

Research article

Contrast Heat–Cold Versus Thermoneutral Showering in Trained Combat Sport Athletes: A Randomized Field Trial of Recovery Outcomes

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Abstract

Contrast heat-cold showering is popular for recovery, but multi-system evidence and persistence after cessation remain unclear. To evaluate whether a 4-week post-training contrast heat-cold shower intervention produces an integrated recovery profile across four domains (autonomic, endocrine, perceptual, and microvascular) rather than testing a single isolated physiological pathway, across baseline (T0), post-intervention (T1), and 2-week wash-out (T2). Sixty combat-sport athletes were randomized to contrast showers (10 min alternating warm 38 - 40°C and cold 13 - 15°C) or an active thermoneutral-shower comparator (10 min, 32 - 34°C) after training for 4 weeks. Primary analyses were per-protocol ($\geq 75\%$ compliance; $n = 57$), with intention-to-treat sensitivity analyses. Outcomes were Total Quality Recovery (TQR), morning salivary cortisol (two mornings averaged), resting HRV, and post-occlusive reactive hyperemia (PORH). From T0 to T1, favorable between-group changes were observed for resting HR ($\Delta\Delta -2.10$ bpm, 95% CI -2.24 to -1.96; $g -0.65$; $p < 0.001$), $\ln\text{RMSSD}$ ($\Delta\Delta 0.123$ log units, 95% CI 0.108 to 0.137; $g 0.74$; $p < 0.001$), with similar T1 effects for RMSSD and SDNN, and TQR ($\Delta\Delta 0.61$ points, 95% CI 0.27 to 0.95; $g 0.80$; $p < 0.001$). These T1 autonomic and perceptual changes were not maintained at T2 (all T2-T0 $\Delta\Delta p > 0.05$). Cortisol and PORH-derived outcomes showed no statistically clear between-group differences at T1 or T2 (all $p > 0.05$). Compared with thermoneutral showering, 4 weeks of post-training contrast heat-cold showering produced short-term favorable between-group changes in autonomic regulation and perceived recovery, but not in morning cortisol or PORH-derived microvascular reactivity. These effects were not maintained after wash-out; therefore, causal attribution to the shower intervention alone and claims of persistent physiological adaptation should be made cautiously. Trial registration: ISRCTN15418049.

Key words: Recovery, combat sports, heart rate variability, post-occlusive reactive hyperemia.

Introduction

High-performance combat sports impose repeated, high-intensity mechanical and metabolic stressors that require efficient recovery to sustain training quality and reduce the risk of maladaptive fatigue (Ostapiuk-Karolczuk et al.,

2025). These stressors are distinctive because combat athletes are exposed to repeated grip fighting, clinch and pulling actions, striking impacts, dense sparring blocks, mixed aerobic-anaerobic energy demands, and, in many competitive contexts, weight-management practices that may further challenge hydration, sleep, endocrine regulation, and perceived recovery (James et al., 2016; Franchini, 2023). Monitoring recovery using objective physiological markers is increasingly emphasized because subjective recovery alone can diverge from underlying stress-adaptation biology in athletes (Coutts et al., 2021). Accordingly, sport science encourages integrated evaluation across vascular, autonomic, and endocrine systems to better characterize recovery status and training adaptation (Lee et al., 2017). In this contexts, autonomic indices reflect cardiac regulatory state and parasympathetic reactivation, endocrine markers provide information about systemic hypothalamic-pituitary-adrenal-axis load, perceptual scores capture the athlete's conscious recovery appraisal, and microvascular measures represent local perfusion responsiveness. These domains are related but not interchangeable, and they may change at different rates after training or recovery interventions (Babos, 2013; Mishica et al., 2021).

Autonomic regulation is a central component of recovery physiology, and resting heart rate variability (HRV) provides a noninvasive window into cardiac autonomic modulation in athletes (Aubert et al., 2003). Time-domain vagal indices such as RMSSD and $\ln\text{RMSSD}$ are widely used in applied monitoring because they are sensitive to training load and recovery fluctuations in athletic populations (Esco and Flatt, 2014). In parallel, salivary cortisol reflects hypothalamic-pituitary-adrenal axis activity and has been repeatedly used as a marker of training-related stress and recovery balance in sport contexts (Cevada et al., 2014). However, basal morning cortisol should be distinguished from acute cortisol stress reactivity since morning values primarily index the basal circadian/endocrine state under standardized sampling conditions, whereas acute reactivity requires repeated sampling around a defined stressor or exercise bout (Díaz et al., 2013; Cevada et al., 2014). If a recovery intervention meaningfully reduces

accumulated physiological strain, the expected direction would generally be a lower or more stable morning cortisol profile relative to control, rather than a transient post-exposure cortisol spike (Cevada et al., 2014). Nevertheless, cortisol is strongly influenced by circadian timing, sleep, psychosocial stress, and recent training load, so small intervention effects may be difficult to detect in field scenarios (Bonato et al., 2017). Microvascular regulation is mechanistically relevant to recovery because it governs tissue perfusion dynamics that influence oxygen delivery and metabolite clearance following loading (Morales et al., 2005). Post-occlusive reactive hyperemia (PORH) assessed with laser Doppler methods is a well-established approach to quantify microvascular reactivity and has demonstrated reproducible parameterization for research applications (Yvonne-Tee et al., 2005). Inter-day reproducibility of PORH has also been documented in methodological work, supporting its use for longitudinal designs that test physiological adaptation over time (Roustit et al., 2010b). Because laser Doppler PORH primarily quantifies cutaneous microvascular perfusion at the measurement site, it should not be interpreted as a direct measure of muscle oxygenation or systemic cardiovascular function (Fabregate-Fuente et al., 2019).

Contrast water therapy is widely implemented as a pragmatic recovery strategy, and systematic review evidence indicates it can influence recovery-related outcomes after exercise-induced muscle damage, although study quality and effects vary (Bieuzen et al., 2013). Meta-analytic findings in sport contexts similarly suggest potential benefits of cold and contrast water interventions for recovery, but outcomes depend on protocols, comparators, and the specific endpoints measured (Higgins et al., 2017). Importantly, much of the existing evidence concerns cold-water or contrast-water immersion (Wilcock et al., 2006), whereas the research in contrast heat-cold showering is limited (Shadgan et al., 2018). Immersion and showering are not physiologically equivalent since immersion provides hydrostatic pressure, greater body-surface contact, and different heat-transfer and tissue-cooling characteristics, whereas showering mainly delivers a surface thermal stimulus without the same hydrostatic loading (Torres-Ronda and Schelling i del Alcázar, 2014). Therefore, immersion findings are transferable to shower protocols only at the level of shared alternating thermal exposure and plausible thermoregulatory/autonomic stimulation, not at the level of hydrostatic-pressure-mediated fluid shifts or whole-limb immersion effects (Wilcock et al., 2006; Shadgan et al., 2018).

Plausible explanations commonly propose that alternating thermal exposure induces cyclic vasoconstriction-vasodilation and modifies local hemodynamics, yet direct multi-system validation in applied athletic scenarios remains limited (Shadgan et al., 2018). Moreover, cutaneous blood flow, intramuscular oxygenation, and systemic cardiovascular responses may not change in parallel because they represent different compartments and regulatory mechanisms, therefore using PORH assessment can be relevant to represent cutaneous microvascular reactivity (Shadgan et al., 2015; 2018).

In combat-sport context, a recent single-blind randomized trial in MMA-trained athletes reported that partial contrast-water immersion produced acute shifts in forearm muscle mechanics, reducing stiffness/elasticity indices while increasing pressure pain threshold and maximal isometric force within ~5 - 60 minutes post-treatment (Trybulski et al., 2025). Given the central role of forearm flexor function for gripping and clinch control, these short-term biomechanical changes are plausibly relevant to session-to-session readiness in combat sports, even though they do not establish longer-term multi-system adaptation (Trybulski et al., 2025). This combat-sport-specific evidence motivates the present trial because it suggests that thermal contrast may influence recovery-relevant perception and tissue-level function in athletes exposed to repeated gripping and striking demands. However, it does not determine whether a more feasible shower-based protocol produces coordinated autonomic, endocrine, perceptual, and cutaneous microvascular changes over repeated post-training use.

Critically, most contrast-water research has not tested whether repeated exposure produces coordinated adaptations across microvascular reactivity, autonomic modulation, and endocrine stress markers, nor whether any changes persist after stopping the intervention (Higgins et al., 2017). This limits interpretation and practical decision-making for coaches who need to know whether contrast showers produce only transient effects or longer-lasting adaptive responses (Higgins et al., 2017). The exact gap addressed here is whether repeated post-training contrast heat-cold showering, compared with thermoneutral showering, produces measurable multisystem recovery changes in trained combat-sport athletes.

Therefore, the objective of this study was to evaluate the effects of a four-week post-training contrast heat-cold shower intervention, compared with an active thermoneutral-shower comparator, on resting autonomic modulation, perceived recovery, morning salivary cortisol, and cutaneous microvascular reactivity across three time points (T0 baseline, T1 post-intervention, T2 after wash-out). The primary contrasts were the between-group differences in change from T0 to T1 and from T0 to T2. Resting heart rate variability (HRV) indices and total quality recovery (TQR) were treated as the main recovery outcomes of interest because they were expected to be most responsive to short-term recovery-state modulation, whereas morning cortisol and PORH-derived cutaneous microvascular outcomes were interpreted as complementary endocrine and microvascular domains within the integrated recovery profile. We hypothesized that, compared with thermoneutral showering, contrast heat-cold showering would produce favorable T0-to-T1 between-group changes in resting autonomic indices and perceived recovery, with smaller or less consistent changes in morning cortisol and PORH-derived cutaneous microvascular outcomes. The two-week wash-out was used to test whether any observed effects persisted after cessation of the shower protocol.

