3/–	–	–	–
McClung et al. [36]	Walking @ 1.56 m/s, 4 % gradient	10 × 85 min	0	49	20	20	–/–	–/–	–/–	19	–	–
Mitchell et al. [107]	Cycling @ 40–50 % VO_{2max}	10 × 240 min	0	45	42	–	–/–	–/–	–/–	–	120	–
Moran et al. [145]	Walking @ 1.56 m/s, 5 % gradient	10 × 140 min	0	49	20	–	–/–	–/–	–/–	–	–	–
	Walking @ 1.56 m/s, 5 % gradient	10 × 140 min	0	49	20	–	–/–	–/–	–/–	–	–	–
Nadel et al. [146]	Cycling @ 50 % VO_{2max}	10 × 60 min	0	45	–	–	–/–	–/–	–/–	–	–	–
	Cycling @ 50 % VO_{2max}	10 × 60 min	0	36	–	–	–/–	–/–	–/–	–	–	–
Nielsen et al. [55]	Cycling @ 50 % VO_{2max}	11 × 90 min	0	41	13	67	–/–	–/–	–/–	–	–	3

Table 3 continued

References	HA activity	HA frequency and duration	Days between	T_{amb} (°C)	RH (%)	Performance/ capacity (% Δ)	T_{core} resting/ mean (°C Δ)	T_{skin} resting/ mean (°C Δ)	HR resting/ mean (b min^{-1} Δ)	Sweat rate (% Δ)	BM loss (%) Δ	Resting PV (% Δ)
Nielsen et al. [56]	Cycling @ 45 % VO_{2max}	11 × 49 min	0	35	87	17	−0.3/–	–/–	–/–	26	–	9
O'Toole et al. [147]	Cycling @ 46 % VO_{2max}	10 × 120 min	0	40	50	–	–/–	–/–	–/–	–	–	–
Pandolf et al. [57]	Walking @ 1.56 m/s, 5 % gradient	10 × 140 min	0	49	20	31	–/–	–/–	–/–	–	–	–
	Walking @ 1.56 m/s, 5 % gradient	10 × 140 min	0	49	20	0	–/–	–/–	–/–	–	–	–
Poh et al. [95]	Walking @ 1.56 m/s, 5 % gradient	10 × 90 min	0	33	40	–	–/–	–/–	−8/–	–	–	–
Poirier et al. [19]	Cycling @ 50 % VO_{2max}	14 × 90 min	0	40	20	–	–/–	–/–	–/–	–	–	–
Racinais et al. [88]	Australian Rules training (unspecified)	14 × 90 min	0	31	44	–	–/−0.1	–/−0.5	–/−10	–	–	5
Racinais et al. [20]	Cycle training (unspecified)	13 × 240 min	0	34	18	16	–/–	–/–	–/−1	–	−10	–
Radakovic et al. [148]	Walking at 5.5 km/h + rest	10 × 180 min	0	35	40	–	–/–	–/–	–/–	–	–	–
	Rest	10 × 180 min	0	35	40	–	–/–	–/–	–/–	–	–	–
Regan et al. [77]	Cycling. Controlled hyperthermia (target T_{core} = 38.5 °C)	10 × 60 min	0	38	40	–	0/–	–/–	–/–	9–27	−5	–
Robinson et al. [112]	Walking @ 1.56 m/s, 4–5.6 % gradient	9 × 70 min	0	40	–	–	–/–	–/–	–/–	12	–	–
Rowell et al. [89]	Walking @ 3.5 m/h, 0 % gradient	12 × 73 min	0	48	–	–	–/−1.3	–/−1.4	–/−32	–	–	–
Saat et al. [78]	Rest, cycling @ 60 % VO_{2max}, and rest	14 × 90 min	0	31	70	–	−0.2/–	0.2/–	3/–	–	2	3
Schmit et al. [59]	Triathlon training	8 × ~105 min	0	30	74	4	–/–	–/–	–/–	–	–	16
Senay [79]	Bench stepping @ ~0.9 L/min	8 × 240 min	0	33	–	–	–/–	–/–	–/–	–	–	–
	Bench stepping @ ~0.9 L/min	8 × 240 min	0	33	–	–	–/–	–/–	–/–	–	–	–
Senay [80]	Bench stepping @ 35 W	12 × 240 min	0	33	–	–	−0.1/–	–/–	–/–	–	4	0
Senay et al. [100]	Cycling @ 40–50 % VO_{2max}	10 × 240 min	0	33	–	–	–/–	–/–	–/–	–	–	–
Shido et al. [82]	Rest (am)	10 × 240 min	0	42	40	–	−0.0/–	0.4/–	−4/–	–	–	–
	Rest (pm)	10 × 240 min	0	42	40	–	−0.2/–	−0.1/–	−6/–	–	–	–

Table 3 continued

References	HA activity	HA frequency and duration	Days between	T_{amb} (°C)	RH (%)	Performance/ capacity (% Δ)	T_{core} resting/ mean (°C Δ)	T_{skin} resting/ mean (°C Δ)	HR resting/ mean (b min^{-1} Δ)	Sweat rate (% Δ)	BM loss (%) Δ	Resting PV (% Δ)
Shido et al. [81]	Rest (am)	10 × 240 min	0	46	20	–	–/–	0/–	−2/–	–	–	−1
	Hot water-immersion (leg)	10 × 60 min	0	42	–	–	–/–	0/–	−3/–	–	–	3
Shin et al. [83]	Hot water-immersion (lower body)	10 × 27 min	1	42	–	–	−0.1/–	–/–	–/–	–	14	–
Shvartz et al. [149]	Bench stepping @ 12 steps/min	8 × 240 min	0	34	–	–	–/–	–/–	–/–	–	–	–
	Rest	8 × 240 min	0	34	–	–	–/–	–/–	–/–	–	–	–
Stearns et al. [150]	Walking @ 3.5 m/h	10 × 90 min	0	33	40	–	–/–	–/–	–/–	–	–	–
Strydom et al. [151]	Bench stepping @ 12 steps/min	12 × 300 min	0	36	40	–	–/–	–/–	–/–	–	–	–
Vesic et al. [152]	Walking @ 5.5 km/h + passive heat exposure	10 × 180 min	0	35	40	–	–/–	–/–	–/–	–	–	–
	Passive heat exposure	10 × 180 min	0	35	40	–	–/–	–/–	–/–	–	–	–
Voltaire et al. [58]	Daily training	14 × ? min	0	33	78	4	−0.1/–	–/–	–/25	55	71	–
Wagner et al. [41]	Walking @ 5.6 km/h	8 × 70 min	0	49	–	80	–/–	–/–	–/–	–	–	–
	Walking @ 5.6 km/h	8 × 84 min	0	49	–	34	–/–	–/–	–/–	–	–	–
	Walking @ 5.6 km/h	8 × 85 min	0	49	–	14	–/–	–/–	–/–	–	–	–
Wyndham et al. [101]	Cycling at 40–50 % VO_{2max}	10 × 300 min	0	36	40	–	–/–	–/–	–/–	–	–	–
Yamada et al. [37]	Walking/running @ 56 % VO_{2max}	10 × 100 min	0	43	28	92	–/–	–/–	–/–	–	–	3
Yeargin et al. [86]	Pre-season training	8 × 120 min	0	28	78	–	−0.0/–	–/–	–/–		−14	–

