

## RESEARCH ARTICLE

# Acute physiological and psychophysical responses to different modes of heat stress

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**Funding information**

School of Physical Education, Sport and Exercise Sciences, University of Otago, Postgraduate Research, Grant/Award Number: held by H.A.C

Edited by: Shigehiko Ogoh

**Abstract**

Heat stress is common and arises endogenously and exogenously. It can be acutely hazardous while also increasingly advocated to drive health and performance-related adaptations. Yet, the nature of strain (deviation in regulated variables) imposed by different heating modes is not well established, despite the potential for important differences. We, therefore, compared three modes of heat stress for thermal, cardiovascular and perceptual strain profiles during exposure and recovery when experienced as a novel stimulus and an accustomed stimulus. In a crossover design, 13 physically active participants (five females) underwent 5 days of 60-min exposures to hot water immersion (40°C), sauna (55°C, 54% relative humidity) and exercise in the heat (40°C, 52% relative humidity), and a thermoneutral water immersion control (36.5°C), each separated by  $\geq 4$  weeks. Physiological (thermal, cardiovascular, haemodynamic) and psychophysical strain responses were assessed on days 1 and 5. Sauna evoked the warmest skin (40°C;  $P < 0.001$ ) but exercise in the heat caused the largest increase in core temperature, sweat rate, heart rate (*post hoc* comparisons all  $P < 0.001$ ) and systolic blood pressure ( $P \leq 0.002$ ), and possibly decrease in diastolic blood pressures ( $P \leq 0.130$ ), regardless of day. Thermal sensation and feeling state were more favourable on day 5 than on day 1 ( $P \leq 0.021$ ), with all modes of heat being equivalently uncomfortable ( $P \geq 0.215$ ). Plasma volume expanded the largest extent during immersions ( $P < 0.001$ ). The current data highlight that exercising in the heat generates a more complex strain profile, while passive heat stress in humid heat has lower tolerance and more cardiovascular strain than hot water immersion.

**KEYWORDS**

exercise in heat, heat strain, hot water immersion, sauna

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## 1 | INTRODUCTION

Heat is a common stressor, and whether caused by exercise (endogenous) or the environment (exogenous), it confers strain on all physiological systems and ultimately the impairment of aerobic power, work capacity, comfort and resilience against other environmental stressors (e.g., gravity or reduced water availability). Responses to heat and one's tolerance of heat can vary markedly between individuals, and so too might different modes of heating induce different physiological and psychophysical strain profiles (Akerman et al., 2019; Gibbons et al., 2021; Kissling et al., 2019; Périard et al., 2015). Common modes of heat stress include hot air (e.g., sauna or heat waves), hot water immersion (HWI; e.g., spa bathing) and exercising, especially in warm or humid environments. Despite the variety of ways to induce heat stress, to our knowledge there are very few direct comparisons in regard to the physiological or psychophysical strain profiles they elicit during heat exposure or recovery. Yet, such knowledge is important for reasons including insight into the causation of acute heat stress tolerance, safety, and mechanisms of adaptation.

Different modes of heating might differentially affect strain during and after heating for several reasons. For example, water has much higher density and specific heat than air, thus nullifying evaporative heat transfer, maximising convective transfer and perhaps thermal discomfort (Gagge & Gonzalez, 1974). Water also provides more hydrostatic pressure, the effects of which will acutely influence blood pressure regulation, natriuresis (via atrial natriuretic peptide) and blood volume (Norsk & Epstein, 1988). Exercise in the heat (ExH), when compared with passive heat stress alone, provides additional metabolic and cardiovascular strain consequent to increased muscle work (Akerman et al., 2017; Kondo et al., 2001), but may lessen heat-related discomfort due to higher weighting of core to skin temperature (Chatonnet & Cabanac, 1965).

The primary purpose of this study was therefore to characterise the thermal, cardiovascular and psychophysical responses to different forms of heating (HWI, Sauna and ExH), matched as closely as possible for rate and volume of core heating. A secondary purpose was to investigate the extent to which these responses may or may not be attenuated when individuals were more accustomed to the stimulus, that is, by the fifth exposure. We hypothesised that: (1) HWI would maintain the highest mean arterial blood pressure (MAP) during exposure, whereas ExH would cause the greatest post-exposure hypotension; (2) acute thermal and cardiovascular strain would be less pronounced by day 5, compared to day 1 of exposure for ExH but not for passive heat stress (for which heat loss mechanisms are nullified); and relatedly (3) thermal discomfort and feeling state would be improved on day 5 despite matched thermal strain.

## 2 | METHODS

### 2.1 | Ethical approval

Procedures were approved by the Institutional Human Ethics Committee of University of Otago (Project No. H17/090) and

### New Findings

- **What is the central question of this study?**

What are the profiles of acute physiological and psychophysical strain during and in recovery from different modes of heating, and to what extent do these diminish after repeated exposure?

- **What is the main finding and its importance?**

Mode of heating affects the strain profiles during heat stress and recovery. Exercise in the heat incurred the greatest cardiovascular strain during heating and recovery. Humid heat was poorly tolerated despite heat strain being no greater than in other heating modes, and tolerance did not improve with multiple exposures.

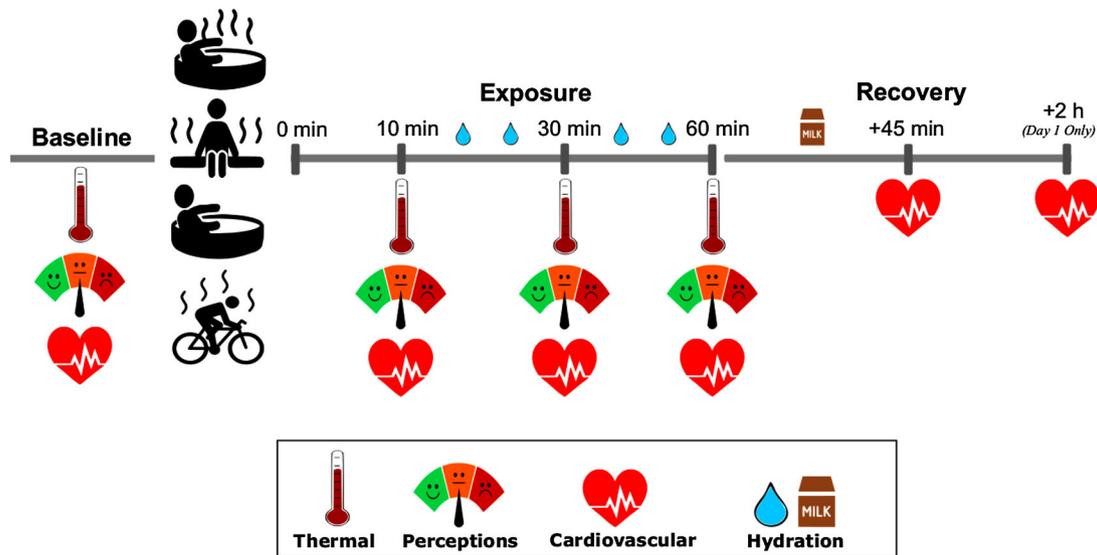
conformed to the *Declaration of Helsinki* (2013), other than prior registration in a database. Written informed consent was provided by all individuals prior to participation.