Methods

Study design and setting

This study was a randomized, field-based, parallel-group trial conducted under real-world training conditions in trained combat sport athletes recruited from two clubs located in Cieszyn and Żory, Poland. The trial was prospectively registered at ISRCTN (ISRCTN49499065) on 23 June 2025, before recruitment of the first participant, as part of a broader randomized controlled research programme evaluating different recovery strategies in physically active individuals. Ethical approval was granted by the Ethics Committee for Scientific Research of Physiotherapists of the Polish Physiotherapy Association (Resolution No. 1.06.2025, approved on 11 June 2025). All procedures were conducted in accordance with the Declaration of Helsinki. All participants provided written informed consent before enrolment after receiving detailed information about the study procedures.

Recruitment was conducted between 22 September 2025 and 20 October 2025. Baseline assessments were performed immediately after enrolment between 22 September and 24 October 2025. Each participant subsequently completed a 4-week intervention followed by a 2-week wash-out period. Final follow-up assessments were completed on 5 December 2025. The control condition consisted of thermoneutral showering and should therefore be interpreted as an active comparator rather than a no-treatment control. Consequently, the study does not estimate the absolute efficacy of contrast heat-cold showering versus no intervention, but rather its relative effect compared with an equivalent-duration thermoneutral shower routine. All physiological and subjective assessments were performed at the same external medical facility (Provita Medical Center, Żory) under standardized clinical conditions to minimize site-related variability in measurement procedures.

The experimental period consisted of a 4-week intervention followed by a 2-week wash-out period to evaluate persistence or reversibility of effects after cessation of the recovery protocol, with measurements at three predefined time points: baseline (T0), immediately post-intervention (T1), and post wash-out (T2).

Participants and eligibility criteria

A convenience sampling approach was used, recruiting athletes from local combat-sport academies/gyms and training centers through direct contact with coaches, on-site announcements, and study flyers/shared posts on academy communication channels. Because recruitment was conducted through open club announcements, coach-mediated invitations, and shared academy communication channels, the total number of athletes exposed to recruitment materials could not be precisely enumerated. Sixty athletes formally contacted the research team, underwent eligibility screening, met the inclusion criteria, provided written informed consent, and were randomized. No athlete was excluded at formal screening. Reasons for non-participation among athletes who may have seen recruitment materials but did not contact the research team were not recorded. Interested athletes completed an eligibility screening (training history, health status, and medication use) and, if eligible, provided written informed consent before enrollment. Sixty combat sport athletes (49 men, 11 women) were recruited. Athletes were actively training throughout

the study and represented Brazilian jiu-jitsu ($n = 18$), kick-boxing ($n = 15$), karate ($n = 14$), and mixed martial arts ($n = 13$), with at least 3 years of continuous training experience and a minimum habitual training frequency of three sessions per week. Participants were classified as Tier 3 - 4 athletes (trained to highly trained) according to the McKay Framework (McKay et al., 2022), reflecting systematic involvement in organized sport and a high level of training status. In female participants, menstrual-cycle phase and hormonal contraceptive status were not used as eligibility criteria and were not standardized for the timing of T0, T1, or T2 testing.

Inclusion criteria were age ≥ 18 years and the absence of acute musculoskeletal injury at enrollment. Exclusion criteria included known cardiovascular, neurological, or metabolic disorders and the use of medications likely to influence autonomic regulation or vascular function, in order to reduce confounding of HRV and microvascular endpoints.

Sample size planning

An a priori sample size was determined to ensure adequate power to detect the primary longitudinal treatment effect, defined as the between-group difference in change ($\Delta\Delta$) corresponding to the Group $\times$ Time interaction in the planned longitudinal models. The sample-size calculation was anchored to lnRMSSD, which was selected as the primary outcome for sample-size planning because it represents a vagally mediated HRV index, is commonly used in applied athlete recovery monitoring, and was expected to be more responsive to short-term recovery-state modulation than morning cortisol or PORH-derived cutaneous microvascular outcomes. For planning, the primary power-driving contrast was the between-group $\Delta\Delta$ in lnRMSSD from baseline to post-intervention (T1-T0). The T2-T0 contrast was retained as the planned persistence contrast, but the sample-size calculation was based on detecting the post-intervention lnRMSSD effect. We used a conservative change-score framework, with two-sided $\alpha = 0.025$ to account for the two planned temporal contrasts (T1-T0 and T2-T0) and 80% power. The smallest effect considered practically relevant was set to a moderate standardized $\Delta\Delta$ of $d = 0.60$, defined as the between-group difference in lnRMSSD change standardized by the pooled baseline SD. This value was considered consistent with the magnitude of autonomic effects reported in water-based post-exercise recovery contexts and allowed for additional variability expected under real-world training conditions (Ravier et al., 2022; Ahokas et al., 2025). Under these assumptions, 55 participants were required. Therefore, 60 athletes were randomized to allow for non-compliance or attrition. The study was not powered independently for TQR, salivary cortisol, or PORH-derived outcomes, which were analyzed using the same longitudinal rationale and interpreted using effect estimates, confidence intervals, and standardized effects.

Randomization, allocation concealment, and blinding

Participants were randomly allocated to a contrast heat-cold recovery group or a control group. The allocation sequence was generated before enrollment by an independent

researcher who was not involved in recruitment, intervention delivery, outcome assessment, or data analysis. Randomization was stratified by club and implemented with proportional sex allocation within each club. The random sequence was generated using Research Randomizer online application with simple stratified 1:1 allocation. Randomization was conducted separately within each club to minimize club-specific effects and to achieve balanced allocation, with proportional distribution by sex within each club. For sex allocation, separate allocation lists were prepared within each club-by-sex stratum. Allocation concealment was maintained using sequentially numbered, opaque, sealed envelopes, which were opened only after eligibility confirmation, written consent, and baseline testing. Outcome assessors remained blinded to group allocation throughout physiological testing. Due to the nature of the recovery intervention, participant blinding was not feasible. To reduce detection bias, all physiological outcome assessments were performed by a blinded medical team (physiotherapists and nurses) who were unaware of group allocation, and all assessments were carried out at the same medical facility using identical environmental and organizational procedures.

Intervention and control conditions

Contrast water group

The recovery intervention consisted of contrast heat-cold water exposure applied after training sessions. The protocol was based on commonly described contrast-water therapy procedures used in sports and rehabilitation research and was designed to provide a meaningful thermoregulatory and vascular stimulus while remaining feasible in club-based settings. The intervention lasted 10 minutes and involved alternating exposures to warm and cold water in a 1:1 ratio. The protocol always started with warm-water exposure and ended with cold-water exposure, consistent with standard contrast water-therapy approaches aimed at recovery. This sequence is recommended to enhance tolerance of the intervention, prepare the vascular system for thermal stress, and promote vasoconstrictive responses during the final phase of exposure. The sequence consisted of five cycles of 1 minute of warm water followed by 1 minute of cold water.

The water temperature was maintained at 38–40°C, a range shown to induce peripheral vasodilation without excessive thermal strain. The cold-water temperature was maintained between 13 and 15°C, a range commonly reported in the literature as sufficient to elicit vascular and sensory responses while remaining tolerable during repeated exposure. Prior to the start of the intervention period, water temperatures were measured and verified in both clubs using calibrated thermometers to establish reference values and confirm the ability to maintain target temperatures. Implementation of the protocol was facilitated by the thermostatic mixing valves installed in the shower facilities of both clubs.

Exposure time was controlled using portable timers installed in the shower area. Participants were instructed to self-monitor the duration of each phase and to switch water temperature precisely every 60 seconds according to the predefined protocol. Before the intervention period, all

participants received standardized instructions and a brief practical demonstration on how to correctly perform the contrast procedure and control both time and temperature. The shower sessions were self-administered by athletes in the club shower facilities after standardized instruction. Protocol fidelity was supported by coach/research-team contact, weekly adherence recording, portable timers, thermostatic shower controls, and periodic temperature and timing spot checks. Participants were instructed to complete the assigned shower as soon as possible after each scheduled training session, before leaving the club facilities and before undertaking any additional recovery modality. The time from training cessation to shower initiation was within 5 minutes. The intended exposure was whole-body showering, with participants instructed to expose the trunk and upper and lower limbs as uniformly as possible during each phase. Participants completed the assigned shower protocol in the same club shower facilities in which the intervention had been standardized.

During the 4-week intervention, protocol fidelity was monitored using periodic spot checks of water temperature and phase timing. Water temperature was checked twice weekly in both clubs using calibrated thermometers, and phase duration was verified using the installed timers. Recorded temperatures remained within the prespecified ranges for warm exposure ($38.6 \pm 0.9^\circ\text{C}$), cold exposure ($14.1 \pm 0.7^\circ\text{C}$), and thermoneutral control showers ($33.3 \pm 1.1^\circ\text{C}$).

Thermoneutral shower control

The thermoneutral shower control condition consisted of a 10-minute shower using thermoneutral water intended to avoid eliciting marked thermoregulatory or vascular responses. Water temperature in the control condition was maintained between 32 and 34°C, a range commonly described in the literature as thermoneutral for human skin and not inducing marked vasodilation or vasoconstriction. As in the intervention group, shower duration in the control condition was controlled using timers, and participants followed standardized instructions. The use of a fixed sequence of thermal exposures, standardized temperature ranges, and controlled exposure times allowed for consistent application of both intervention and control protocols across participants and clubs, thereby reducing variability in the execution of the procedures.

The recovery intervention was performed after every scheduled training session throughout the intervention period. Training frequency at both clubs typically ranged from 3 to 5 sessions per week, and the contrast heat-cold protocol was applied after each completed training session. Consequently, participants were exposed to the recovery intervention up to five times per week during the four-week intervention period. Attendance at training sessions and completion of the assigned recovery protocol were recorded weekly. Missed training sessions and omitted recovery interventions were documented, allowing for the calculation of individual compliance with the intervention protocol.