HA heat adaptation, T_{amb} ambient temperature, *RH* relative humidity, T_{core} core body temperature, T_{skin} skin temperature, *HR* heart rate, *BM* body mass, *PV* plasma volume, VO_{2max} maximal oxygen consumption

Table 4 A summary of the investigations that adopted a long-term heat adaptation (15+ days) approach reviewed in this meta-analysis

References	HA activity	HA frequency and duration	Days between	T_{amb} (°C)	RH (%)	Performance/ capacity (% Δ)	T_{core} resting/ Mea (°C Δ)	T_{skin} resting/ Mea (°C Δ)	HR resting/mea (b min^{-1} Δ)	Sweat rate (% Δ)	Δ BM loss (% Δ)	Resting PV (% Δ)
Armstrong et al. [40]	Aerobic circuit @ 50–70 % VO_{2max}	24 × 90 min	2	37	35	65	–/–0.4	–/–1.1	–/–24	–	–	–
Buskirk et al. [153]	3 × 30 min walking with 2 × 30 min rest	15 × 150 min	0	49	–	–	–/–	–/–	–/–	–	–	–
Griefahn [61]	Treadmill walking @ 4 km/h (warm and humid)	15 × 100 min	0	37	70	–	–/−0.3	–/–	−10/–	–	40	–
	Treadmill walking @ 4 km/h (hot and dry)	15 × 100 min	0	50	15	–	–/−0.4	–/–	−14/–	–	15	–
	Treadmill walking @ 4 km/h (radiant heat)	15 × 100 min	0	25	40	–	–/−0.5	–/–	15/–	–	3	–
Marcus [106]	Treadmill. Controlled hyperthermia (target T_{core} = 38.3 °C)	17 × 60 min	0	40	–	–	–/–	–/–	–/–	–	64	–
Ormerod et al. [124]	Submaximal circuit training	18 × 90 min	1	36	60	–	–/–	–/–	–/–	–	–	–
Patterson et al. [105]	Cycling. Controlled hyperthermia (target T_{core} = 38.5 °C)	16 × 90 min	0	40	59		–/–	–/–	–/–	–	–	10
Patterson et al. [31]	Cycling. Controlled hyperthermia (target T_{core} = 38.5 °C)	16 × 90 min	0	40	59	15	−0.3/–	0/–	−10/–	33	–	–
Wagner et al. [41]	Uncontrolled outdoor sports (young, males)	17 × ~ 74 min	1	33	–	5	–/–	–/–	–/–	–	–	–
	Uncontrolled outdoor sports (old, males)	18 × ~53 min	1	33	–	4	–/–	–/–	–/–	–	–	–
Wyndham et al. [17]	Bench stepping @ 12 steps/min	17 × 240 min	0	34	–	–	–/–	–/–	–/–	–	–	–

HA heat adaptation, T_{amb} ambient temperature, *RH* relative humidity, T_{core} core body temperature, T_{skin} skin temperature, *HR* heart rate, *BM* body mass, *PV* plasma volume, VO_{2max} maximal oxygen consumption