### 2.2 | Experimental design

A randomised crossover design was used to compare the effects of three modes of heat stress and one thermoneutral control. The four conditions were: (1) HWI in  $40.1 \pm 0.2^\circ\text{C}$  water, with temperate air ( $24 \pm 2^\circ\text{C}$ ,  $30 \pm 6\%$  relative humidity (RH), or 0.9 kPa); (2) Sauna in humid air at  $55 \pm 1^\circ\text{C}$ ,  $54 \pm 3\%$  RH (8.5 kPa); (3) ExH in air at  $40 \pm 0^\circ\text{C}$ ,  $52 \pm 1\%$  RH (3.8 kPa); and (4) thermoneutral water immersion (TWI) in  $36.5 \pm 0.2^\circ\text{C}$  water, with temperate air ( $23 \pm 1^\circ\text{C}$ ,  $31 \pm 5\%$  RH, or 0.9 kPa). Each mode of heat stress was undertaken for 60 min on five consecutive days; day 1 and 5 exposures and recovery were monitored and are reported here, as illustrated in Figure 1 and detailed below. The intention with each heating condition was to increase core temperature (rectal;  $T_{re}$ ) by  $\sim 1.5^\circ\text{C}$  as rapidly as possible and at similar rates between modes of heating, whereas TWI aimed to maintain baseline  $T_{re}$  (see Section 2.4 for exceptions).

The 60-min duration was chosen as a balance between the volume and intensity of exposure that would be tolerable yet sufficient to allow comparisons across modes of heating whilst also driving adaptation within the accompanying heat acclimation studies (see below). A 5-day period was used for studying habituation effects because the companion paper (Kissling et al., 2022) investigates the kinetics of short-term heat acclimation, and much of the psychophysical and physiological cardiovascular effect becomes evident within this timeframe (Sawka et al., 2000).

At least a 4-week washout period (4–15 weeks) between heat conditions was ensured to minimise the risk of carry-over adaptations, and only a 1-week washout (1–10 weeks) was given after TWI as no adaptation was anticipated. All sessions were completed between



**FIGURE 1** Schematic representation of experimental procedure on day 1 and 5 in four conditions: hot water immersion, sauna, thermoneutral water immersion and exercise in heat. Measures: thermal: rectal and skin temperature; perceptions: thermal sensation, thermal discomfort and feeling scale; cardiovascular: heart rate, blood pressure and haematocrit; hydration: chocolate milk (250 ml) and water (200 ml)

autumn and spring, with an outdoor average dry bulb temperature in Dunedin, New Zealand of  $14.3 \pm 4.6^\circ\text{C}$  and  $70 \pm 16\%$  RH.

Prior to the experimental conditions, all participants completed a familiarisation session that consisted of ExH. Cycling peak aerobic power ( $\dot{V}_{O_{2peak}}$ ) was tested in temperate conditions at one point within the overall period of participation and at least 2 weeks apart from any given heat condition, for the purposes of characterising the cohort.

### 2.3 | Participants

Thirteen participants (body mass  $70.2 \pm 6.2$  kg, height  $175 \pm 6$  cm, body mass index  $23 \pm 1$  kg/m<sup>2</sup>, aged  $23 \pm 4$  years, eight males and five females) completed the study. Participants were aerobically fit ( $\dot{V}_{O_{2peak}}$ :  $54 \pm 8$  ml/min/kg), all physically active (i.e., averaged >30 min/day on 5 days/week), familiar with maximal exercise exertion, and unacclimated to heat. Participants were asked to refrain from alcohol and caffeine for 24 and 12 h, respectively, before each session. They were asked to record dietary and exercise details and to maintain similar practices between each of the four conditions (confirmed verbally). All women were using oral contraception and sessions occurred during the 3-week active pill phase.

### 2.4 | Experimental procedure (Figure 1)

Participants arrived at the laboratory at the same time of day for each exposure to minimise any effect of circadian rhythm. After voiding their bladder, recording nude mass and inserting a rectal thermistor, participants rested seated and were instrumented for continuous measurement of heart rate and skin temperature

(Section 2.5). After  $\geq 10$  min arterial blood pressure was measured before capillary blood samples were obtained from a finger or ear prick (i.e., standardised within participants). Participants then entered the specific environment and began the exposure, detailed below.

Blood pressure, haematocrit (Hct) and psychophysiological measurements were obtained at 10, 30 and 60 min of heat stress. Water (~200 ml) was provided every 12.5 min of heat exposure to standardise fluid intake and maintain approximate euhydration. At 60 min or early termination of the exposure all measures were collected before participants left the environment to be reweighed (nude and towelled dry) and consume 250 ml of chocolate milk. This was used to facilitate rehydration and albumin synthesis (Okazaki et al., 2009), and to standardise protein and carbohydrate intake prior to the +45 min measures.

On days 1 and 5 they returned to the laboratory at 45 min and 120 min (day 1 only) after heat stress for 10-min seated measurements of heart rate, blood pressure and haematocrit. Participants were not required to standardise their activity or posture between heat stress and returning for recovery measures at +45 min, however, most remained in the laboratory.

#### 2.4.1 | Modes of heating

In the immersion conditions (HWI and TWI), participants were immersed to nipple height, with arms out for ~30 min, then to the neck (~20 min) until  $T_{re}$  reached  $+1.5^\circ\text{C}$ , followed by shallower immersion to maintain  $T_{re}$ . In Sauna, participants were seated upright until 60 min elapsed or they became hypotensive or pre-syncopal and either lay down or voluntarily terminated the exposure. During ExH, participants cycled upright at a workrate of 1.75 W/kg, which was then reduced to

clamp  $T_{re}$  at  $+1.5^{\circ}\text{C}$ , or if  $+1.5^{\circ}\text{C}$  could not be achieved they cycled at their maximal tolerable work rate. Exposures were ended prematurely only if participants reached volitional tolerance or  $T_{re}$  reached  $40^{\circ}\text{C}$ . Eight participants were part of an overlapping study of 9 days' heat acclimation in HWI and ExH conditions. On day 1 and 5 of HWI and ExH (day 1 only), these participants completed heat stress tests which consisted of a matched heat load profile throughout the 60 min, that is, nipple level immersion, arms out in HWI and fixed 1.75 W/kg work rate for 30 min followed by 30-min time trial in ExH.

## 2.5 | Measurements

Participants' nude body mass was measured to a precision of 0.02 kg (D1-10, Wedderburn, Dunedin, New Zealand) before and after each exposure. Urine specific gravity was measured using refractometry (Uricon - N, Atago, Tokyo, Japan), and if  $>1.020$ , participants were required to drink 400 ml of water. Rectal temperature was measured at a depth of  $\sim 15$  cm (General purpose thermistors, 400 series, Mallinckrodt Inc., St Louis, MO, USA), and skin temperature (2.3K3A1B Thermistor NTC, Betatherm, Galway, Ireland) was measured at four sites: posterior gastrocnemius, anterior mid-thigh, dorsal forearm and the inferior angle of the scapula. All thermometry measures were collected and stored every 10 s on a portable logger for later analysis (DaqPRO 5300, Omega, Norwalk, CT, USA).