As in the contrast heat-cold shower group, thermoneutral shower control sessions were self-administered in the standardized club shower facilities after scheduled

training sessions, using portable timers to standardize the 10-minute duration. Participants were instructed to expose the body as uniformly as possible under the shower stream and to complete the assigned shower before leaving the club facilities.

Adherence monitoring and analysis populations

The recovery protocol was performed after every scheduled training session during the 4-week intervention period. Typical training frequency was three to five sessions per week, resulting in up to five recovery exposures weekly, and attendance plus completion of the assigned recovery protocol were recorded weekly. Adherence was calculated as the percentage of completed assigned shower sessions relative to the number of scheduled eligible post-training shower sessions during the intervention. A per-protocol threshold of $\geq 75\%$ compliance was predefined to ensure adequate exposure to the stimulus; participants not meeting this threshold were excluded from per-protocol analyses, while their data were retained for complementary intention-to-treat analyses when appropriate.

Standardization of training context and monitoring of training load and fatigue

The study was conducted under natural training conditions, with athletes continuing their usual sport-specific programs prescribed by their clubs, and no study-driven modifications to training volume, intensity, or structure were imposed. To reduce confounding from intergroup differences in training exposure, both clubs implemented a comparable weekly microcycle structure consisting of approximately 90-minute primary sessions, including task-based training, sparring, general motor preparation/cross-training-type work, and grappling/wrestling technique sessions, with an optional low-intensity session on Saturday and a scheduled rest day on Sunday.

Subjective internal training load was monitored using the session-RPE approach. After each training session, athletes recorded session-RPE in training diaries and reported weekly summaries to coaches, who forwarded the data to the research team. Weekly session-RPE training load was calculated as session duration in minutes multiplied by session-RPE and by the number of recorded sessions completed during that week (Haddad et al., 2017). The primary club sessions were approximately 90 minutes, weekly load was expressed in arbitrary units per week. Diary completeness was checked weekly. No missing entries were observed. Training frequency, mean session-RPE, weekly session-RPE load, fatigue, wellness, and shower compliance were summarized by group across the intervention.

Measurement procedures and testing environment

All assessments followed a standardized protocol in clinical rooms with ambient temperature maintained at 21 - 23°C and relative humidity 40 - 60% to minimize environmental influences on vascular and autonomic measures. The same pre-assessment restrictions were applied to PORH, HRV, and cortisol testing, i.e., participants were instructed to avoid vigorous exercise, caffeine, nicotine, alcohol, and heavy meals before testing, and compliance

with these restrictions was confirmed verbally at each visit before physiological measurements began.

Participants completed a resting acclimatization period before each testing session to stabilize cardiovascular parameters. Testing order was fixed across all time points to reduce procedural interactions: HRV assessment was conducted first to avoid potential carryover effects from vascular occlusion or anticipatory stress related to biological sampling, then PORH assessment, then saliva collection for cortisol, and finally subjective ratings and diary-derived outcomes.

Heart rate variability assessment

Heart rate variability (HRV) was analyzed to characterize resting modulation of the autonomic nervous system. RR intervals were recorded using a Polar H10 telemetry chest strap (Polar Electro, Finland) connected via Bluetooth to the HRV Logger mobile application (Altini, version 2.6). Recordings were performed at the same time of day for each participant, within the standardized morning testing window used for physiological assessments. Participants were instructed to avoid vigorous exercise, caffeine, nicotine, alcohol, and heavy meals before testing according to the same pre-assessment restrictions applied across physiological outcomes. After arrival, participants completed a standardized resting stabilization period of 5 minutes in a supine position before HRV acquisition. RR intervals were then recorded for 5 minutes under quiet resting conditions (Malik et al., 1996). Participants were instructed to remain still, avoid speaking, and breathe spontaneously. Paced breathing was not imposed because the aim was to assess resting autonomic regulation under standardized but ecologically conditions.

Resting autonomic modulation was evaluated using heart rate variability analysis based on RR interval recordings. RR interval data were exported as.txt files and processed in Kubios HRV Scientific software (version 4.2.0, University of Eastern Finland, Kuopio, Finland). Recordings were visually inspected and processed using the same artifact-correction settings across all time points. Recordings were excluded or repeated when there was signal loss, non-sinus rhythm, ectopic-beat clustering, or excessive artifact correction exceeding 5% of beats. Analyses were restricted to time-domain metrics selected for robustness and practical interpretability in applied settings, specifically mean heart rate (bpm), mean RR interval (ms), RMSSD (ms), lnRMSSD, and SDNN (ms). HRV was obtained under standardized resting conditions and repeated at T0, T1, and T2, with consistent procedures across time points to support longitudinal comparability.

Microvascular reactivity assessment (post-occlusive reactive hyperemia)

Microvascular function was assessed using post-occlusive reactive hyperemia (PORH) under standardized resting conditions. Participants rested ≥ 10 min in a semi-recumbent position with standardized limb positioning to stabilize systemic hemodynamics and minimize prior-activity effects. Cutaneous perfusion was recorded continuously using laser Doppler flowmetry (LDF; Perimed, Perimed AB, Sweden) and expressed in perfusion units

(PU); the LDF system was calibrated per manufacturer before data collection. The LDF probe was positioned on the pulp of the great toe of the examined lower limb, avoiding visible veins, calluses, wounds, bruising, or skin irritation. The toe and foot were immobilized with adhesive tape affixed to a stable support to minimize motion artefacts. Probe placement was standardized using the same anatomical site at each visit and was verified before each recording. Measurements were performed bilaterally on the right and left lower limbs, with randomized limb order and a 15-min interval between limbs to allow perfusion to return to baseline and minimize carryover from the preceding ischemic stimulus. Right- and left-limb PORH values were averaged at each time point to generate one participant-level value per PORH variable for the primary analysis.

Before PORH, peripheral arterial suitability was verified by assessing arterial flow and ankle-brachial index (ABI). Doppler ultrasound (SonoScape system, linear probe 3–18 MHz) was used to evaluate flow at a standardized anatomical site with consistent insonation angle and ultrasound gel. ABI and Doppler findings were used as control measures to confirm adequate perfusion prior to proceeding with occlusion. Skin temperature was not directly monitored. Environmental standardization, acclimatization, and repeated same-site testing were used to reduce temperature-related variability.

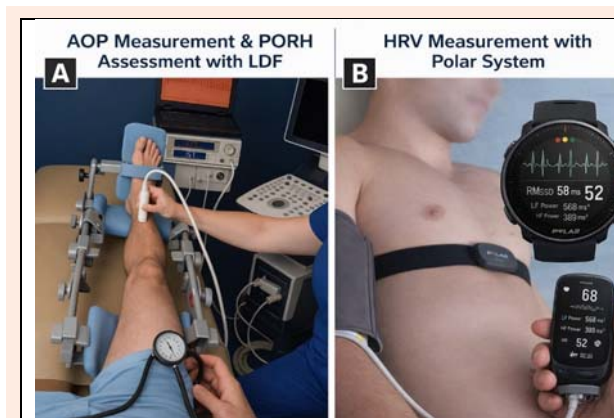


Figure 1. Assessment of vascular and autonomic function using non-invasive measurement techniques. Ankle occlusion pressure (AOP) and post-occlusive reactive hyperemia (PORH) were evaluated with laser Doppler flowmetry (LDF) to assess peripheral microcirculatory function. Heart rate variability (HRV) was measured using the Polar system with a chest-strap heart rate sensor, providing an assessment of autonomic nervous system activity.

For PORH (Figure 1), a pneumatic cuff was applied to the proximal upper arm (proximal edge contacting the inferior border of the axilla) using a 10-cm-wide adult cuff (clinically approved devices). After obtaining a stable baseline LDF trace for 2–3 min, cuff pressure was increased from 0 mmHg in 10-mmHg steps, holding each level for 30 s to stabilize the LDF signal, until maximal arterial occlusion pressure (AOPmax) was reached (defined as complete cessation of arterial inflow). AOPmax was individualized and recorded separately for each limb. The mean AOPmax used during PORH testing was 177.3 ± 15.9 mmHg across participants and visits. Occlusion was then maintained for 5 min, followed by rapid cuff deflation to initiate reperfusion; the LDF recording continued

through the hyperemic response and recovery. Outcomes extracted from the perfusion trace were: resting flow (RF) (baseline), biological zero (BZ) (signal during complete occlusion), peak hyperemia (RHmax/Peak), time to peak (TTP) (from release to peak), and recovery time (TR) (time to return to baseline). Parameters were computed using automated analysis with consistent smoothing/peak-detection settings applied across all sessions. Values of variability of measures were 9.1% in RF, 8.2% in RHmax, 7.4% in TTP, and 9.9% in TR.

Salivary cortisol assessment

Salivary cortisol was assessed as an index of systemic stress/physiological load at three time points: T0 (pre-intervention), T1 (immediately post-intervention), and T2 (post-2-week washout). To reduce diurnal and day-to-day variability, saliva was collected on two consecutive mornings (Monday and Tuesday) at each time point within the same fixed morning time window, and the mean of the two values was used for analysis. Samples were collected between 07:00 and 09:00 and at approximately 30 min after habitual waking. The same clock-time window and wake-to-sampling interval were maintained as closely as possible for each participant across T0, T1, and T2. Sampling was scheduled to be consistent relative to participants' habitual wake time (i.e., collected at a similar clock time and as comparable as feasible across time points) to limit confounding from circadian cortisol dynamics.