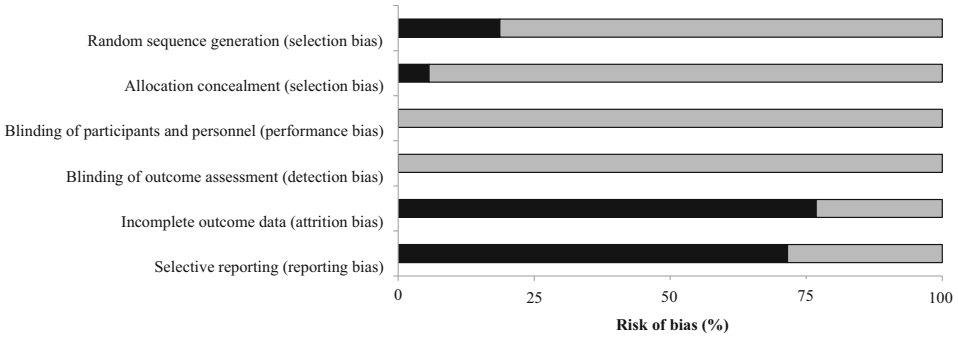

Fig. 2 Risk of bias assessment. *Grey* high risk; *black* low risk

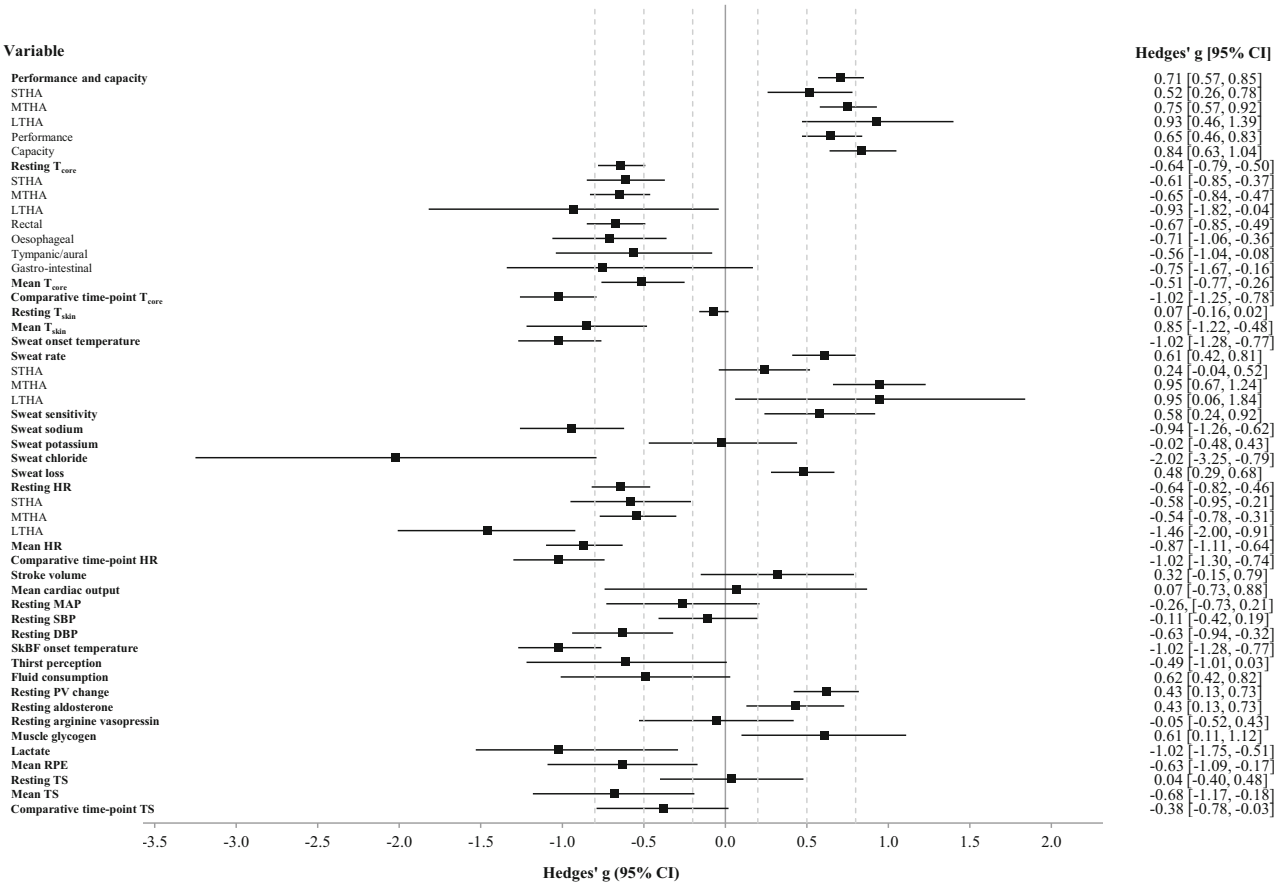

Fig. 3 Forest plot summarizing the effect [±95 % confidence intervals] of heat adaptation on exercise performance and capacity, and on physiological and perceptual responses. Data are from all studies, regardless of HA regimen, unless stated. Data for the four classic markers of HA as identified by Sawka et al. [10] are displayed collectively and for STHA, MTHA and LTHA sub-categories. Dashed lined denote small (Hedges' $g = 0.20$–0.49), medium (Hedges' $g = 0.50$–0.79) and large (Hedges' $g > 0.80$) effect sizes. *STHA* short-term heat adaptation, *MTHA* medium-term heat adaptation, *LTHA* long-term heat adaptation, *HR* heart rate, T_{skin} skin temperature, T_{core} core body temperature, *MAP* mean arterial pressure, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *SkBF* skin blood flow, *PV* plasma volume, *RPE* rating of perceived exertion, *TS* thermal sensation

loss (+38 ± 70 %, $n = 234$). BM loss values were highly variable with the greatest effect sizes observed following LTHA (31 ± 27 %, $n = 32$) compared to STHA (74 ± 115 %, $n = 81$) and MTHA (23 ± 38 %, $n = 121$), and this is likely to be due to the moderate effect of HA on increasing total SR (+19 ± 21 %, $n = 234$) and sweat sensitivity (+25 ± 10 %, $n = 70$). STHA approaches had a small effect on increasing SR (+5 ± 11 %, $n = 101$); however, large increases were only observed following MTHA (+29 ± 29 %, $n = 122$) and LTHA (+33 %, $n = 11$) approaches. There were strong positive relationships between the effect size reported for SR and the duration

($\rho = 0.52$, $p = 0.001$), ambient temperature ($\rho = 0.53$, $p = 0.001$), and frequency ($\rho = 0.49$, $p = 0.002$) of the HA regimen. There was no correlation between the effect size reported for SR and maximal oxygen consumption ($\rho = -0.01$, $p = 0.50$). Despite limited data, HA appeared to have a moderate effect on reducing the sensation of thirst (median: −11 (−12 to 0) arbitrary units, $n = 22$) and a small effect on decreasing volitional fluid consumption [median: −20 (−44 to 75) %, $n = 37$].

HA had a moderate, positive effect on increasing resting PV (+4.3 ± 4.7%, $n = 209$); however, there was no relationship between the percentage change in PV and HA duration ($\rho = -0.15$, $p = 0.25$), ambient temperature ($\rho = -0.24$, $p = 0.15$) or frequency ($\rho = 0.17$, $p = 0.24$). HA reduced sweat sodium (−22 ± 16 mmol/L[1], $n = 92$), and chloride (−13 ± 13 mmol/L, $n = 11$), loss; however, there appeared to be no effect on sweat potassium loss (−0.2 ± 0.7 mmol/L, $n = 39$). Resting aldosterone and arginine vasopressin concentrations show large levels of inter-investigation variability, but HA had a small positive effect on resting aldosterone concentrations (+25 ± 35 %, $n = 94$) and no effect on resting arginine vasopressin concentrations (−5 ± 15 %, $n = 28$) (Fig. 3).

3.6 The Effect of HA on Skin Blood Flow

Data relating to the SkBF responses to HA are limited. Data from five investigations suggested that HA had a large effect on reducing the temperature at which SkBF increases occur (−0.25 ± 0.10 °C, $n = 44$). HA increased SkBF sensitivity in response to hyperthermia [+42 ± 40 % (+13 to 70 %), Hedges' $g = 0.58$ (−0.10 to 1.25), $n = 18$] and in response to the infusion of acetylcholine at 1 mmol/L [+21 %, Hedges' $g = 0.84$ (−0.00 to 1.68), $n = 12$], 10 mmol/L [+15 %, Hedges' $g = 0.89$ (0.05–1.74), $n = 12$] and 100 mmol/L (+9 %, $g = 0.84$ (−0.09 to 1.58), $n = 12$) [34], but had a trivial effect on increasing maximal SkBF, assessed using red blood cell flux, [+4 %, Hedges' $g = 0.18$ (−0.41 to 0.78), $n = 22$] [34, 35] and on decreasing brachial artery blood flow [−21 mL/min, Hedges' $g = -0.30$ (−1.11 to 0.50), $n = 12$] [34] (Fig. 3).

3.7 The Effect of HA on Skeletal Muscle Metabolism and Heat Shock Protein Expression

Six investigations measured oxygen consumption before and after HA. Data from these showed that HA had a moderate effect on lowering oxygen consumption during steady-state, fixed-intensity exercise [Hedges' $g = -0.39$ (−0.75 to −0.05); $n = 68$; −2.5 ± 2.0 %].

Data are limited, but HA may have had a moderate effect on glycogen-sparing resulting in higher mean (+35 ± 47 %) and median (9 (8–89) %) muscle glycogen concentrations post-exercise [Hedges' $g = 0.61$ (0.11–1.12), $n = 33$]. One investigation reported that HA increased the power output at lactate threshold, although the magnitude of effect is uncertain [+1.5 W/kg, Hedges' $g = 0.03$ (−0.77 to 0.83), $n = 12$] [33], and data from three investigations suggested that HA may reduce lactate concentrations during exercise [median = −1.0 (−3.2 to −0.8) mmol/L, Hedges' $g = -1.02$ (−1.52 to −0.51), $n = 36$].