Heart rate was measured from the ventricular depolarisation and recorded as the R-R interval of successive beats (RS800x, Polar, Kempele, Finland). Brachial arterial blood pressure was measured using a manual sphygmomanometer (Diagonstix 972, ADC, Hauppauge, NY, USA), in triplicate, and following international guidelines (Whelton et al., 2018). Hct was obtained in triplicate and measured using a custom-built microcapillary tube reader after 10-min centrifugation at 855 g (MircoCL 17, Thermo Fisher Scientific, Waltham, MA, USA). Both measurements were always taken by the same investigator within a condition; MAP coefficient of variation: 7.1% (95% confidence interval: 5.8, 9.3%), typical error: 6.0 mm Hg (4.9, 7.7 mm Hg); Hct coefficient of variation: 5.1% (4.2, 6.7%).

Sweat rate was estimated from the change in nude body mass accounting for fluid consumed and mass lost as urine (measured to a resolution of 1 and  $\sim 10$  g, respectively). Psychophysical measures were; thermal sensation (1–13, unbearably cold–unbearably hot), thermal discomfort (1–10, comfortable–extremely uncomfortable; both extended by Cotter & Taylor, 2005), and overall feeling state (+5 to  $-5$ , very good–very bad).

## 2.6 | Data and statistical analyses

Changes in Hct were used to calculate changes in plasma volume (PV) according to the formula of Strauss et al. (1951):

$$\% \Delta \text{PV} = 100 \times (1 - \text{Hct}_{\text{time}}) / (1 - \text{Hct}_{\text{baseline}}) - 100. \quad (1)$$

Using haemoglobin concentration to correct for differential effects of stress on mean red cell volume is not reported because of inadequate reliability in haemoglobin concentration (coefficient of variation: 4.8% (4.0, 6.3%)), and stability of hydration.

MAP was calculated as the sum of two-thirds diastolic blood pressure (DBP) and one-third systolic blood pressure (SBP).

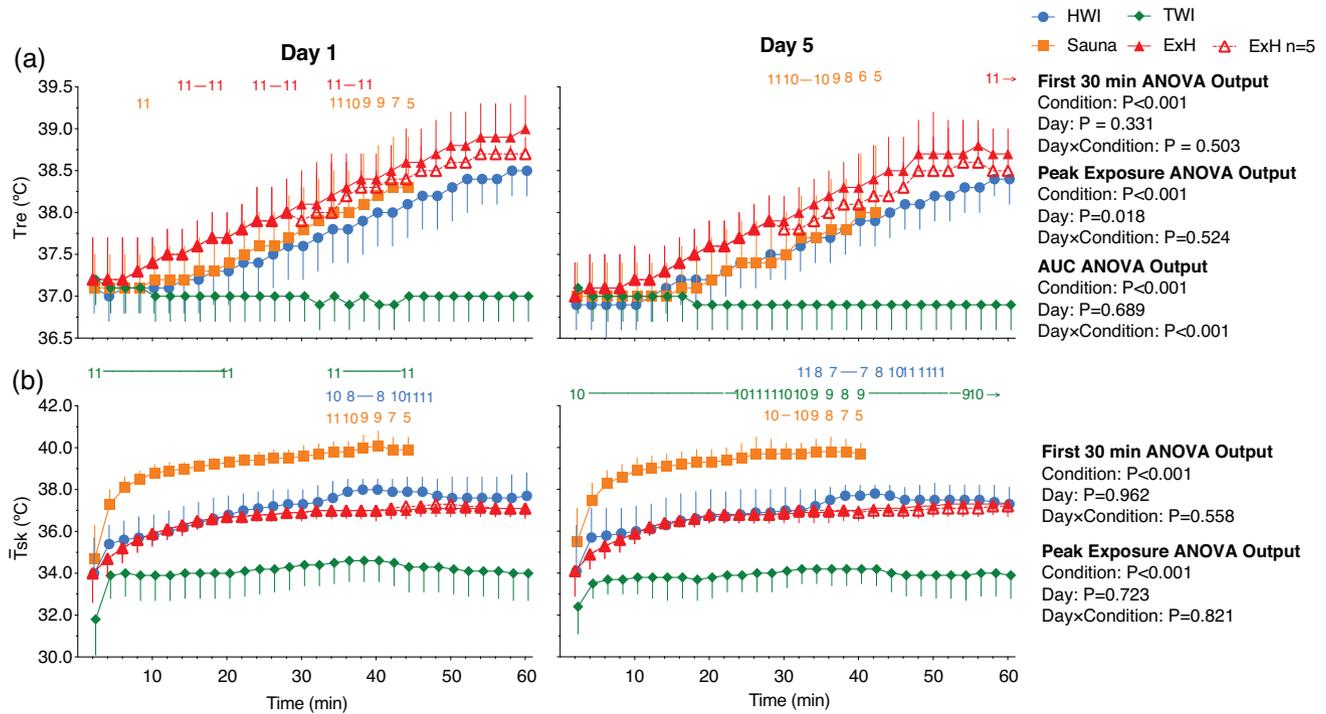
Mean skin temperature ( $\bar{T}_{sk}$ ) was estimated from area weightings, adapted from the formula of Ramanathan (1964):  $(0.2 \times \text{Calf}) + (0.2 \times \text{Thigh}) + (0.3 \times \text{Scapula}) + (0.3 \times \text{Forearm})$ . Rate of increase in  $T_{re}$  was calculated across 30 min, from 10 to 40 min of exposure.

Mean and peak myocardial workload of each condition was calculated using the rate pressure product (RPP) equation (heart rate  $\times$  SBP).

Effects of heating mode on strain *during* exposure were analysed via peak change and area under the curve (AUC; via trapezoid method), for reasons of maximising statistical power and representing the most physiologically plausible stimuli for adaptation (i.e., intensity and AUC, respectively). Effects of heating mode on recovery were analysed separately, at 45 min following end of exposure, because it addresses different questions/contexts.

Variables were analysed using linear mixed models. The heat mode (TWI, HWI, Sauna and ExH) and time point (day 1 and 5) were modelled as fixed effects, and participants (and associated interactions) were modelled as a random effect (where appropriate, see below). Order of condition was included to statistically account for carry-over effects, despite none being anticipated. Homogeneity of variances were assessed visually via plotting of residuals versus model-fitted values, and formally with Levene's test across all combinations of factors in the model. Linearity and approximate normal distribution of residuals were assessed via visual inspection of histograms and Q-Q plots of model and individual residuals, and formally with the Shapiro–Wilk test. Approximate normal distribution of random effects was assessed via visual inspection of Q-Q plots. Akaike's information criterion and model parsimony were used to determine variance/covariance structure of model errors, random and fixed effect structure, and model inclusion. Multiple comparisons were made using the estimated marginal means contrasts derived from the linear mixed models, and pairwise comparisons were made with the Tukey method. Mixed model analysis was performed using packages (Lenth, 2021; Pinheiro et al., 2020) developed for R and figures were generated using Inkscape (v1.0, Inkscape Developers, 2003) and GraphPad Prism (v8.4.3, GraphPad Software, La Jolla, CA, USA).