Saliva collection was performed at Provita Medical Center during the standardized morning assessment visits. Participants were instructed to standardize pre-sampling conditions by refraining from food, caffeine, smoking/nicotine, and vigorous physical activity for ≥ 60 min before collection, and to avoid behaviors that can contaminate saliva or acutely alter readings (e.g., tooth brushing and mouthwash immediately before sampling). Immediately prior to sampling, participants rinsed their mouth with water and waited a short, consistent period before providing saliva. Samples were collected using a consistent method across all sessions (Salivette-type swab), then stored under standardized conditions (refrigerated promptly and frozen at $-20^{\circ}\text{C}/-80^{\circ}\text{C}$ until analysis) to preserve hormone stability and reduce pre-analytical variability.

Cortisol concentrations were quantified using a competitive ELISA with the Cortisol Saliva ELISA kit (IBL International GmbH, Hamburg, Germany; catalog no. RE52611), strictly following the manufacturer's protocol. The assay limit of quantitation sensitivity was 0.138 nmol/L ($0.005 \mu\text{g/dL}$), with intra-assay and inter-assay coefficients of variation of 4.3% and 13.2%, respectively, according to manufacturer specifications. All samples were analyzed in duplicate, and all time-point samples from the same participant were run on the same plate/assay batch to minimize inter-assay variation. Results were reported in the kit-specified units, with consistent handling of values below the limit of detection (per manufacturer guidance) and identical processing rules applied across all participants and time points.

Subjective outcomes

Perceived recovery was assessed using the Total Quality Recovery (TQR) scale at each time point (Fessi et al.,

2016). This self-reported measure utilizes a 20-point scale, ranging from 6 (indicating incomplete recovery) to 20 (representing complete recovery). At T0, T1, and T2, athletes completed the TQR rating during the assessment visit after the standardized physiological testing sequence. The rating was anchored to the athlete's perceived recovery state that morning and recent training recovery, not to immediate recovery from the laboratory testing procedures. Athletes were instructed to answer the question as an overall current recovery appraisal, considering the preceding training exposure and their perceived readiness at the time of assessment. The TQR scale is intended to capture the athlete's perceived recovery state (Kenttä and Hassmén, 1998). Participants received standardized instructions before the study and completed ratings independently to support consistency and reduce social influence.

Statistical procedures

All analyses were designed to quantify differential longitudinal change between groups across baseline (T0), post-intervention (T1), and wash-out (T2), while emphasizing effect magnitude and precision. The primary analysis was per-protocol, including participants with $\geq 75\%$ compliance, with an intention-to-treat sensitivity analysis retaining all randomized participants and all available observations. Outcomes are summarized as mean $\pm$ SD by group and time point. For each endpoint, inference used linear mixed-effects models with fixed effects for Group, Time (categorical), and Group $\times$ Time, and a participant-level random intercept to account for within-subject dependence; sex and club were included as prespecified covariates where estimable, and training load/fatigue variables were evaluated in prespecified adjusted sensitivity models (e.g., intervention-period averages) to test robustness to concurrent training exposure. Model assumptions were checked using residual diagnostics, with log-transformation applied when needed for skewed outcomes (notably cortisol and selected PORH variables) and results reported consistently

on the interpretable scale.

Between-group effects were quantified as differences in change ($\Delta\Delta$) for T1-T0 and T2-T0 derived from the Group $\times$ Time interaction, reported with 95% CIs and two-sided p-values ($p < 0.001$ when applicable). Within-group changes were estimated from the same models using estimated marginal means and pairwise contrasts within each group (T1 vs T0, T2 vs T0, T2 vs T1), with multiplicity control for the three time comparisons per group. Practical relevance was addressed using standardized effects for pretest-posttest control designs (Hedges' g for the between-group difference in change, standardized by pooled baseline SD) with 95% CIs obtained by participant-level bootstrap; within-group standardized mean changes were computed analogously for context. Figures display group means across time with SD error bars in black-and-white to facilitate direct visual comparison. Statistical analysis was conducted in R environment (R 4.5.2, R Core Team).

Results

Participant flow, adherence, and outcome availability

Sixty athletes were randomized after baseline testing, with 30 allocated to contrast heat-cold showering and 30 allocated to thermoneutral showering. All randomized participants received the allocated condition and were retained for intention-to-treat sensitivity analyses. The primary per-protocol analysis included 57 participants who achieved at least 75% compliance with the assigned shower protocol: 28 in the contrast heat-cold group and 29 in the control group. Three randomized participants were excluded from the per-protocol analysis for compliance below the pre-specified threshold (contrast $n = 2$; control $n = 1$). Participant flow, per-protocol exclusions, and outcome-specific analytic samples are summarized in Figure 2. Baseline participant characteristics for the per-protocol sample are presented in Table 1. TQR, HRV, PORH and morning salivary cortisol were available for all 57 per-protocol participants.

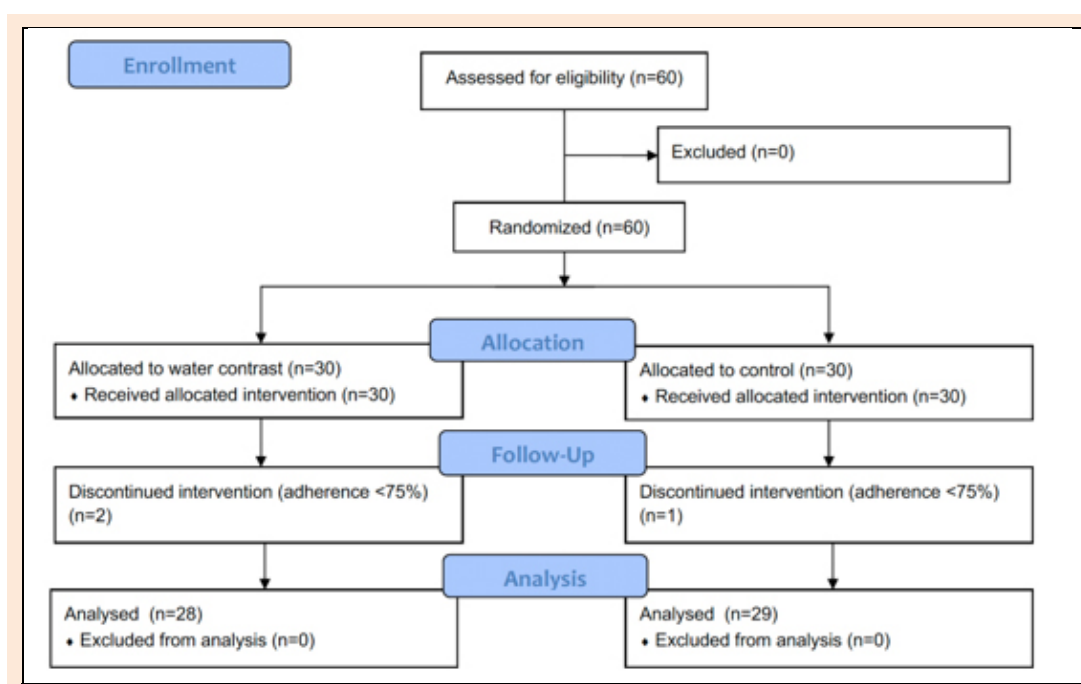


Figure 2. Participants flow diagram.

Table 1. Baseline characteristics.

Variable	Thermoneutral shower control			Contrast heat-cold		
	Total (n = 29)	Men (n = 23)	Women (n = 6)	Total (n = 28)	Men (n = 24)	Women (n = 4)
Participants, n	29	23	6	28	24	4
Men, n (%)	23 (79.3)	23 (100.0)	0 (0.0)	24 (85.7)	24 (100.0)	0 (0.0)
Women, n (%)	6 (20.7)	0 (0.0)	6 (100.0)	4 (14.3)	0 (0.0)	4 (100.0)
Age (years)	29.8 ± 4.8 (19.0 - 38.0)	29.4 ± 5.1 (19.0 - 38.0)	31.0 ± 3.2 (26.0 - 34.0)	27.3 ± 6.5 (18.0 - 37.0)	27.1 ± 6.5 (18.0 - 37.0)	28.2 ± 7.3 (18.0 - 34.0)
Body height (cm)	179.1 ± 8.1	182.4 ± 4.7	166.5 ± 5.4	180.6 ± 8.7	183.0 ± 6.1	166.1 ± 7.6
Body mass (kg)	79.6 ± 8.0	79.1 ± 8.6	81.5 ± 5.1	78.0 ± 7.3	78.7 ± 7.6	74.0 ± 3.8
BMI (kg/m ²)	25.0 ± 3.5	23.8 ± 2.7	29.4 ± 2.5	24.1 ± 3.2	23.6 ± 2.9	27.0 ± 3.2
Training experience (years)	7.4 ± 3.3	7.2 ± 3.5	8.5 ± 2.3	7.7 ± 3.0	7.5 ± 2.7	8.8 ± 4.5
Training frequency (sessions/week)	4.1 ± 0.3	4.1 ± 0.3	4.2 ± 0.3	4.1 ± 0.3	4.1 ± 0.3	4.1 ± 0.1
Weight class						
Lightweight, n (%)	6 (20.7)	5 (21.7)	1 (16.7)	6 (21.4)	5 (20.8)	1 (25.0)
Middleweight, n (%)	17 (58.6)	14 (60.9)	3 (50.0)	18 (64.3)	15 (62.5)	3 (75.0)
Heavyweight, n (%)	6 (20.7)	4 (17.4)	2 (33.3)	4 (14.3)	4 (16.7)	0 (0.0)
Baseline recovery and physiological outcomes						
TQR (a.u.)	13.1 ± 0.8	13.1 ± 0.8	13.2 ± 0.8	13.4 ± 0.7	13.4 ± 0.7	13.2 ± 1.0
Morning salivary cortisol (nmol/L)	16.0 ± 1.4	16.4 ± 1.2	14.6 ± 1.0	16.0 ± 1.8	16.5 ± 1.3	13.0 ± 0.9
Resting HR (bpm)	61.4 ± 3.2	61.0 ± 3.2	62.7 ± 2.8	60.2 ± 3.2	60.1 ± 3.3	60.6 ± 2.8
lnRMSSD (log units)	4.13 ± 0.15	4.13 ± 0.16	4.14 ± 0.15	4.10 ± 0.17	4.12 ± 0.18	4.01 ± 0.13
RMSSD (ms)	62.9 ± 9.8	62.8 ± 10.2	63.2 ± 9.1	61.5 ± 11.0	62.5 ± 11.4	55.5 ± 6.8
SDNN (ms)	88.1 ± 13.3	87.7 ± 12.9	89.8 ± 15.8	86.7 ± 19.0	89.1 ± 19.4	73.2 ± 10.1
PORH resting flow (PU)	24.1 ± 1.9	24.3 ± 1.9	23.5 ± 1.7	24.1 ± 1.9	24.3 ± 2.0	22.8 ± 1.0
PORH peak (PU)	35.2 ± 3.7	34.5 ± 3.5	38.3 ± 2.7	35.3 ± 4.5	35.3 ± 4.5	34.7 ± 5.4
PORH time to peak (s)	22.9 ± 4.6	22.8 ± 4.1	23.3 ± 6.6	25.0 ± 4.9	25.4 ± 4.8	21.9 ± 5.3
PORH recovery time (min)	6.26 ± 0.87	6.32 ± 0.94	6.02 ± 0.54	6.14 ± 0.93	6.16 ± 0.97	6.00 ± 0.74