Human heat shock protein data are also lacking. The data currently available suggest that HA might have had a trivial effect on increasing extracellular concentrations of Hsp72 [median: +4 (−15 to 25) %, Hedges' $g = 0.18$ (−0.31 to 0.66), $n = 35$]; however, the data are equivocal. One investigation [36] reported that HA increased the ratio of extracellular concentrations of Hsp72 and 90 to β-tubulin by 17.7 and 21.1 %, respectively, while two others have reported HA-induced increased concentrations of intracellular Hsp72 of 110 % [37] and 320 % [38].

3.8 The Effect of HA on Rating of Perceived Exertion and Thermal Sensation

Data relating to the effect of HA on RPE are limited; however, HA appears to have had a moderate effect on reducing mean RPE and reduced it by a median of 1 (−3 to −1) arbitrary units ($n = 40$) in the three studies which reported it. The number of investigations that reported resting, mean and comparative time-point perceived ratings of thermal sensation were 4, 3 and 4, respectively. HA had no effect on resting thermal sensation [−0.2 (−0.3 to 0.6) arbitrary units, $n = 58$] but a small effect on reducing mean [−0.9 (−2 to −0.9) arbitrary units, $n = 35$] and comparative time-point [−0.8 (−2 to −0.3) arbitrary units, $n = 53$] ratings (Fig. 3).

4 Discussion

The primary aim of this review was to provide a comprehensive overview of the effect of HA on physiological and perceptual responses to heat exposure and on exercise performance and capacity in a hot environment. The meta-analysis reviewed 96 articles and summarised the current literature on over 30 performance, capacity, physiological and perceptual variables. Where appropriate, the meta-analysis has also analysed the effects of different HA approaches on these variables. The following section will discuss these integrated data and offer practical recommendations for HA delivery and future HA research based upon this systematic review of the literature.

4.1 The Ergogenic Effect of HA

Although individual studies show variation in the magnitude of the benefit that HA has on subsequent exercise performance and capacity, data from the current meta-analysis show that HA has a moderate to large beneficial effect on subsequent exercise capacity and performance in the heat. These data also show that the benefit is dependent upon the temperature and duration of HA, but not on the maximal oxygen consumption, or age of the participants. The size of the improvement observed was greater in capacity tests [mean +23 ± 29 %; median 8 (0–92) %; 23/26 benefit >1 %] than performance tests [mean +7 ± 7 %; median +4 (0–33) %; 24/27 benefit >1 %]. Capacity tests are more variable than performance tests, due to the open-loop nature of the task, [39] and greater variability in the effects of heat-mitigating interventions on capacity versus performance tests has recently been reported elsewhere [4].

The benefit to exercise performance and capacity is related to the duration of the HA protocol, thus when it comes to HA, the longer the exposure the better. As expected, LTHA protocols tended to have the greatest duration of HA; however, as shown in Table 1, some STHA protocols actually had a greater total HA exposure than some of the LTHA alternatives. The magnitude of benefit of HA is also dependent upon the frequency of heat exposures, with the greatest effect sizes being observed following LTHA [31, 40, 41] regimens; that said, STHA [20, 22, 30, 42–47] and MTHA [20, 26, 32, 33, 36–38, 41, 42, 48–59] regimens both still offer moderate benefits. This could be particularly useful because factors such as sudden changes in the weather, the scheduling of events in warm climates at relatively short notice (e.g. subsequent matches in a knock-out tournament following successful progression), training programmes and/or financial restrictions often require compromises in preparation and the adoption of sub-optimal practices. Performance data have seldom been recorded part-way during an HA intervention, but a recent manuscript included in this meta-analysis [20] reported that cycling performance was improved following 6 days of HA (STHA) and was further improved after 14 days of HA (MTHA), and these data correspond well with the results from this review. While there are no data to categorically state the number of heat exposures required for optimal performance/capacity gains, data from 70 years ago suggested that it might take up to a month [60]. There was a weak-moderate correlation between the ambient temperature used for HA and magnitude of benefit to exercise performance/capacity data; however, it is worth noting that the ambient temperatures used are fairly homogenous. All protocols used ambient temperatures in excess of 25 °C with mean and median temperatures of 40 °C (Table 1). Thermal adaptation requires a series of sufficiently overloading thermal impulses exceeding an adaptation threshold [9]. If the impulse is insufficient, adaptation will not occur or will occur sub-optimally. Only one study reviewed investigated the effect of different ambient conditions on HA; however, they did not measure exercise performance or capacity [61]. While the current data suggest that there is a weak–moderate effect of the temperature used in HA on the magnitude of benefit offered to exercise performance or capacity, this variable has not been systematically investigated. Based upon the overload model of heat adaptation, it seems prudent to suggest that the use of an ambient temperature in excess of natural heat exposure is required (this is especially important when considering individuals with high levels of natural HA [62]) and that this temperature may need to be increased during the course of the HA regimen until individuals are adapting to the desired temperatures.

Interestingly, there was no correlation between the participants' age or aerobic capacity and magnitude of benefit to exercise performance/capacity data after HA; however, as with ambient temperature, the data are quite homogenous (Table 1). Only two of the reviewed articles directly investigated the effect of age on the ergogenic effect of HA [41, 57], while one directly investigated the effect of aerobic fitness [26]. Pandolf et al. [57] reported that while exercise capacity was lower in young (~21 years) individuals compared to older (~46 years) individuals pre-acclimation, acclimation negated the advantage. All trials were terminated due to participants reaching the ethical cut-off core body temperature and so, while HA improved exercise capacity by ~31 % in the young participants, the older group effectively maintained their capacity following HA (+1 %). Wagner et al. [41] investigated the effect of HA on male participants between the ages of 11 and 67. As with Pandolf et al. [57], the protocols had a fixed maximal duration (in this case up to 90 min) and following HA all sub-groups were able to complete the task. Due to marked differences in the duration that could be completed pre-HA, there were marked differences in the benefit of HA between the age groups, and this response suggested that the beneficial effect of HA on subsequent exercise capacity was greater in younger compared to older individuals.

Regarding aerobic fitness, Cheung and McLellan [26] reported that 60 min of daily treadmill walking in the heat (40 °C) whilst wearing nuclear, biological and chemical protective clothing increased exercise capacity by 5 and 1 % in moderately (<50 mL/kg/min) and highly (>55 mL/kg/min) fit males, respectively. Participants in the high fitness group had a greater exercise tolerance, but the magnitude of improvement following HA was greatest in the moderately fit group. These investigations suggest that there may be an effect of age or aerobic fitness on HA, but

the meta-analysis' correlation data do not support this notion. It would be interesting to systematically investigate the role of each of these factors due to the effect of age [63] and aerobic fitness [26] on human thermoregulation.