Descriptive statistics are reported as raw means  $\pm$  standard deviation (SD) in text, whereas comparisons of interest are reported as estimated marginal means with corresponding 95% confidence interval (lower limit, upper limit). To aid in interpretation, main effects and any associated interactions are provided in figures, while *post hoc* comparisons and associated statistics of significant factors are presented in text. For the sake of brevity, we have collated the major statistical comparisons into summary statements, the details and all comparisons of which can be found in Table 1 of the Supporting information.



**FIGURE 2** (a) Rectal temperature ( $T_{re}$ ) was elevated as rapidly as possible using each of hot water immersion (HWI), Sauna, and exercise in heat (ExH), or clamped at baseline using thermoneutral water immersion (TWI), in day 1 and day 5 exposures. (b) Mean skin temperature ( $\bar{T}_{sk}$ ) differed according to the mode of heating. All  $n = 13$  or 12 unless otherwise colour-coded above data point.  $n = 5$  from 30 min in ExH due to the difference in protocols on day 1. Note, data for Sauna from 44 min not shown as only four participants remained (and their  $T_{re}$  was modest; see Supporting information); at exit in ExH some participants were seated to clamp core temperature whilst some remained cycling, and; the rise in  $\bar{T}_{sk}$  at ~40 min in HWI and TWI due to time matched period that arm was in water. Data are means  $\pm$  SD

## 3 | RESULTS

### 3.1 | Exit times

Nine of the 13 participants could not tolerate 60 min in Sauna, and their tolerance time was lower on day 5 ( $42 \pm 12$  min) than on day 1 ( $45 \pm 11$  min;  $P = 0.031$ ). Thus, mean exposure time was 16 min less than in the other conditions ( $P < 0.001$ ). One participant finished at 50 min on day 5 of ExH because  $T_{re}$  reached  $40^\circ\text{C}$ , and one participant's day 5 Sauna data are excluded because of instability in environmental conditions, resulting in a reduced sample ( $n = 12$ ) for that exposure. All other sample sizes are  $n = 13$  for 60-min exposure and recovery points unless stated otherwise.

### 3.2 | Thermal (Figure 2)

Regardless of the day, rate of rise in  $T_{re}$  (from 10 to 40 min) was not significantly different between ExH and Sauna, but these were  $0.9^\circ\text{C}/\text{h}$  faster than in HWI and  $3.6^\circ\text{C}/\text{h}$  faster than in TWI, which remained stable (Day effect:  $P = 0.584$ ; Condition effect:  $P < 0.001$ ; Interaction:  $P = 0.867$ ). These outcomes all remained the same when baseline  $T_{re}$  was accounted for as a covariate. Peak  $T_{re}$  was  $0.2^\circ\text{C}$  ( $0.0, 0.3^\circ\text{C}$ ) higher on day 1 than on day 5 regardless of the condition ( $P = 0.008$ ). Similarly, regardless of the day, peak  $T_{re}$  was  $0.5^\circ\text{C}$  higher in ExH than in HWI and

Sauna, and  $1.8^\circ\text{C}$  higher than in TWI, whereas HWI and Sauna were  $1.3$  and  $1.2^\circ\text{C}$ , respectively, higher than in TWI (all  $P < 0.001$ ). These differences were retained when data were expressed as AUC of  $T_{re}$  (Day:  $P = 0.689$ ; Time:  $P < 0.001$ ; Condition:  $P < 0.001$ ).  $T_{re}$  for HWI and Sauna were not significantly different ( $0.1^\circ\text{C}$ ;  $P = 0.948$ ).

Peak  $\bar{T}_{sk}$  was not different between day 1 or 5 but was highest in Sauna ( $1.7$ – $5.2^\circ\text{C}$  above other conditions), followed by HWI ( $1.4$ – $3.5^\circ\text{C}$ ), and then ExH ( $2.1^\circ\text{C}$ ; all  $P < 0.001$ ).

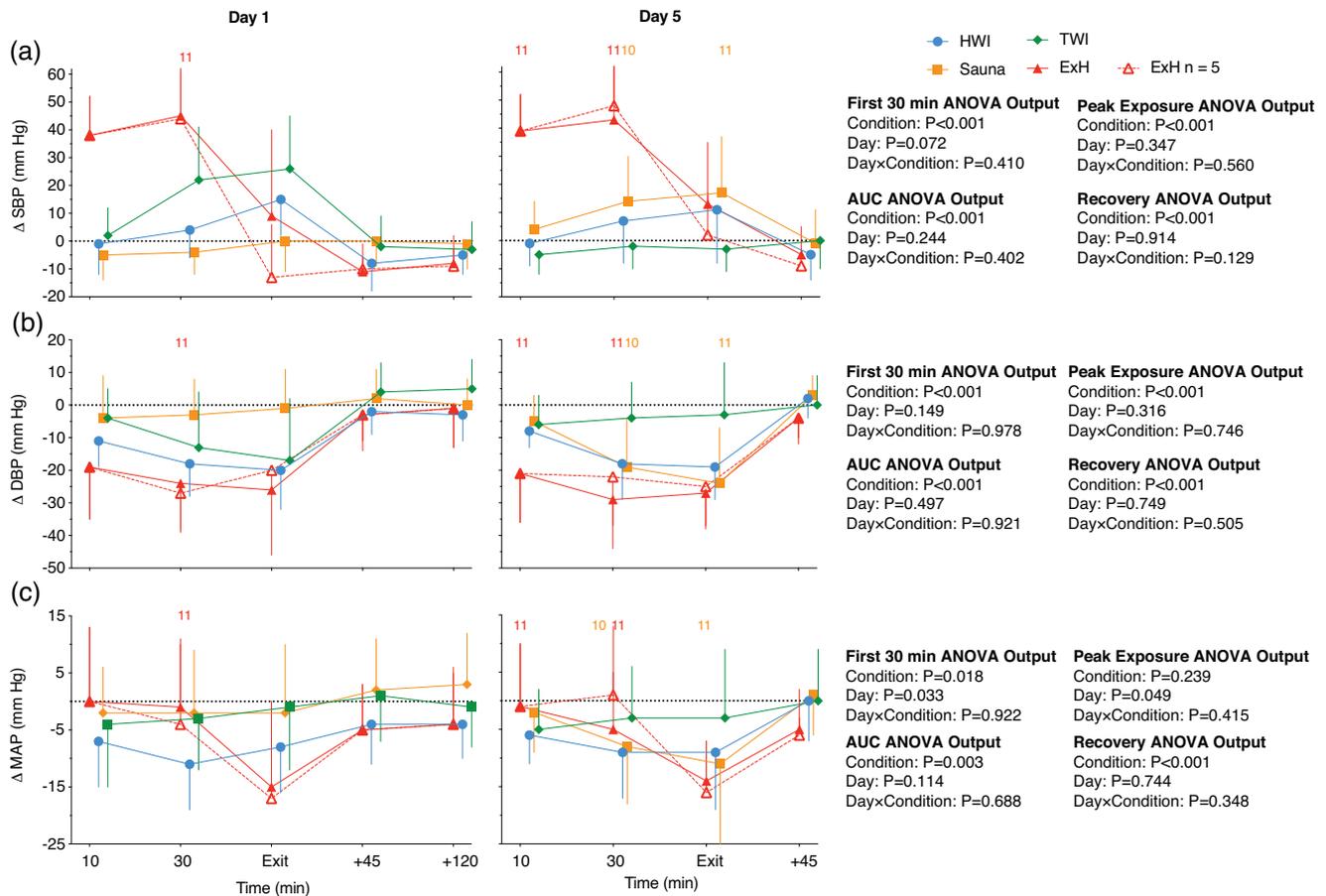
### 3.3 | Cardiovascular

#### 3.3.1 | Blood pressure (Figure 3)

The SBP, DBP and MAP response profiles between conditions were not dependent on the day of exposure, for either the peak exposure or subsequent recovery (Interactions:  $P \geq 0.129$ ). The responses below, therefore, represent the main effects of Condition and Day, separately for exposure and recovery.