Values are mean ± SD. Percentages are calculated within each column. Recorded training frequency reflects the mean number of recorded training/shower sessions per week during the 4-week intervention period. BMI: body mass index; TQR: Total Quality Recovery; HR: heart rate; lnRMSSD: natural logarithm of the root mean square of successive differences; RMSSD: root mean square of successive differences; SDNN: standard deviation of NN intervals; PORH: post-occlusive reactive hyperemia; PU: perfusion units.

Training-load, adherence, and wellness monitoring during the intervention

Training-load and wellness monitoring data are reported in Table 2. Across the four-week intervention, recorded weekly training frequency was similar between groups (contrast 4.05 ± 0.30 sessions/week; control 4.13 ± 0.31 sessions/week; difference -0.08 sessions/week, 95% CI -0.24 to 0.09 ; $p = 0.353$). Mean session-RPE was also similar between groups (contrast 6.28 ± 0.45 ; control 6.22 ± 0.47 ; difference 0.06 , 95% CI -0.19 to 0.30 ; $p = 0.643$). Consequently, estimated weekly sRPE training load did not show a between-group imbalance (contrast 2289 ± 214 AU/week; control 2314 ± 264 AU/week; difference -26 AU/week, 95% CI -153 to 102 ; $p = 0.686$).

Fatigue scores were comparable between groups (contrast 5.51 ± 0.39 ; control 5.53 ± 0.46 ; difference -0.02 , 95% CI -0.24 to 0.21 ; $p = 0.882$), and wellness scores showed no clear between-group imbalance (contrast 6.26 ± 0.48 ; control 6.13 ± 0.47 ; difference 0.13 , 95% CI -0.12 to

0.38 ; $p = 0.308$). Completed assigned shower sessions were similar between groups across the 4-week intervention (contrast heat-cold shower group: 16.2 ± 1.2 sessions; thermoneutral shower control group: 16.5 ± 1.2 sessions). Mean shower-protocol adherence was also similar between groups (contrast heat-cold shower group: $81.07 \pm 5.99\%$; thermoneutral shower control group: $82.76 \pm 6.06\%$; difference -1.69% , 95% CI -4.89 to 1.51 ; $p = 0.295$).

Between-group effects for autonomic, endocrine, perceptual, and microvascular outcomes

Descriptive statistics across T0, T1, and T2 and the primary between-group differences in change are reported in Tables 3 and 4, with corresponding time-course plots shown in Figure 3, Figure 4, and Figure 5. The primary interpretation is based on the between-group difference in change ($\Delta\Delta$; contrast minus control) from T0 to T1 and from T0 to T2, rather than within-group change alone.

Table 2. Weekly training-load, fatigue, wellness, and adherence monitoring during the four-week intervention.

Variable	Week	Contrast heat-cold	Thermoneutral shower control	Period-average difference (95% CI)	p-value
Training frequency (sessions/week)	4-week average	4.05 ± 0.30	4.13 ± 0.31	-0.08 (-0.24 to 0.09)	0.353
Session-RPE (0 - 10)	4-week average	6.28 ± 0.45	6.22 ± 0.47	0.06 (-0.19 to 0.30)	0.643
sRPE training load (AU/week)	4-week average	2289 ± 214	2314 ± 264	-26 (-153 to 102)	0.686
Wellness score (AU/week)	4-week average	6.26 ± 0.48	6.13 ± 0.47	0.13 (-0.12 to 0.38)	0.308
Shower-protocol compliance (%)	4-week average	81.07 ± 5.99	82.76 ± 6.06	-1.69 (-4.89 to 1.51)	0.295

Values are mean ± SD. Period-average differences are contrast minus control. sRPE training load was calculated as weekly training frequency × mean session-RPE × 90 minutes, reflecting the approximate duration of the primary training sessions. AU, arbitrary units; CI, confidence interval; RPE, rating of perceived exertion.

Table 3. Autonomic, endocrine, and subjective recovery outcomes.

Outcome	Thermoneutral shower control T0	Thermoneutral shower control T1	Thermoneutral shower control T2	Contrast heat-cold T0	Contrast heat-cold T1	Contrast heat-cold T2	Between-group $\Delta\Delta$ (T1-T0 / T2-T0), effect size, p-value
TQR (a.u.)	13.4 $\pm$ 0.5	15.9 $\pm$ 0.5	13.5 $\pm$ 0.5	13.1 $\pm$ 1.0	16.2 $\pm$ 0.9	13.3 $\pm$ 0.5	$\Delta\Delta$ 0.61 (95% CI 0.27 to 0.95), $p < 0.001$; g 0.80. $\Delta\Delta$ 0.12 (95% CI -0.37 to 0.62), $p = 0.622$; g 0.16.
Salivary cortisol (nmol/L)	16.0 $\pm$ 1.5	15.2 $\pm$ 1.4	16.1 $\pm$ 1.5	16.0 $\pm$ 1.7	15.9 $\pm$ 1.4	16.0 $\pm$ 1.5	RRR 1.05 (95% CI 0.99 to 1.11), $p = 0.132$; g 0.43. RRR 1.00 (95% CI 0.93 to 1.07), $p = 0.962$; g -0.02.
Resting HR (bpm)	61 $\pm$ 3	61 $\pm$ 3	61 $\pm$ 3	60 $\pm$ 3	58 $\pm$ 3	60 $\pm$ 3	$\Delta\Delta$ -2.10 (95% CI -2.24 to -1.96), $p < 0.001$; g -0.65. $\Delta\Delta$ 0.01 (95% CI -0.14 to 0.15), $p = 0.934$; g 0.00.
lnRMSSD (log units)	4.13 $\pm$ 0.15	4.13 $\pm$ 0.16	4.13 $\pm$ 0.15	4.10 $\pm$ 0.17	4.23 $\pm$ 0.17	4.10 $\pm$ 0.18	$\Delta\Delta$ 0.123 (95% CI 0.108 to 0.137), $p < 0.001$; g 0.74. $\Delta\Delta$ -0.002 (95% CI -0.016 to 0.012), $p = 0.776$; g -0.01.
RMSSD (ms)	62.9 $\pm$ 9.8	63.1 $\pm$ 10.1	62.9 $\pm$ 9.9	61.5 $\pm$ 11.0	69.7 $\pm$ 12.3	61.3 $\pm$ 10.9	$\Delta\Delta$ 7.97 (95% CI 6.91 to 9.02), $p < 0.001$; g 0.75. $\Delta\Delta$ -0.12 (95% CI -1.02 to 0.77), $p = 0.783$; g -0.01.
SDNN (ms)	88.1 $\pm$ 13.3	89.5 $\pm$ 14.0	85.4 $\pm$ 13.9	86.7 $\pm$ 19.0	98.6 $\pm$ 18.1	85.9 $\pm$ 16.2	$\Delta\Delta$ 10.48 (95% CI 6.47 to 14.49), $p < 0.001$; g 0.63. $\Delta\Delta$ 1.87 (95% CI -1.90 to 5.64), $p = 0.325$; g 0.11.

Values are mean $\pm$ SD. TQR, Total Quality Recovery; HR, heart rate; lnRMSSD, natural logarithm of the root mean square of successive differences between normal-to-normal RR intervals; RMSSD, root mean square of successive differences between normal-to-normal RR intervals; SDNN, standard deviation of normal-to-normal RR intervals; $\Delta\Delta$ denotes the between-group difference in change from baseline (contrast minus control). Effect sizes are Hedges g for pretest-posttest control designs. RRR denotes a ratio of ratios for the log-modeled cortisol outcome.

Table 4. Microvascular reactivity outcomes from post-occlusive reactive hyperemia.