The isothermal HA approach is often purported to be the optimal approach, and so it is interesting to observe that the magnitude of effect for this method regarding the enhancement of exercise performance and capacity was similar to that for the controlled work rate HA approach. Both approaches had a moderately beneficial effect on subsequent exercise performance or capacity; however, it is worth noting that the isothermal approach [31, 38, 44, 45, 52] has received less attention than the controlled work rate method [22, 26, 32, 33, 36, 37, 41–43, 46–51, 53–57, 64], and as a result of the fewer investigations available does have less certainty regarding its effect. Heat adaptation requires a progressively overloading thermal impulse to induce changes, and so the similarity between the controlled work rate and isothermal methods is surprising. Both of these approaches were less effective than the self-regulated approach, in which participants are able to self-select the intensity [20, 41, 58, 59] and this may have been due, in part, to the participants used. Racinais et al. [20], Schmit et al. [59] and Voltaire et al. [58] used well-trained athletes and allowed them to undertake their normal training in elevated ambient conditions, whereas Wagner et al. [41] used healthy individuals able to undertake their usual recreational activities through the summer months. It is possible that if participants are sufficiently motivated they self-select an intensity which provides a sufficient cumulative adaptation impulse to facilitate heat adaptation and that less motivated individuals may self-select an intensity which is insufficient to cause heat adaptations, but this is speculative at present. There were no performance or capacity data from passive isothermal investigations and only one investigation that reported performance data following fixed-duration passive HA [30]. Zurawlew et al. [30] reported that 6 days of passive HA [40 min of hot water (40 °C) immersion] administered immediately after 40 min of exercise in temperature conditions (18 °C) improved 5-km time-trial performance in hot ambient conditions (33 °C) by ~5 %. This approach offers a potentially practical HA option for athletes.

4.2 The Effect of HA on Physiological Adaptations to Heat

4.2.1 Body Temperature

There were moderate to large beneficial effects of HA on reducing resting [18, 22, 26, 27, 30–32, 35, 38, 43–45, 47–49, 52, 53, 56, 58, 65–86], mean [18, 32, 35, 40, 45, 52, 61, 64, 87–89] and comparable time-point [26, 36, 44, 48, 50, 56, 61, 70, 73, 76–78, 82, 84] T_{core} regardless of measurement site. Interestingly, the reduction was not correlated with participants' age or maximal aerobic capacity nor the HA duration or ambient temperature. Additionally, the effect of resting T_{core} was very similar between STHA and MTHA regimens. The size of the effect was smaller in STHA and MTHA groups (−0.17 °C) than in the single LTHA investigation (−0.32 °C) [35], but a reduction in resting T_{core} of ~0.2 °C is still likely to be physiologically important. While a reduction in T_{core} prior to exercise is not required for improvements in subsequent exercise, reductions in T_{core} can improve subsequent exercise performance and capacity [4]. It has been suggested that the majority of T_{core} adaptations occur within 7 days of HA [60, 90], and the data from the present meta-analysis offer support for this. There were no differences between the effect of STHA (≤7 days) and MTHA (8–14 days); however, the larger effect observed in the single LTHA investigation suggests that adaptations can continue past this time-frame. Patterson et al. [31] ensured that the thermal impulse was maintained throughout the HA regimen by using an isothermal model and reported that, although there was no statistical difference between the reduction observed in T_{core} after 8 and 22 days, T_{core} was 0.20 and 0.32 °C lower after 8 and 22 days of HA, respectively. It is possible that if the thermal impulse is maintained, then HA regimens in excess of 14 days may offer additional benefit; however, the LTHA data available were obtained from a single study [31]. Garrett et al. [44, 45] also adopted an isothermal regimen, and while they reported that 5 days (STHA) of heat exposure had no effect on resting T_{core}, it did reduce T_{core} during exercise. The reductions observed following STHA and MTHA approaches are almost identical and the lack of relationship between changes in resting T_{core} and HA regimen characteristics suggest that STHA protocols are sufficient to lower T_{core}. Rectal temperature is the most commonly measured estimate of T_{core} in the HA literature, although a number of sites have been used. The effect sizes observed are comparable across the measurement sites; however, the consistency in response should not be interpreted as an endorsement of the use of aural or tympanic temperature. While they may be similarly sensitive to change, it is well established that aural and tympanic temperatures are invalid estimates of T_{core} [91, 92].

As with T_{core}, it has been suggested that most of the T_{skin} adaptations to heat occur within 7 days [60, 90], but data from this meta-analysis show that resting T_{skin} is unaffected by HA of any duration [26, 27, 31, 38, 43, 47, 48, 65, 70, 76, 78, 81, 82, 84]. These meta-data correspond well with data from Patterson et al. [31], who observed that resting T_{skin} was unaffected by 8 or 22 days of heat exposure. HA has a large effect on reducing mean T_{skin} during exercise [35, 38, 40, 87–89]. A reduction in T_{skin} during exercise

suggests either a reduction in heat brought to the skin by reductions in peripheral SkBF and/or improvements in heat loss pathways once the heat has reached the skin. Data from this meta-analysis (see Sect. 4.2.4) show that SkBF response is augmented following HA and so an improved heat loss mechanism must explain the reduction in T_{skin} during exercise. Increases of ~11 % in evaporative heat loss have recently been reported [19], and such increases would seem to explain the conflicting observations of reduced T_{skin} and increased SkBF.

4.2.2 Cardiovascular Stability

The impaired ability to exercise in the heat is more often related to cardiovascular insufficiency than hyperthermia per se [93, 94], and so any cardiovascular adaptations to HA would be potentially very important to an athlete. Improved cardiovascular stability can be demonstrated by the maintenance of cardiac output and arterial pressure despite reductions in HR, due to compensatory increases in SV. The data from this meta-analysis show that HA offers a moderate, beneficial effect on lowering resting [18, 26, 27, 30–32, 47, 48, 52, 53, 61, 66, 67, 70, 72, 74–76, 78, 82, 85, 95] and mean HR [18, 20, 32, 35, 40, 45, 46, 51, 52, 58, 64, 69, 87–89]. HR adaptations to heat are often reported to occur first, and it has been said that most of the HR adaptation occurs within the first 4–5 days and that full adaptation takes ~7 days [8]. Data from the current meta-analysis support this because, despite a lack of correlation between the effects of HA on HR and the duration of heat exposure, the effects of STHA [27, 30, 67, 70, 76, 85] and MTHA [18, 26, 32, 48, 52, 53, 66, 72, 74, 75, 78, 81, 95] on lowering resting HR were similar to one another. This suggests that HA protocols lasting 7–14 days are no more effective than those lasting less than 7 days. Despite this, the effect sizes were much lower for STHA and MTHA than for LTHA. There were only two LTHA investigations [31, 61] and so these results could be due to the smaller sample size or could suggest that, once again, more frequent heat exposures offer a greater heat adaptation and that complete adaptation takes far longer than 7 days. Griefahn [61] investigated the effect of three different HA approaches varying based upon their ambient conditions (warm-humid, hot-dry and radiant-heat) and observed that, regardless of condition, HR adaptations occurred rapidly at the start of each HA regimen and plateaued after approximately 7, 7 and 11 days, respectively. In contrast, Patterson et al. [31] reported that resting HR was reduced by 5 beats/min after 8 days of HA and was reduced by a further 5 beats/min after 22 days of HA. It has been suggested that the continued response observed by Patterson et al. [31] may be due to the adoption of a study design which carefully ensured a constant thermal impulse. While the time-course of HR adaptations to HA requires further investigation, the reduction in HR for a given intensity as adaptation occurs is well accepted and supported by the data in this meta-analysis. Recently, Periard et al. [8] proposed that as a result of the reduction in HR observed as HA occurs, HR could be used as a simple way to set workloads which provide a consistent forcing function and ensure that a sufficient thermal impulse is maintained for effective heat adaptation. It is possible that in self-regulated HA this is already occurring because athletes may be sufficiently physiologically self-aware to increase the pace of training intensity in line with cardiovascular adjustments [96], but this is speculative.