#### Exposure

Peak SBP deviation was largest in ExH, increasing by an average of 18, 27 and 44 mm Hg more than in Sauna, HWI and TWI, respectively ( $P \leq 0.002$ ), followed by Sauna (by 9 and 26 mm Hg vs. HWI and TWI;  $P \leq 0.234$ ), and then HWI (by 17 mm Hg vs. TWI;  $P = 0.002$ ). The peak



**FIGURE 3** Change in blood pressure response from baseline during 60-min exposure and recovery on day 1 and 5 for four conditions: hot water immersion (HWI), Sauna, thermoneutral water immersion (TWI) and exercise in heat (ExH). (a) Systolic blood pressure ( $\Delta$ SBP) rose more during exercise than in passive air or water immersions, during both day 1 and day 5 exposures. (b) Diastolic blood pressure ( $\Delta$ DBP) was reduced in all conditions and showed the same pattern between conditions. (c) Mean arterial pressure ( $\Delta$ MAP) was reduced and did not depend on the mode of heating. All  $n = 12$  or  $13$  unless otherwise colour-coded above data point.  $n = 5$  from 30 min in ExH due to the difference in protocols on day 1. Note, at exit in ExH some participants were seated to clamp core temperature whilst some remained cycling. Data are means  $\pm$  SD

DBP responses also showed the largest effect for ExH, whereby the reduction in ExH was larger than in TWI (by 25 mm Hg;  $P = 0.001$ ) and HWI (by 9 mm Hg;  $P = 0.015$ ), and the reduction was larger in HWI and Sauna than in TWI (by 17 and 15 mm Hg, respectively;  $P \leq 0.001$ ). These differences in SBP and DBP were also apparent for the volume of strain (i.e., AUC). Peak MAP deviation did not differ between conditions, but the volume of hypotensive response was larger in HWI than in Sauna and TWI ( $P \leq 0.023$ ), but not different to ExH ( $P = 0.547$ ). No other differences between conditions were evident ( $P \geq 0.898$ ). All differences in blood pressure profiles were still apparent when controlling for baseline.

On average, neither peak increase in SBP nor decrease in DBP differed statistically between days, though MAP tended to decrease more on day 5 than on day 1 (4 mm Hg (0, 9 mm Hg);  $P = 0.077$ ).

#### Recovery

SBP, DBP and hence MAP in recovery (+45 min) were all 6–9 mm Hg lower in ExH than in Sauna and TWI ( $P \leq 0.009$ ). DBP and MAP tended to be lower following exposure to hot water (HWI) than air (Sauna), by

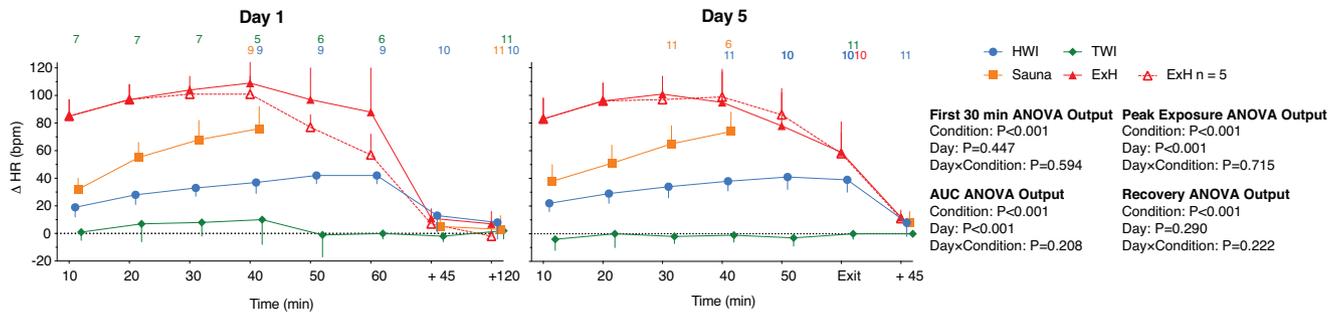
3–4 mm Hg ( $P \leq 0.107$ ). Blood pressures did not depend on the day of acclimation (all Day:  $P \geq 0.744$ ; Condition:  $P \leq 0.001$ ; Interaction:  $P \geq 0.129$ ).

### 3.3.2 | Heart rate (Figure 4)

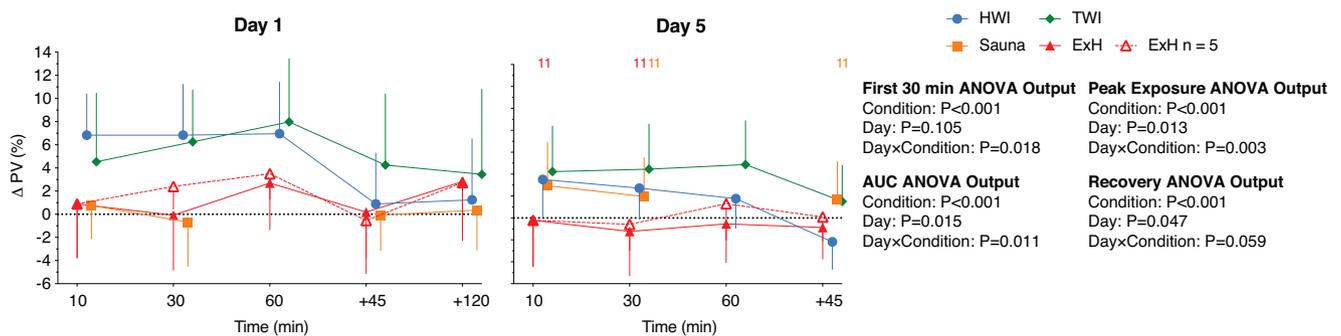
The heart rate profile across days did not depend on the condition (see Figure 4), and thus the responses below represent the main effects of Day and Condition for strain, that is, increase from baseline.

#### Exposure

On average, peak and AUC of heart rate responses were higher on day 1 than on day 5, regardless of whether baseline was controlled for (all  $P \leq 0.001$ ). The peak rise in heart rate was largest in ExH (by a mean of 34–111 beats/min more than all other conditions; all  $P < 0.001$ ), followed by Sauna (36–77 beats/min vs. HWI and TWI; all  $P < 0.001$ ), and then HWI (by 40 beats/min vs. TWI;  $P < 0.001$ ). The same pattern was evident when the data were expressed as an AUC (all  $P \leq 0.070$ ), and when baseline was accounted for.