Outcome	Thermoneutral shower control T0	Thermoneutral shower control T1	Thermoneutral shower control T2	Contrast heat-cold T0	Contrast heat-cold T1	Contrast heat-cold T2	Between-group $\Delta\Delta$ (T1-T0 / T2-T0), effect size, p-value
PORH Peak (PU)	35.7 $\pm$ 4.1	38.01 $\pm$ 5.38	36.4 $\pm$ 4.6	34.8 $\pm$ 4.0	37.5 $\pm$ 5.4	35.6 $\pm$ 4.4	$\Delta\Delta$ 0.36 (95% CI -1.24 to 1.96), $p = 0.651$; g 0.09. $\Delta\Delta$ 0.08 (95% CI -0.70 to 0.86), $p = 0.830$; g 0.02.
Resting flow RF (PU)	23.9 $\pm$ 1.8	24.30 $\pm$ 1.81	24.0 $\pm$ 1.9	24.3 $\pm$ 2.0	24.9 $\pm$ 2.2	24.4 $\pm$ 2.2	$\Delta\Delta$ 0.15 (95% CI -0.18 to 0.47), $p = 0.365$; g 0.08. $\Delta\Delta$ 0.03 (95% CI -0.28 to 0.35), $p = 0.833$; g 0.02.
Time to peak TTP (s)	23.5 $\pm$ 5.2	22.37 $\pm$ 4.89	23.5 $\pm$ 5.1	24.3 $\pm$ 4.4	23.1 $\pm$ 3.9	24.5 $\pm$ 4.5	$\Delta\Delta$ 0.01 (95% CI -0.74 to 0.77), $p = 0.972$; g 0.00. $\Delta\Delta$ 0.27 (95% CI -0.08 to 0.61), $p = 0.126$; g 0.05.
Recovery time (min)	6.2 $\pm$ 0.9	5.81 $\pm$ 0.84	6.1 $\pm$ 0.9	6.2 $\pm$ 0.9	5.9 $\pm$ 1.0	6.1 $\pm$ 1.0	$\Delta\Delta$ 0.02 (95% CI -0.20 to 0.23), $p = 0.873$; g 0.02. $\Delta\Delta$ -0.09 (95% CI -0.19 to 0.02), $p = 0.108$; g -0.09.

Values are mean $\pm$ SD. PORH, post-occlusive reactive hyperemia; PU, perfusion units; RF, resting flow; TTP, time to peak; $\Delta\Delta$ denotes the between-group difference in change from baseline (contrast minus control). Effect sizes are Hedges g for pretest-posttest control designs.

Autonomic outcomes showed the clearest post-intervention between-group difference. From T0 to T1, the contrast heat-cold group showed lower resting heart rate relative to control ($\Delta\Delta$ -2.10 bpm, 95% CI -2.24 to -1.96; g -0.65; $p < 0.001$) and higher HRV indices, including lnRMSSD ($\Delta\Delta$ 0.123 log units, 95% CI 0.108 to 0.137; g 0.74; $p < 0.001$), RMSSD ($\Delta\Delta$ 7.97 ms, 95% CI 6.91 to 9.02; g 0.75; $p < 0.001$), and SDNN ($\Delta\Delta$ 10.48 ms, 95% CI 6.47 to 14.49; g 0.63; $p < 0.001$). These effects were not maintained at T2 versus T0 for resting heart rate ($\Delta\Delta$ 0.01 bpm, 95% CI -0.14 to 0.15; g 0.00; $p = 0.934$), lnRMSSD ($\Delta\Delta$ -0.002 log units, 95% CI -0.016 to 0.012; g -0.01; $p = 0.776$), RMSSD ($\Delta\Delta$ -0.12 ms, 95% CI -1.02 to 0.77; g -0.01; $p = 0.783$), or SDNN ($\Delta\Delta$ 1.87 ms, 95% CI -1.90 to 5.64; g 0.11; $p = 0.325$). The confidence intervals for resting heart rate and lnRMSSD were checked against the observed change-score variability and reflected low partic-

ipant-level variability in T0-to-T1 change scores (heart-rate Δ SD: 0.32 bpm in contrast and 0.18 bpm in control; lnRMSSD Δ SD: 0.035 and 0.019 log units, respectively), whereas SDNN showed larger change-score variability (6.90 and 7.98 ms), explaining its wider confidence interval.

Subjective recovery showed a post-intervention between-group effect in the same temporal direction as the autonomic outcomes. TQR increased from 13.4 $\pm$ 0.5 to 15.9 $\pm$ 0.5 in the control group and from 13.1 $\pm$ 1.0 to 16.2 $\pm$ 0.9 in the contrast heat-cold group, corresponding to a T0-to-T1 $\Delta\Delta$ of 0.61 TQR points (95% CI 0.27 to 0.95; g 0.80; $p < 0.001$). The T2-to-T0 between-group contrast was smaller and not statistically clear ($\Delta\Delta$ 0.12 TQR points, 95% CI -0.37 to 0.62; g 0.16; $p = 0.622$), indicating that the post-intervention perceptual effect was not retained after wash-out.

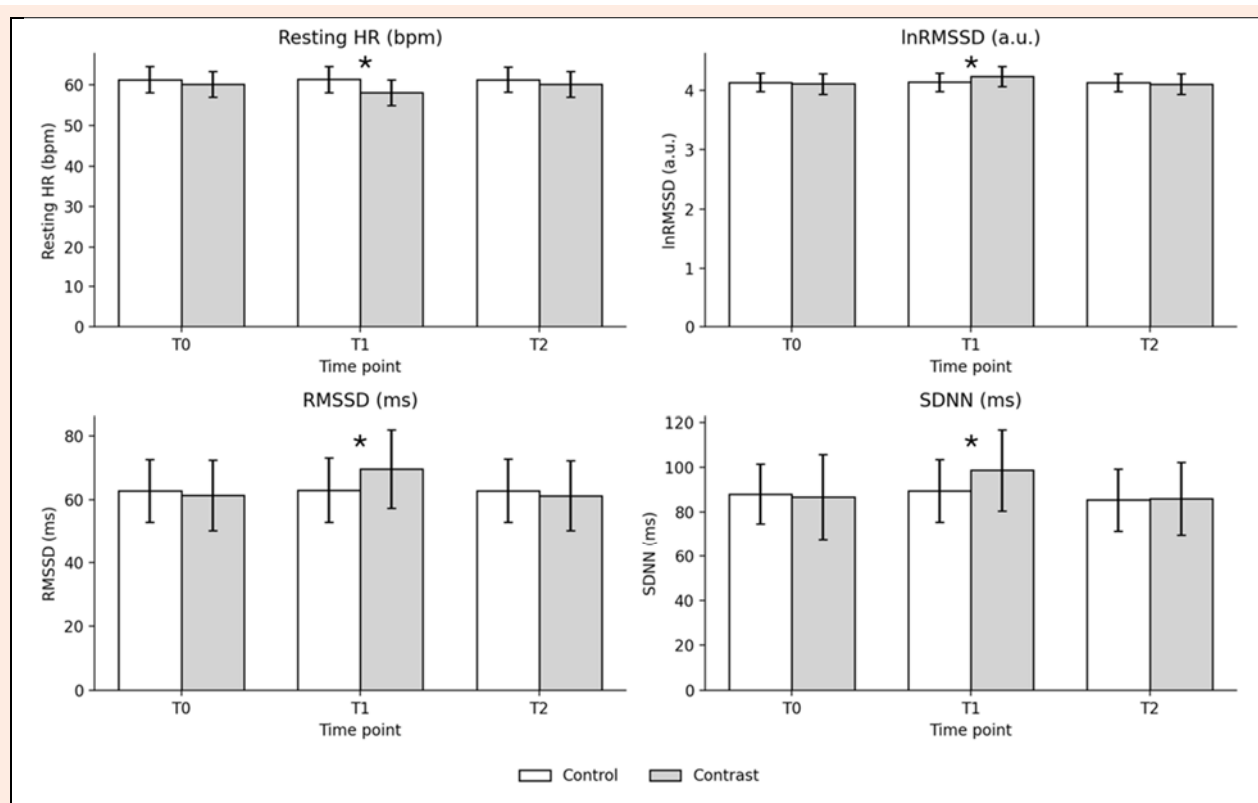


Figure 3. Resting HRV outcomes across time points. Bars show mean values and error bars show SD. Control and contrast heat–cold groups are displayed side-by-side at each time point (T0 baseline, T1 post-intervention, T2 post wash-out). lnRMSSD, natural logarithm of the root mean square of successive differences; RMSSD, root mean square of successive differences; SDNN, standard deviation of NN intervals; *: indicates a significant between-group difference in change for a given timepoint ($p < 0.05$).