Smaller samples provide less certainty over the effect that HA has on the other variables of note, but it appears that systolic [40, 75, 82, 95], diastolic [40, 75, 82, 95] and mean [70, 75, 85] arterial pressures are maintained or reduced, cardiac output is maintained [33] and stroke volume is increased [17, 33, 55, 56], which offers further support for the suggestion that HA can improve cardiovascular stability. The largest effect on arterial pressures was observed in reducing diastolic pressure. These data are in agreement with animal data which showed an improved diastolic pressure-volume relationship following heat treatment [97]. Acute changes in T_{core} induced by passive heating have no effect on arterial pressure [98]; however, without concomitant changes in other cardiovascular variables prolonged heat exposure could cause disturbances. The effect size for the increase in SV (Hedges' $g = 0.32$) was 'small' although the mean increase was 12 %—the reason for this discrepancy is that two articles reported increases of ~20 mL whereas the others reported changes of between −4 and 4 mL. The median change of +4 mL is consistent with the effect size reported. Increases in SV occur predominantly due to increases in PV (hypervolemia). HA has a moderate, positive effect on increasing resting PV; nevertheless, there is no relationship between the effect size reported for changes in PV and the participants' age or aerobic capacity, nor the duration, frequency or ambient temperature of the HA regimen used. Many researchers agree that PV expansion is an adaptation that is required to reduce cardiovascular strain during subsequent heat exposure [99–101] and despite different methods used, and the associated potential errors [102], the data from the meta-analysis show that such a response is commonly observed following HA regardless of regimen. The magnitude of PV expansion appears to be dependent upon the exercise intensity [103], and therefore the thermal impulse, and so traditional, constant-work models tend to observe a reduction in the magnitude of PV change observed over time as the thermal impulse declines [87]. It has recently been suggested that PV expansion is "generally 4–15%" [8]; however, this appears a slight overestimation based

upon the mean (+4.3 ± 4.7 %), median (+3 %) and range (−1.9 to +16 %) of response observed in 20 manuscripts reviewed for this meta-analysis [22, 31–33, 37, 44, 45, 55, 56, 59, 66, 70–72, 76, 78, 80, 81, 88, 104]. Previously it has been proposed that the increase in PV is a transient response [17]; however, more recent data using an isothermic HA have shown that if the magnitude of thermal strain is maintained throughout the HA regimen, PV expansion can persist throughout the protocol [105]. The mechanism(s) explaining the PV expansion remain unclear [105], but merit further investigation because the benefits to cardiovascular stability are well accepted.

4.2.3 Sweat Responses and Markers of Fluid Balance

The main characteristics of sudomotor function adaptation to heat are changes in the onset temperature at which sweat commences (sudomotor threshold), changes in the sensitivity of the sweat response to changes in body temperature, and changes in the capacity of the sweat glands. Data from the current meta-analysis show that HA markedly reduces the sudomotor threshold and has a moderate effect on increasing sweat sensitivity, sweat rate and total sweat loss.

Total sweat loss was increased following HA with the greatest effect observed following LTHA [61, 106] compared to STHA [20, 22, 35, 43, 45, 67, 76, 87] and MTHA [20, 26, 33, 49, 50, 58, 66, 77, 78, 80, 83, 86, 87, 107] and this is likely to be due to the moderate effect of HA on increasing total SR and sensitivity. STHA elevated SR [22, 24, 30, 35, 46, 47, 64, 84, 108, 109]; however, large increases were only observed in MTHA [26, 32, 36, 50, 52–54, 56, 58, 66, 72, 77, 110–112] and LTHA [31] subgroups. It has been proposed that it might take up to 1 month of heat exposure for complete changes in sweat response to be observed [60], and while the data from the meta-analysis are unable to confirm this, it does show that there is a strong positive relationship between the effect size reported for SR and the duration of the HA regimen and between SR and the ambient temperature of the HA regimen. An increase in total sweat loss (i.e. sudomotor capacity) is only a beneficial adaptation to HA if matched with increases in evaporative heat loss. HA can increase the body's evaporative capacity by 10–11 % [19, 107], but because the magnitude of increase in evaporative capacity is less than the magnitude of increase in HA-induced sweat production (∼10 vs. ∼30 %), there is a ∼200 % increase in the amount of unevaporated sweat [107]. While some have suggested that this may offer a benefit to humid-heat adaptation [113], such unmatched adaptations are likely to augment the magnitude of thermal strain experienced and perceived in the heat. In unacclimatised individuals, higher SRs often lead to more concentrated sweat [114]; however, HA may enhance the reabsorption of electrolytes resulting in more dilute sweat. Data from the present meta-analysis show that HA reduces the concentration of sodium [24, 35, 47, 56, 76, 88, 111, 115], and chloride [47, 66] in sweat lost, but has no effect on sweat potassium concentrations [24, 47, 78, 115]. Sodium is actively and selectively reabsorbed in the sweat gland whereas the reabsorption of chloride is passive and tracks the reabsorption of sodium [116]. A more dilute sweat can be more readily evaporated due to the resultant reduction in cutaneous water vapour pressure [9]. Increasing aldosterone concentrations can increase the amount of sodium reabsorbed by the sweat glands [117], and so the reductions observed in sodium and chloride concentrations in sweat lost may be, at least in part, due to the HA-induced increases in resting aldosterone concentrations reported in most [24, 44, 45, 55, 56, 104, 111] but not all [22, 24, 66, 81] HA investigations. Resting aldosterone and arginine vasopressin concentrations show large levels of inter-investigation variability, and while HA had a small positive effect on resting aldosterone concentrations [22, 24, 44, 45, 55, 56, 66, 81, 104, 111], no such effect was observed for resting arginine vasopressin concentrations [44, 55, 56, 81]. Arginine vasopressin is a fluid regulatory hormone and although, like aldosterone, it is elevated following hyperthermia-induced hypohydration relative to euhydration trials [118, 119] none of the reviewed articles reported a change following HA [44, 55, 56, 81].