**FIGURE 4** The rise in heart rate differed between all modes of heat exposure; exercise in heat (ExH), passive heat (Sauna), hot water immersion (HWI), and remained at baseline during thermoneutral water immersion (TWI), during day 1 and day 5 exposures. All  $n = 13$  or 12 unless otherwise colour-coded above data point.  $n = 5$  from 30 min in ExH due to the difference in protocols on day 1. Note, data for Sauna from 40 min not shown as only four participants remained (see Supporting information), and at exit in ExH some participants were seated to clamp core temperature whilst some remained cycling. Data are means  $\pm$  SD



**FIGURE 5** Water immersion acutely increased plasma volume ( $\Delta$ PV), regardless of whether the water was hot (HWI) or thermoneutral (TWI), whereas neither Sauna nor exercise in the heat (ExH) affected plasma volume. The immersion-induced expansion was blunted after five exposures.  $n = 13$  or 12 unless otherwise colour-coded above data point.  $n = 5$  from 30 min in ExH due to the difference in protocols on day 1. Note, data for Sauna at 60 min not shown as only four participants remained (see Supporting information), and; at exit in ExH some participants were seated to clamp core temperature whilst some remained cycling. Data are mean  $\pm$  SD

### Recovery

Heart rate in recovery was lower following TWI than the Sauna and ExH, and ExH was higher than Sauna, independent of day of exposure (Day:  $P = 0.290$ ; Condition:  $P < 0.001$ ; Interaction:  $P = 0.222$ ).

### 3.3.3 | Plasma volume (Figure 5)

Both the peak and AUC of change in PV during and after exposure differed between conditions and between days. So, each day is reported separately.

### Exposure

On day 1, PV peaked at 7–9% above baseline during immersion regardless of water temperature (i.e., HWI and TWI), and exceeded the small responses during Sauna and ExH (all  $P < 0.001$ ). On day 5, the peak increase in PV remained larger during TWI than ExH, by 6% (2, 10%) ( $P = 0.001$ ), but no other differences were evident. From day 1 to day 5, the PV peak was blunted by 4–5% in both water conditions whereas Sauna increased by 1% ( $P = 0.002$  and  $0.021$  for Sauna vs. HWI and TWI changes across days).

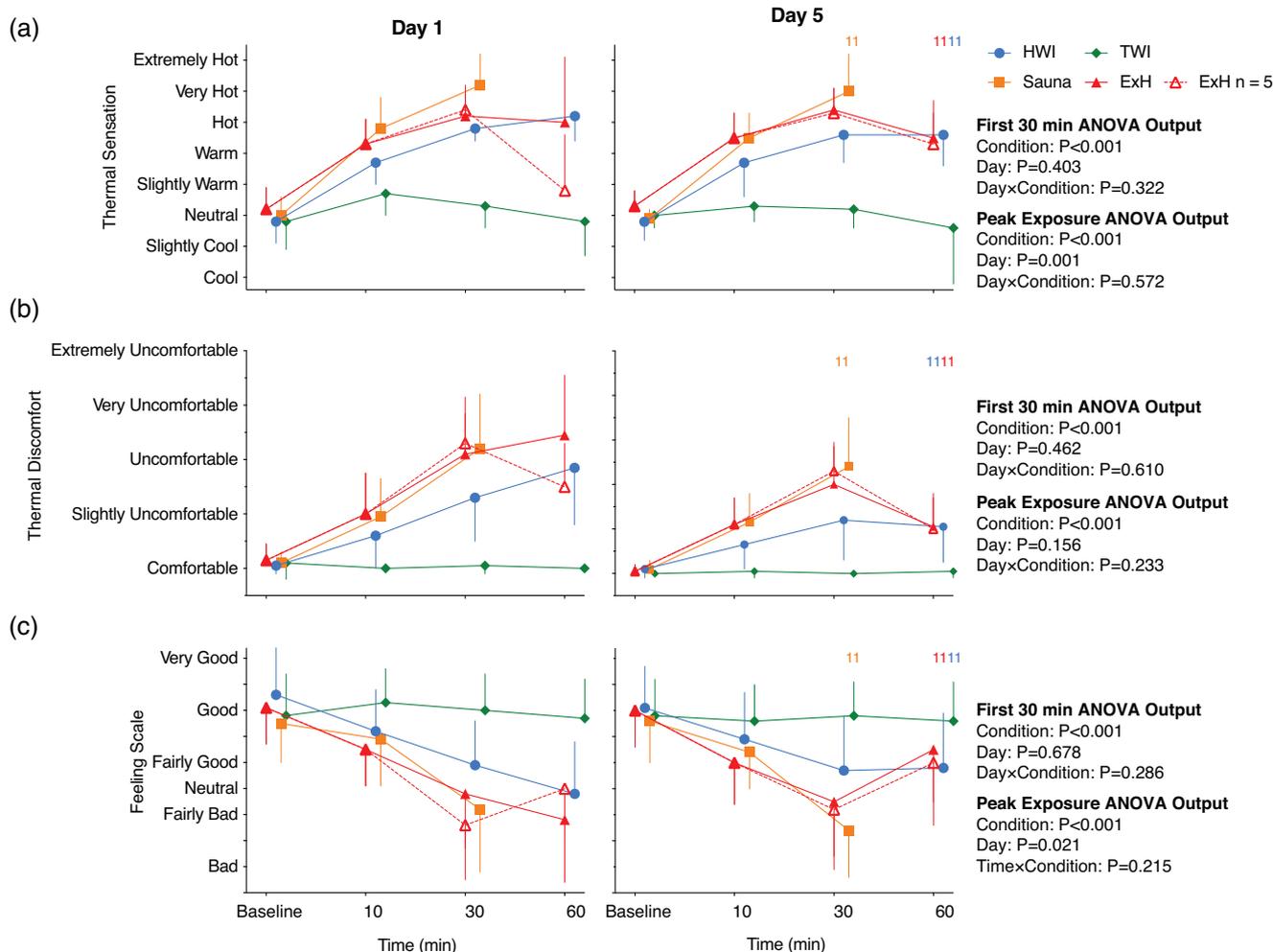
The AUC of change in PV showed the same findings as for peak PV changes, within each day and between days, except that on day 5 (i) both immersion conditions remained higher than for ExH (by 3–4%·h;  $P = 0.025$  and  $0.001$  for HWI and TWI, respectively), but (ii) the AUC of PV expansion in HWI was smaller than on day 1 ( $P = 0.001$ ).

### Recovery

Pooled across days, the PV at 45 min recovery was higher in TWI than in HWI (by 4% (1, 6%);  $P = 0.001$ ), and ExH (by 3% (1, 6%);  $P = 0.003$ ), but not Sauna (by 2% (0, 5%);  $P = 0.102$ ), nor were any other differences evident ( $P \geq 0.426$ ).

## 3.4 | Sweat rate

On average, sweat rate was largest in ExH, at 1.15 l/h (by 0.2 l/h vs. HWI and Sauna, and 1.3 l/h vs. TWI; all  $P < 0.001$ ), followed by HWI and Sauna at 0.98 and 0.79 l/h, respectively (by 1.1 l/h; both  $P < 0.001$  and  $P = 0.924$  between HWI and Sauna;  $n = 9$  for Sauna). On average, participants sweated more on day 5 than on day 1, by 0.1 l/h ( $P < 0.001$ ).