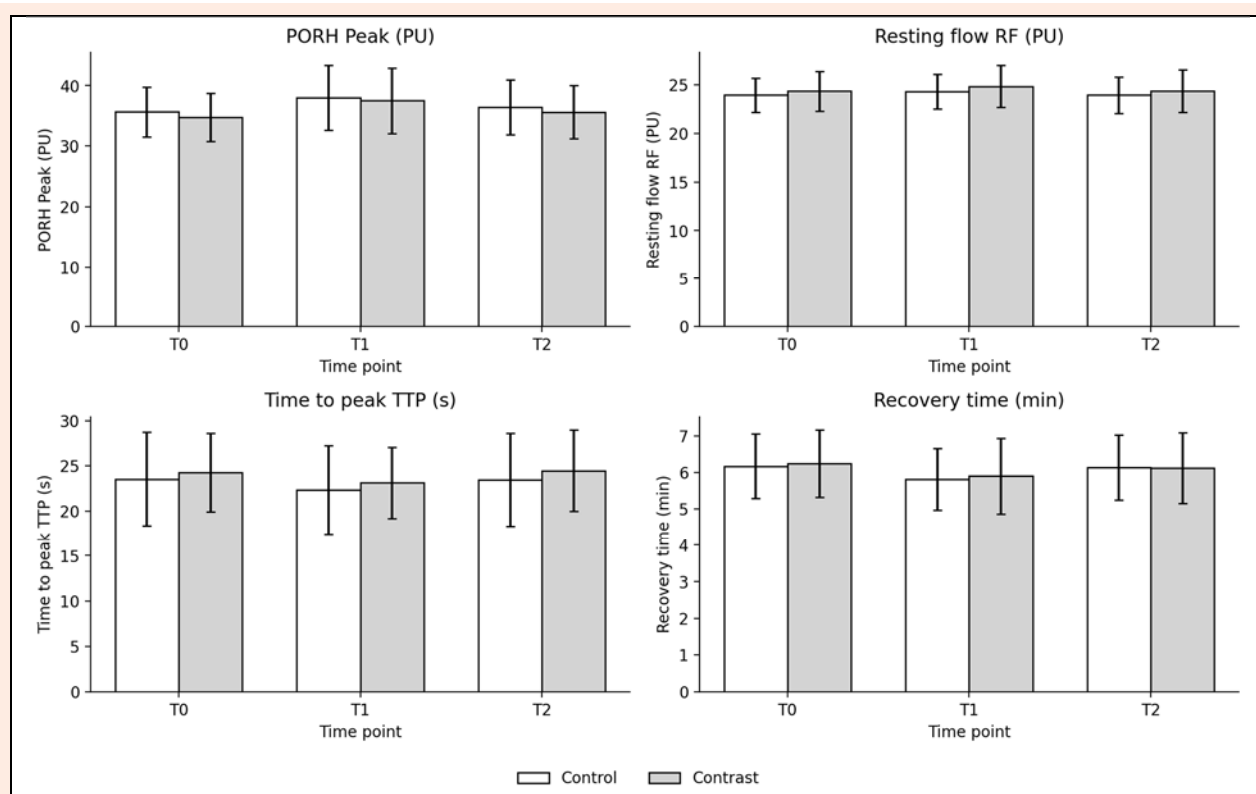


Figure 4. PORH microvascular outcomes across time points (per-protocol). Bars show mean values and error bars show SD. Control and contrast heat–cold groups are displayed side-by-side at each time point (T0 baseline, T1 post-intervention, T2 post wash-out). HRV, heart rate variability; lnRMSSD, natural logarithm of the root mean square of successive differences; RMSSD, root mean square of successive differences; SDNN, standard deviation of NN intervals; PORH, post-occlusive reactive hyperemia; TTP, time to peak; TQR, Total Quality Recovery; SD, standard deviation; CI, confidence interval; RRR, ratio of ratios.

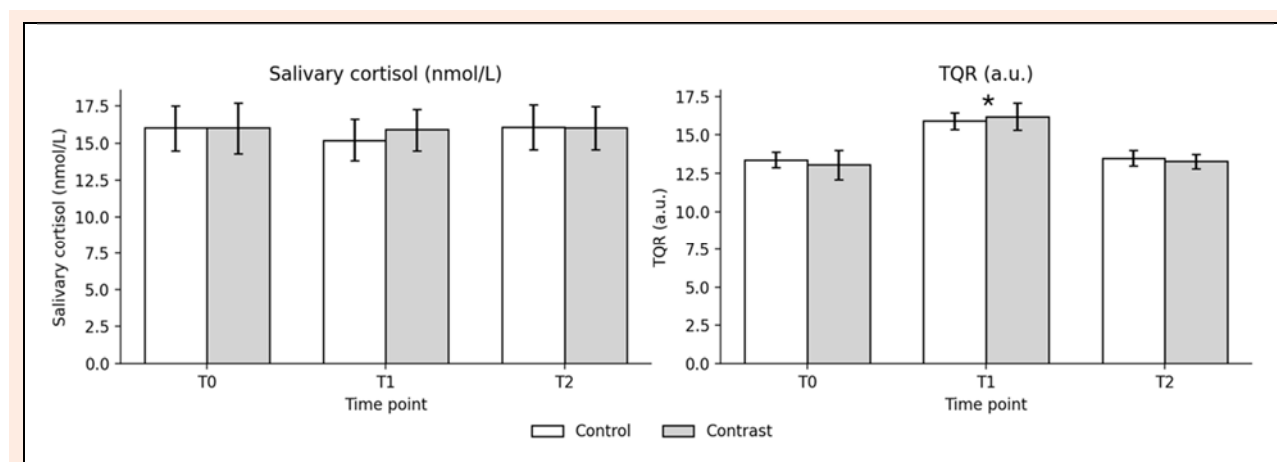


Figure 5. Salivary cortisol and perceived recovery (TQR) across time points. Bars show mean values and error bars show SD. Control and contrast heat–cold groups are displayed side-by-side at each time point (T0 baseline, T1 post-intervention, T2 post wash-out). *: indicates a significant between-group difference in change for a given timepoint ($p < 0.05$).

Morning salivary cortisol did not show a statistically clear intervention-specific change. The T0-to-T1 ratio of ratios was 1.05 (95% CI 0.99 to 1.11; g 0.43; $p = 0.132$), and the T2-to-T0 ratio of ratios was 1.00 (95% CI 0.93 to 1.07; g -0.02; $p = 0.962$).

PORH-derived microvascular outcomes are summarized in Table 4 and Figure 4. They are interpreted using point estimates, confidence intervals, and effect sizes rather than a formal equivalence or non-inferiority framework. From T0 to T1, the between-group $\Delta\Delta$ estimates were 0.36 PU for PORH Peak (95% CI -1.24 to 1.96; g 0.09; $p = 0.651$), 0.15 PU for resting flow RF (95% CI -0.18 to 0.47; g 0.08; $p = 0.365$), 0.01 s for time to peak TTP (95% CI -0.74 to 0.77; g 0.00; $p = 0.972$), and 0.02 min for recovery time (95% CI -0.20 to 0.23; g 0.02; $p = 0.873$). From T0 to T2, the corresponding $\Delta\Delta$ estimates were 0.08 PU for PORH Peak (95% CI -0.70 to 0.86; g 0.02; $p = 0.830$), 0.03 PU for resting flow RF (95% CI -0.28 to 0.35; g 0.02; $p = 0.833$), 0.27 s for time to peak TTP (95% CI -0.08 to 0.61; g 0.05; $p = 0.126$), and -0.09 min for recovery time (95% CI -0.19 to 0.02; g -0.09; $p = 0.108$).

Intention-to-treat sensitivity estimates are provided for every endpoint and contrast in Table 5. From T0 to T1, ITT analyses showed post-intervention between-group effects for resting HR, HRV indices, and TQR, whereas cortisol and PORH-derived outcomes remained statistically unclear. From T0 to T2, the ITT estimates did not indicate persistence of the autonomic or TQR effects after wash-out.

Discussion

In this randomized field trial in trained combat sport athletes, a four-week contrast heat-cold shower protocol showed short-term relative improvements compared with thermoneutral showering in resting autonomic indices (lower resting heart rate and higher vagally mediated HRV) and perceived recovery. These relative improvements were not maintained after the two-week wash-out, indicating predominantly transient rather than persistent group-level responses. In contrast, morning salivary cortisol did not

show a consistent intervention-specific signal, and microvascular reactivity assessed via PORH improved over time in both groups without detectable between-group separation, suggesting that several observed changes likely reflected time- or training-related influences rather than a unique physiological effect of contrast showers.

Autonomic outcomes showed the most coherent intervention pattern, with the contrast group demonstrating a post-intervention shift consistent with greater parasympathetic modulation that was not retained after wash-out (Laborde et al., 2024). This interpretation should remain cautious because the present study assessed resting HRV at scheduled testing time points and did not measure acute parasympathetic reactivation, baroreflex sensitivity, catecholamines, skin temperature, core temperature, or other mediator variables. Therefore, mechanisms such as reflex parasympathetic reactivation or thermally induced autonomic shifts remain plausible explanations rather than processes directly demonstrated in this trial (Al Haddad et al., 2010). Some evidence indicates that post-exercise recovery techniques, and cold-water immersion in particular, can yield small-to-moderate increases in vagally mediated HRV (typically RMSSD), supporting the physiological plausibility of the short-term HRV improvements observed in our contrast protocol (Laborde et al., 2024). Previous report in in combat sport contexts also support sensitivity of HRV to training stress and recovery status, indicating that autonomic measures are responsive in athlete groups with training patterns similar to those studied here (Morales et al., 2014). However, because the present protocol used contrast showering rather than immersion and because no acute autonomic mediator measurements were collected, the observed HRV changes should be interpreted as resting autonomic recovery indicators, not as proof of a specific autonomic pathway (Sheng and Zhu, 2018). The lack of persistence at wash-out is also consistent with the concept that many recovery modalities primarily modulate acute homeostatic state rather than inducing durable training-like adaptations, especially when the stimulus is brief and administered post-exercise rather than as a standalone conditioning exposure (Higgins et al., 2017).

Table 5. Intention-to-treat sensitivity estimates for all endpoints and primary contrasts.