It is worth noting that the magnitudes of reduction observed in resting core and sweat onset temperatures (sudomotor threshold) are similar (−0.18 vs. −0.28 °C) and this similarity is enhanced when only using resting core temperature data from investigations which measured both variables in the same participants simultaneously [19, 65, 73, 120] (−0.24 vs. −0.26 °C), or the change in mean core temperature (−0.31 °C). This observation has previously been noted by Patterson et al. [31] and suggests that the sweat response is driven by the magnitude of change in T_{core} rather than exceeding a particular T_{core} threshold. The benefit of such a response is that evaporative heat loss can commence at a lower T_{core} allowing individuals to maintain a reduced T_{core} during steady-state exercise or to increase their rate of heat production during self-paced events. While this suggests a central drive for increased sudomotor sensitivity, Lorenzo et al. [34] observed increases in sweat response following HA and the local infusion of an endothelium-dependent vasodilator (acetylcholine) showing that improvements in sudomotor sensitivity have both peripheral and central causes.

Regulation of the sudomotor response is just one component of the maintenance of fluid balance, the other being fluid consumption. Thirst is a poor indicator of hydration status and drinking based upon the perception of thirst may

lead to dehydration during exercise [121]. While it is often stated that such dehydration impairs exercise performance in the heat [122], recent data have shown that acute dehydration up to 3 %, with or without the modification of thirst perception, has no effect on exercise performance lasting 20–25 km (~36 to 40 min) [5, 123]. Despite limited data, HA appears to have a moderate effect on reducing the sensation of thirst [22, 86, 124], and, therefore, unsurprisingly decreases volitional fluid consumption during subsequent exercise performed in hot conditions [20, 22, 47, 86, 124]. Despite the overall reduction in volitional fluid consumption, both increases [20, 47] and reductions [22, 86, 124] have been observed. Ormerod et al. [124] sought to characterise the fluid consumption of women over a prolonged (6 weeks) HA regimen, and in addition to a progressively reduced volitional fluid consumption, they also reported a progressive reduction in the number of drinks voluntarily consumed and a greater time taken to consume the first drink. HA improves perceptual measures of participant thermal comfort in the heat (see Sect. 4.2.7) and this appears to be the same regarding indices of perceived thirst. If thirst is a key driver of fluid consumption it stands to reason that a decreased perception of thirst would reduce volitional fluid consumption. Progressive dehydration may augment the magnitude of HA adaptations by placing a greater strain on the body [125] and so this effect may be beneficial; however, prolonged dehydration may compromise the ability to exercise and potentially compromise the health of the individual and so this is an area that requires further research before safe, and effective, recommendations can be confidently made.

4.2.4 Skin Blood Flow

One profound response to heat exposure is a redistribution of blood flow to the periphery, which helps to attenuate increases in T_{core} when T_{skin} is lower than both T_{core} and the ambient temperature. Despite potentially reducing thermoregulatory strain, this redistribution of blood flow places the body under greater cardiovascular strain [126]. As highlighted, data relating to the SkBF responses to HA are limited, but data from the five investigations reviewed that measured such variables show that SkBF increases are initiated earlier following HA [19, 70, 71, 85, 120]. An earlier increase in SkBF might explain the reductions observed in T_{core} following HA as internal heat is more readily transferred to the periphery and this, coupled with reductions in T_{skin} during exercise following HA, suggests that HA improves heat loss pathways. Increases in SkBF, and the associated delivery of heat to the periphery, increase the cutaneous water vapour pressure, which would increase evaporative heat loss at a constant sweat flow [9]. Other data related to the effects of HA on SkBF-related variables are even more limited. Lorenzo et al. [34] reported that HA increased cutaneous vascular conductance in response to the infusion of acetylcholine at concentrations of 1, 10 and 100 mmol/L, while Fujii et al. [70, 71] observed similar responses following both active and passive heat stress. Neither Lorenzo et al. [34] nor Neal et al. [35] observed a meaningful effect of HA on maximal SkBF. Lorenzo et al. [34] suggested that the increase in SkBF response following HA was due to local adaptations in the skin microcirculation. Specifically, they were likely to be due to an up-regulation of the cyclooxygenase pathway or an enhanced endothelium-derived hyperpolarising factor mechanism; however, the study design did not enable the authors to rule out a nitric oxide component. Enhanced SkBF following HA could enhance convective and evaporative cooling and if matched with increases in blood volume (e.g. due to PV expansion) would do so without compromising cardiovascular stability. While the data suggest that SkBF can be enhanced by HA, it is worth re-stating the lack of data currently available.

4.2.5 Metabolism

HA appears to have a moderate effect on lowering the oxygen cost of a given exercise intensity. Five of the six investigations [24, 35, 51, 56, 69, 89] reported a reduction in the oxygen cost, although there is a range in the effect sizes reported (Hedges' $g = -1.38$ to 0.00). Improvements in exercise efficiency would be expected to lead to a decreased heat production or an ability to sustain a higher power output, and as a result improve subsequent exercise performance. It has been suggested that the improvements are related to the exercise rather than the heat exposure [9]; however, Lorenzo et al. [33] suggested that the improvements observed in time-trial performance following HA in their study were independent of the training due to the high basal fitness levels of the participants and the lack of improvement observed in the control group. Critically, Lorenzo et al. [33] did not control the volume of training undertaken by their participants, and so it is still possible that HA does not explain all/any of the performance benefits observed; however, recent data from Neal et al. [35] reported similar magnitudes of change in the HA and temperate control groups. As well as reducing the oxygen cost of exercise, HA may alter substrate metabolism. HA had a moderate effect on glycogen sparing, resulting in higher muscle glycogen concentrations post-exercise [51, 69]. HA also may be able to increase the lactate threshold [33] and reduce lactate concentrations during exercise [69, 78], but the mechanisms are unclear. It has been suggested that HA may change the lactate kinetics by reducing the aerobic metabolic rate [127] or reducing the rate of glycogenolysis [69]; however, it has also been suggested

that the HA-induced increase in central blood volume may increase lactate removal [128].

4.2.6 Heat Shock Proteins

The heat shock response is often one of the markers of improved thermotolerance, and is characterized by the expression of Hsps, which are grouped according to their molecular mass and the location of their expression. Intracellular Hsps regulate protein structure and transport, whereas extracellular Hsp expression appears to have an anti-inflammatory response [129], and the extent of the expression of these proteins is dependent upon the magnitude of heat strain [130]. While well-trained individuals may have an improved Hsp response to heat exposure [131], the human heat shock response to HA is somewhat unclear and data are lacking and equivocal. Data from this meta-analysis suggest that HA may have a trivial effect on increasing extracellular concentrations of Hsp72. One investigation [38] reported that HA increases the ratio of extracellular concentrations of Hsp72 and 90 to β-tubulin by 17.7 and 21.1 %, respectively, while two others have reported HA-induced increases in the concentrations of intracellular Hsp72 of 110 % [39] and 320 % [40]. Together, these data suggest that intracellular Hsp may be more sensitive to heat stress and HA than extracellular Hsp; however, the lack of data also highlights the need for further work to investigate the effects of HA on the human heat shock response and the practical meaning of any such response.