**FIGURE 6** Thermal sensation (a), thermal discomfort (b) and feeling scale (c) during exposure on day 1 and 5 for four conditions: hot water immersion (HWI), Sauna, thermoneutral water immersion (TWI) and exercise in heat (ExH). All  $n = 13$  or  $12$  unless otherwise colour-coded above data point.  $n = 5$  from 30 min in ExH due to the difference in protocols on day 1. Note, data for Sauna at 60 min not shown as only four participants remained (see Supporting information), and at exit in ExH some participants were seated to clamp core temperature whilst some remained cycling. Data are means  $\pm$  SD

Dehydration was modest in all three modes of heating, on average reaching  $-0.44\%$  body mass in HWI,  $-0.15\%$  body mass in Sauna and  $-0.49\%$  body mass in ExH.

### 3.5 | Psychophysical (Figure 6)

The psychophysical responses across the days of the interventions did not vary dependent on the condition (Interaction effects: all  $P \geq 0.215$ , see Figure 6). The responses below represent the main effects of Time and Condition for strain responses.

Peak perceived body temperature (thermal sensation) was higher on day 1 than on day 5 ( $P = 0.003$ ), but this did not translate to a difference in peak thermal discomfort between days (see Figure 6).

Peak feeling state was higher/better on day 5 than on day 1 ( $P = 0.021$ ). However, these mean differences in perceived body temperature and feeling state were all smaller than the smallest

scale units (i.e.,  $<1$  point), so may have little if any practical relevance.

Peak thermal sensation, discomfort, and feeling demonstrated similar average differences between conditions. For each psychophysical component, TWI demonstrated lower scores (or higher for feeling score) than every other condition (all  $P < 0.001$ ). This was followed by HWI (all  $P < 0.001$  for comparisons to all other conditions), and similar responses between Sauna and ExH for sensation, discomfort and feeling scores ( $P = 0.111, 0.973$  and  $1.000$ , respectively).

## 4 | DISCUSSION

This study examined physiological and psychophysical responses to three different modes of heat stress, all of which are commonly used for recreational, health or athletic conditioning purposes (HWI, Sauna

**TABLE 1** Peak and mean rate pressure product of exposure on day 1 and 5 of four different heat stress conditions

	Rate pressure product	
	Peak	Mean
<b>HWI</b>		
Day 1	13,978 ± 2189 (12)	11,773 ± 1921 (12)
Day 5	13,329 ± 2330 (13)	11,412 ± 1980 (13)
<b>Sauna</b>		
Day 1	21,139 ± 3364 (13)	16,697 ± 2464 (13)
Day 5	18,628 ± 3118 (12)	15,394 ± 2813 (12)
<b>TWI</b>		
Day 1	8,897 ± 2685 (8)	8120 ± 2404 (8)
Day 5	8181 ± 1992 (13)	7312 ± 1738 (13)
<b>ExH</b>		
Day 1	29,019 ± 2998 (13)	24,156 ± 3467 (13)
Day 5	27,638 ± 2511 (11)	22,198 ± 2358 (11)
<b>ExH (n = 5)</b>		
Day 1	28,792 ± 2413 (5)	22,030 ± 1914 (5)
Day 5	28,045 ± 2674 (5)	22,035 ± 1537 (5)
<b>Statistical overview</b>		
Condition	<0.001	<0.001
Day	0.011	0.026
Condition*Day	0.475	0.405

Data are means ± SD (n). ExH, exercise in heat; HWI, hot water immersion; TWI, thermoneutral water immersion.

and ExH). We examined effects of exposures up to 60 min of near-maximal and realistic heat loading, against a thermoneutral water immersion control condition, for both day 1 and day 5 exposures because repeated exposure can modulate both physiological and psychophysical responses to heating. The main findings were that (1) all three modes of heat stress elicited substantive heat strain (core temperature > 38.0°C,  $\bar{T}_{sk}$  > 36°C) and fluid loss (~1 litre) but markedly different cardiovascular strain (especially RPP), which was generally not attenuated after repeated exposures; (2) humid heat was poorly tolerated, which was not attributable to core hyperthermia or arterial hypotension and did not improve after multiple exposures; (3) immersion in hot water induced the least cardiac strain, in conjunction with hypervolaemia, which was mediated by immersion per se; whereas (4) exercise in the heat elicited the most cardiac, haemodynamic and fluid regulatory strain, and was the only mode to cause hypotension during recovery. Therefore, hypothesis 1 was only partially accepted as ExH had the largest post-exercise hypotension whereas the other hypotheses were not supported in that thermal, cardiovascular and psychophysical strain were not meaningfully attenuated on day 5 for any mode of heat stress.

The marked intolerance to very humid air (in which dew point was well above skin temperature, thus incurring rapid heating in the modest dry bulb temperature of 55°C) cannot be attributed to the rate of rise or actual core temperature (Figure 2), nor

hypotension (Figure 3). Skin temperature exceeded the rising core temperature throughout, and was 100% wetted, by design; all three of these thermal factors will have contributed to the substantial heat discomfort, especially as heat loss mechanisms were nullified (Vargas et al., 2020). All indices of psychophysical strain were worse in Sauna than HWI, and whilst not exceeding those of ExH, the only behavioural thermoregulation available during Sauna was to terminate exposure, which most participants did. The combined thermal and orthostatic stressors of Sauna resulted in moderate cardiac strain (RPP ~16,000; Table 1) and reflects the need to support cutaneous perfusion (Chou et al., 2018), which – paradoxically – exacerbates heat strain in this uncompensable environment. In a follow-up study focused on the cerebrovascular impact of one-off exposure to these three modes of heat stress maintained until thermal intolerance (Gibbons et al., 2021), the Sauna constrained cerebral conductance and uniquely reduced its perfusion, but still resulted in an elevated intracranial pressure index. Reduced cerebral perfusion will increase the temperature of the brain, a highly metabolic organ, and high brain temperature is a known mediator of heat intolerance (in rats: Walters et al., 2000). We, therefore, speculate that participants' limited tolerance of a humid Sauna may be attributable at least partly to accumulating brain temperature and pressure along with elevated autonomic strain and perceptual discomfort, although roles for other factors such as condensation in the respiratory tract remain possible.

HWI incurred large heat strain but with less discomfort and cardiac strain than during exercise or passive heat stress in humid air. These results support other recent findings and are not attributable to a slower rate of rise of core temperature (Figure 1) or the index of measurement, because the rate was matched in other studies that used either rectal (Francisco et al., 2021) or oesophageal (Gibbons et al., 2021) indices. This mode of heat stress confers high shear stress in the conduit arteries of both upper and lower limbs (Francisco et al., 2021) while not diminishing cerebral conductance or perfusion (Gibbons et al., 2021). The RPP was low, ~13,500 (Table 1) and just half that of exercise during both naïve and accustomed exposures, thereby reinforcing its merit as a low-risk form of therapy for cardiovascular health (Francisco et al., 2021). Immersion itself elicited an acute hypervolaemia during exposure (Figure 5). This hypervolaemia along with other haemodynamic effects of immersion – such as reduced transmural pressure and reduced sympathetic activation of systemic arterial vessels, along with external venous compression and an elevation in central blood volume – presumably contributed to the higher stroke volume and lower total peripheral resistance as reported in other studies (Francisco et al., 2021; Gibbons et al., 2021). We did not find a statistically significant post-exposure hypotension for HWI, which differs from other findings in young adults (Francisco et al., 2021) and our own experience of older participants whether healthy or having peripheral arterial disease (Akerman et al., 2019; Thomas et al., 2017). The discrepancy may reflect a type II error (Figure 3) or methodological differences such as less constraint on post-exposure posture and activity, or participants' age or fitness (e.g., higher baseline blood volume).