4.2.7 Ratings of Perceived Exertion and Thermal Sensation

Lowering the perception of effort can improve exercise performance and capacity [132] and it appears, from the limited data available, that HA can reduce the perceived levels of effort in subsequent bouts of exercise in the heat. Data were only available from three investigations, but these showed a moderate effect of HA on reducing the mean RPE reported during exercise [35, 40, 52]. It seems prudent to suggest that reductions in perceived exertion would enable individuals to self-select a higher work-load during performance (closed-loop) events, and to tolerate steady-state exercise for longer in capacity (open-loop) tests. Perception of thermal comfort is a key driver of volitional behaviour (including exercise performance and capacity) in the heat, and it tracks seasonal variations in air temperature (i.e. hot temperatures are better tolerated in the summer months) [133]. This highlights that passive exposure to warmer temperatures can transiently enhance thermal comfort in the heat. The limited data from the current meta-analysis suggest that HA has no effect on resting thermal sensation [42, 48, 78, 86] but that there is a small, beneficial effect on reducing mean [32, 35, 88] and comparative time-point ratings [22, 30, 48, 78]. Thermal comfort and sensation are different psychophysical indices and it seems logical to suggest that HA could improve thermal comfort by shifting equivalent sensations to higher levels of thermal strain; however, data regarding the effectiveness of HA in doing so are severely limited, thus no firm conclusions can be made at this time.

5 Conclusion and Practical Considerations

When designing an HA intervention to be implemented in the field consideration needs to be made of the time available to the athlete and of the training phase of the athlete to make sure that the intensity is appropriate. Often, time pressure means that short interventions are sought and this demand has, in part, fuelled the recent research into STHA. Data from the present meta-analysis show that STHA regimens are effective at inducing adaptations in T_{core} and HR which are comparable to those induced by MTHA approaches. While it has previously been suggested that 7 days of HA is sufficient to induce "optimal" adaptation [8], data from Patterson et al. [31] show that if the thermal impulse is maintained, further benefit can be gained from longer protocols, suggesting that if time allows and an isothermal HA model can be adopted, longer protocols may be preferable. Longer protocols are certainly preferable for inducing beneficial changes in exercise performance and capacity, with the greatest improvements observed in the LTHA investigations. The findings from this meta-analysis indicate that, due to limited data, the other proposed timeframes are less certain, and that the effect on the sweat response is greater in MTHA than STHA. That said, there are insufficient data to support the suggestion that LTHA protocols are better than MTHA with similar magnitudes of benefit observed following each approach. There are also insufficient data to support or refute the proposed time-frame regarding perceptual measures of thermal sensation and comfort. The majority of investigations utilized consecutive days of heat exposure and this approach appears to be preferable if time is a consideration. Ten consecutive days of heat exposure caused a similar extent of heat adaptation as 10 days delivered over a 27-day period [134]. So if time is a constraint, the adoption of consecutive days would be preferable. When time is less of an issue, these data can be used to support the effectiveness of heat exposure delivered every third day; however, periods of 7 days between heat exposures appear to induce no heat adaptation [135]. The magnitude of adaptation is dependent upon the intensity and duration [24] as well as frequency of heat exposure.

Short -duration, moderate-intensity exercise (30–35 min at 75 % maximal oxygen consumption) results in similar heat adaptation to longer-duration, lower-intensity exercise (60 min at 50 % maximal oxygen consumption) [24]; however, unless the absolute intensity is increased with adaptation, the relative intensity will drop, and it is likely that the magnitude of heat adaptation will be compromised [9]. While alterations to exercise intensity might not be practical when considering a HA intervention, because the HA needs to occur sufficiently close to the event (e.g. it might not be possible to choose an optimal intensity for HA during a tapering period), if time is a constraint increasing the exercise intensity may facilitate heat adaptation more efficiently.

Based upon the data the following recommendations can be made:

- Athletes should spend as much time as possible exposed to high ambient temperatures to maximise performance and physiological and perceptual adaptations. Ideally, preparation for competition in hot temperatures should start at least 14 days prior to the event.
- Higher-intensity exercise may offer a more rapid HA stimulus than lower-intensity exercise and so could be considered when time constraints prevent a longer HA regimen.
- Where possible, active, rather than passive, HA should be undertaken.
- The ambient temperatures used should mimic the conditions which will be encountered. Due to reductions in work capacity in the heat the thermal stress may need to be progressively increased to balance HA and training needs.
- Exercise intensity during the HA should be monitored and progressively increased to ensure that overloading and adaptation continue to occur.
- HR may be a practical way to ensure an appropriate magnitude of physiological strain during HA when the measurement of core body temperature is impractical.
- Where possible, consecutive days of HA should be undertaken; however, if this is not possible heat exposure every other, or every third, day can still induce beneficial adaptations.

5.1 Further Research Considerations

Our understanding of the effects of repeated bouts of heat exposure has greatly increased over the last century, but we are still some way from fully understanding all of the frequently, and less frequently, observed responses and establishing an 'optimal' HA intervention for a given set of circumstances and objectives. While some HA studies have directly investigated and/or reported data to comment on the effects of age [41, 57, 120, 136], aerobic fitness [21, 26], ambient conditions [61], exercise intensity [24] and HA [19, 20, 31, 87], many have not. Although HA research is logistically challenging, this meta-analysis has highlighted that while some "typical HA responses" are based upon regular and consistent observations from well-designed investigations, some other claims are based upon single investigations and/or underpowered or less well designed studies. The heterogeneity of the protocols used poses a problem when ascertaining the effects of HA and this is especially the case considering the lack of LTHA and isothermal investigations. The effectiveness of a HA intervention is likely to be dependent upon the frequency, duration, and intensity of the HA, and as a result the classification of HA intervention into STHA, MTHA and LTHA, while helpful, is too simplistic. While there is some consistency in the responses observed between many HA regimens, the data presented in Table 1 highlight the variability that exists among the protocols used. For example, the data clearly show that although the frequency of visits has been used to classify approaches as STHA, MTHA or LTHA, it is possible for participants to be exposed to high ambient temperatures for longer in a STHA protocol than in MTHA or LTHA alternatives in some case. Pandolf [137] stated that "100-min exercise bouts seem optimal to induce the heat-acclimation process" (p 159); however, due to the variation in protocols used there are currently no definitive data to support this claim and in fact shorter, higher-intensity protocols are just as effective as longer, lower-intensity ones [24]. Future research should attempt to systematically address the highlighted gaps in the literature—both from an applied and a mechanistic perspective.

Compliance with Ethical Standards

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Conflict of interest Christopher Tyler, Tom Reeve, Gary Hodges and Stephen Cheung declare that they have no conflicts of interest relevant to the content of this review.

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