Exercise in the heat elicited the highest sweat rate and heart rate, and was the only condition to substantively raise SBP, thereby generating a RPP larger than that during Sauna and twice of that during HWI (Table 1). Exercise in the heat is a more complex stress than either Sauna and HWI, eliciting additional feedforward and feedback activation for sweat glands, chronotropy, inotropy and systemic vasoconstriction to counteract locally mediated vasodilatation in active muscle and redistribute perfusion in favour of muscle and skin. In this regard, the present results broadly support findings of Francisco et al. (2021) and Gibbons et al. (2021).

The post-exercise hypotension (averaging  $\sim 6$  mm Hg) was expected and has been shown previously to reflect both a sustained vasodilatation within previously active musculature and a potentially lower centrally defended pressure. Finally, the wide spectrum of high physiological strain invoked by exercise in the heat, particularly for more aerobically fit individuals, may generate a more effective stimulus for adaptation than is obtained by passive exposure to hot water or humid heat (Figures 1–3; Convertino et al., 1980; Kissling et al., 2019).

#### 4.1 | Limitations

Specific study limitations should be acknowledged. Firstly, rate of heating was not fully matched between conditions or between participants, despite pilot research aimed at matching between conditions. Fortunately, this does not appear to have affected the main findings, as revealed by other recent studies (discussed above). The last half of the initial exposure (day 1) in two modes of heat stress (HWI and ExH) was a self-regulated exposure for eight participants, for logistical reasons described above. This limited statistical power for related comparisons, although this did not create any disparity with the Sauna because none of these eight participants experienced more heat strain during Sauna (i.e., their tolerance and rates of heating both prevented this outcome). However, the lack of matching of Sauna exposure between the 13 participants is a potential limitation, but is also a valid outcome of that form of heat exposure; tolerance was poor throughout, and notably, got worse rather than better over the 5 days despite a reduction in resting  $T_{re}$ . Calculating the change in PV from haematocrit alone ignores effects of dehydration or other osmotic stressors. We had measured haemoglobin concentration to minimise such influence, but the lower reliability with such correction was considered more problematic than the lower validity it provided, particularly given the modest changes in body mass in these 60-min exposures.

#### 4.2 | Perspectives/implications

Short-term adaptation effects of the different modes of heating are addressed in the companion paper (Kissling et al., 2022), and the haemodynamic stimuli for health-related effects were recently reported by Francisco et al. (2021). Relevant to both of these contexts, the present study demonstrates that thermal AUC of core

temperature, as a primary stimulus for adaptation, was not dampened with multiple exposures – perhaps unsurprising given their thermal uncompensability. Nor was there an attenuation of the magnitude of thermal discomfort or its associated negative affect, which is important for at least two reasons. First, discomfort comprises humans' main stimulus for protection (behavioural heat regulation) against acute heat injury and is therefore useful. Second, it can diminish people's adherence to repeated (i.e., chronic) exposures particularly for individuals who are unaccustomed to stressful exposures. This signifies (1) the importance of using progressive heat stress (duration and/or intensity, which is seldom factored into heat acclimation studies, for example), and (2) a need for research to determine dose–response relations of mechanistic acute and adaptive physiological and psychophysical responses to heating.

The markedly lower cardiac work rate (indicated by RPP) of HWI relative to exercise has clear advantages for the safety of cardiovascular conditioning in unconditioned and unselected adult populations, as pointed out by Francisco et al. (2021). This lower myocardial workload may not concomitantly prevent useful ventricular adaptation because ventricular heat stress appears to be more important than its work rate in stimulating the production of stress proteins (Staib et al., 2007). While this hypothesis is generated based on prior rodent research, it is an important topic for future investigation, namely, whether cardiac adaptation is blunted when exposed to relatively less cardiac work rate, or whether the adaptive stimulus of tissue temperature overrides this necessity?

Sauna was markedly less tolerable than other forms of heat stress, which has scientific and practical implications. Notable for both of these contexts, the environment in the present study caused high rates of condensation (and thus heat gain) onto skin and airway membranes, more so than in the follow-on study, for which tolerance was higher (Gibbons et al., 2021). In contrast, classic/Scandinavian saunas facilitate strong evaporation but have higher dry bulb and hence radiant temperatures that facilitate rapid heating (with low skin wittedness) – dictating that exposure time is limited. Notably, in such cultural contexts, one is exposed to these conditions for a much briefer period than the current conditions. As above, the implications for adaptation will depend on the relative importance and time and intensity dose–response relations of target outcomes (e.g., vascular shear stress vs. activation of cellular stress responses in hot and ischaemic downstream organs).

In the context of heating for health or acclimation purposes, any of these modes is effective in elevating core temperature, although it should be borne in mind that these participants were relatively fit (generating  $\sim 7$  W/kg of thermal energy) and exercising in unusually high (rarely available) heat stress. This has at least two implications. First, a mixed-heating protocol might be more effective for less fit individuals, notwithstanding that cardiovascular strain is also an important component of heat acclimation, and that the optimal intensity of heat strain has still not been determined. Second, passive heating will be far more effective for raising core temperature in unfit or clinical populations, but will not substitute for exercise in other respects (Cullen et al., 2020).

### 4.3 | Conclusions

All three common modes of heating (sauna, hot water immersion and exercise in the heat) elicited high skin and core temperatures, but exercise was the only mode to elicit high cardiac strain, that is, high systolic pressure and heart rate, and hence estimated myocardial oxygen demand. This was also the only mode to elicit hypotension during recovery, whereas only hot water immersion induced PV expansion during exposure, which was due to immersion per se. Profiles of strain were generally similar following repeated exposures.

### ACKNOWLEDGEMENTS

We thank the participants, the School of Physical Education, Sport and Exercise Sciences for funding support.

Open access publishing facilitated by University of Otago, as part of the Wiley – University of Otago agreement via the Council of Australian University Librarians.

### COMPETING INTERESTS

The authors declare no competing interests.

### AUTHOR CONTRIBUTIONS

J.D.C., A.P.A., L.S.K., J.R.P. and K.N.T. contributed design of the study. L.S.K., H.A.C., J.R.P., T.D.G., A.P.A. and J.D.C. collected the data at the School of Physical Education, Sport and Exercise Sciences, University of Otago. H.A.C., L.S.K., A.P.A., J.D.C., T.D.G. and K.N.T. contributed to analysis and/or interpretation of the work. H.A.C. J.D.C., A.P.A. and L.S.K. wrote the first draft of the manuscript. All authors provided critical feedback and approved the final version of this manuscript and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

### DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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**How to cite this article:** Campbell, H. A., Akerman, A. P., Kissling, L. S., Prout, J. R., Gibbons, T. D., Thomas, K. N., & Cotter, J. D. (2022). Acute physiological and psychophysical responses to different modes of heat stress. *Experimental Physiology*, 107, 429–440. <https://doi.org/10.1113/EP089992>