Measure	Contrast	ITT estimate	95% CI	Hedges g	p
TQR (a.u.)	T1-T0	$\Delta\Delta$ 0.60 TQR points	0.28 to 0.93	0.78	< 0.001
TQR (a.u.)	T2-T0	$\Delta\Delta$ 0.11 TQR points	-0.38 to 0.59	0.14	0.662
Cortisol (nmol/L)	T1-T0	RRR 1.04	0.98 to 1.10	0.36	0.199
Cortisol (nmol/L)	T2-T0	RRR 0.99	0.93 to 1.06	-0.09	0.788
Resting HR (bpm)	T1-T0	$\Delta\Delta$ -2.10 bpm	-2.24 to -1.96	-0.64	< 0.001
Resting HR (bpm)	T2-T0	$\Delta\Delta$ -0.00 bpm	-0.14 to 0.14	-0.00	0.961
lnRMSSD (log units)	T1-T0	$\Delta\Delta$ 0.122 log units	0.108 to 0.137	0.75	< 0.001
lnRMSSD (log units)	T2-T0	$\Delta\Delta$ -0.002 log units	-0.016 to 0.011	-0.01	0.722
RMSSD (ms)	T1-T0	$\Delta\Delta$ 8.02 ms	6.99 to 9.04	0.77	< 0.001
RMSSD (ms)	T2-T0	$\Delta\Delta$ -0.15 ms	-1.00 to 0.70	-0.01	0.722
SDNN (ms)	T1-T0	$\Delta\Delta$ 10.47 ms	6.52 to 14.42	0.64	< 0.001
SDNN (ms)	T2-T0	$\Delta\Delta$ 0.84 ms	-3.00 to 4.69	0.05	0.662
PORH Peak (PU)	T1-T0	$\Delta\Delta$ 0.55 PU	-0.99 to 2.10	0.13	0.477
PORH Peak (PU)	T2-T0	$\Delta\Delta$ 0.14 PU	-0.59 to 0.87	0.03	0.703
Resting flow RF (PU)	T1-T0	$\Delta\Delta$ 0.18 PU	-0.13 to 0.49	0.09	0.243
Resting flow RF (PU)	T2-T0	$\Delta\Delta$ 0.03 PU	-0.29 to 0.34	0.01	0.851
Time to peak TTP (s)	T1-T0	$\Delta\Delta$ -0.04 s	-0.76 to 0.69	-0.01	0.918
Time to peak TTP (s)	T2-T0	$\Delta\Delta$ 0.22 s	-0.11 to 0.55	0.05	0.180
Recovery time (min)	T1-T0	$\Delta\Delta$ 0.03 min	-0.18 to 0.24	0.03	0.759
Recovery time (min)	T2-T0	$\Delta\Delta$ -0.09 min	-0.19 to 0.01	-0.10	0.070

Estimates are $\Delta\Delta$ (contrast minus control), except cortisol, which is reported as a ratio of ratios (RRR). CI, confidence interval; HR, heart rate; HRV, heart rate variability; PORH, post-occlusive reactive hyperemia; TQR, Total Quality Recovery.

Subjective recovery (TQR) improved after the intervention period, with a clearer post-intervention advantage for the contrast group, but the effect did not persist following wash-out, mirroring the time course observed for autonomic outcomes (Kentt  and Hassm n, 1998). TQR and HRV should be interpreted as complementary rather than interchangeable recovery indicators since HRV reflects resting cardiac autonomic regulation, whereas TQR captures the athlete's perceived recovery state, which may integrate soreness, affective state, sleep, expectancy, and perceived readiness. The parallel short-term pattern suggests convergence between physiological and perceptual recovery indicators, but it does not establish that both outcomes measured the same underlying construct or that one mediated the other. This aligns with systematic reviews indicating that contrast water therapy can reduce soreness and improve perceived recovery in some settings, although estimates are heterogeneous and methodological quality varies across trials (Bieuz n et al., 2013). Combat sport-specific evidence also suggests that cold-water recovery strategies can improve perceived wellness after simulated MMA competition, supporting the notion that perceptual endpoints may be particularly responsive in high-impact, high-stress modalities (Tabben et al., 2018). Importantly, placebo-controlled work indicates that a meaningful portion of the perceived recovery benefit attributed to hydrotherapies can be explained by expectancy and contextual factors, which is highly relevant for interpreting TQR changes in real-world, non-blinded interventions such as ours (Broatch et al., 2014). Perceived recovery may improve through multiple converging pathways including reduced pain perception, altered affective state, and shifts in

autonomic arousal, and hydrotherapy is known to produce systemic physiological effects dependent on water temperature and exposure characteristics (An et al., 2019). Therefore, the practical value of contrast showers may lie chiefly in short-term readiness and perceived restoration rather than durable physiological remodeling, particularly when implemented as a brief post-training routine (Higgins et al., 2017).

Endocrine responses, assessed via averaged morning salivary cortisol, did not display a clear intervention-specific pattern, and within-group fluctuations were modest and bidirectional across time points (Cevada et al., 2014). This outcome is compatible with sport monitoring literature showing that salivary cortisol is sensitive to training load, sleep, and psychosocial stressors, but also exhibits substantial intra-individual variability, which can mask small intervention effects in applied designs (Sinnott-O'Connor et al., 2018). Studies in combat sport demonstrate that acute training and competition preparation can meaningfully perturb physiological stress biomarkers, including endocrine markers, highlighting that concurrent training dynamics may dominate the cortisol signal when recovery interventions are relatively brief (Lindsay et al., 2017). Evidence on hydrotherapy-related cortisol changes is mixed across populations and contexts, which further supports cautious interpretation when null or small effects are observed in athlete field trials (Mooventhan and Nivethitha, 2014). Cold exposure can be perceived as a stressor capable of activating catecholaminergic and neuroendocrine pathways, but repeated exposure may also lead to habituation, and morning cortisol, being strongly governed by circadian regulation, may be relatively

insensitive to modest post-exercise thermal manipulations unless exposure is substantial or timing is targeted (Cevada et al., 2014). From a recovery-monitoring perspective, these results suggest that cortisol may be more informative for detecting broader training-phase stress or maladaptation than for capturing the incremental effects of brief contrast shower routines (Sinnott-O'Connor et al., 2018).

Microvascular reactivity assessed with PORH showed improvements over time in both groups, but without between-group separation, indicating that the contrast protocol did not produce a detectable incremental effect on the PORH-derived endpoints used here (Lenasi and Štruel, 2010). A prior study indicates that regular physical activity and training status can alter reactive hyperemic responses, supporting the plausibility that time- and training-related factors contributed to the within-group changes observed across the study timeline (Lenasi and Štruel, 2010). A previous study also emphasize that PORH indices derived from laser Doppler are reproducible but are also sensitive to site conditions, ambient temperature, and psychophysiological state, which may reduce the detectability of small between-group effects in longitudinal athlete field contexts (Roustit et al., 2010a). Although contrast bathing is often proposed to create a vascular pumping effect through alternating vasoconstriction and vasodilation, experimental work suggests that the magnitude and localization of hemodynamic changes depend strongly on protocol parameters and the tissue compartment assessed (Shadgan et al., 2018). The present data specifically derive from laser Doppler assessment of cutaneous PORH and therefore should not be interpreted as direct evidence regarding skeletal-muscle perfusion, intramuscular oxygenation, brachial artery endothelial function by flow-mediated dilation, or sport-specific tissue oxygenation in combat-relevant muscles. Thus, the absence of a clear between-group PORH signal indicates only that this brief contrast-shower protocol did not produce a detectable incremental effect on the cutaneous microvascular endpoints measured here. It does not exclude possible effects in other vascular compartments, with different thermal doses, or with alternative measurement approaches.

Several limitations should be considered when interpreting these results and planning future work. The sample included a small number of women, which limits sex-specific interpretation. Sex was reported descriptively but was not included as a covariate or effect modifier because the small female subsample would have produced sparse group-by-sex cells and unstable estimates. Therefore, the findings should be interpreted primarily as group-level effects in the combined combat-sport sample rather than as evidence of equivalent responses across sexes. Menstrual-cycle phase and hormonal contraceptive status were not standardized, which further limits interpretation of HRV, cortisol, and vascular responses in female athletes. Future studies should recruit sufficient numbers of women to permit sex-specific analyses and should record or standardize menstrual-cycle phase and contraceptive status where feasible.

Training continued in real-world conditions, and while this enhances ecological validity, variability in train-

ing load, life stress, sleep, and unmeasured recovery behaviors may have influenced endocrine, perceptual, autonomic, and microvascular endpoints (Sinnott-O'Connor et al., 2018). Participants could not be blinded to the thermal characteristics of the shower protocol, and no sham or expectancy-control condition was included. Therefore, perceptual outcomes such as TQR may partly reflect expectancy, preference, or contextual effects rather than physiological recovery alone. Although assessors were blinded, shower timing and temperature switching were self-monitored by participants after standardized instruction, which may have introduced implementation variability despite the predefined temperature ranges and timing protocol. The thermoneutral-shower group was an active comparator rather than a no-treatment control. Consequently, the findings estimate the relative effect of contrast heat-cold showering compared with equivalent-duration thermoneutral showering, not the absolute effect of contrast showering versus no recovery routine. Finally, no direct performance outcomes, sport-specific readiness tests, muscle oxygenation measures, catecholamines, baroreflex indices, or vascular endothelial assessments such as flow-mediated dilation were collected, limiting interpretation to the autonomic, perceptual, endocrine, and cutaneous PORH outcomes measured. The wash-out period was two weeks, which is appropriate for testing short-term persistence but may be insufficient to capture slower vascular remodeling processes, especially if the hydrotherapy stimulus is low-dose compared with protocols designed to induce cardiovascular adaptations. Future research may incorporate standardization or more specific modeling of training load, include expectancy controls or sham procedures where possible, evaluate dose-response effects of temperature contrast, duration, and frequency, and consider performance outcomes and other vascular assessments to clarify whether any effects exist beyond transient autonomic and perceptual indicators.

From a practical perspective, these findings should be interpreted as evidence of short-term relative changes in resting autonomic and perceptual recovery indicators, not as proof of durable recovery adaptation or performance enhancement. Because the broader evidence base shows heterogeneity and meaningful placebo contributions for water-based recovery methods, implementation should prioritize individual preference, tolerability, and consistency while avoiding overinterpretation of benefits as guaranteed physiological adaptations. In combat-sport environments with dense technical, sparring, and conditioning schedules, contrast heat-cold showering may be considered only as a short-term recovery-support strategy when the practical objective is perceived restoration or resting autonomic recovery. However, because this study did not measure sport performance, neuromuscular adaptation, or competition outcomes, no conclusion can be made about performance transfer. Practitioners should therefore integrate contrast showering within a broader recovery framework that includes sleep, nutrition, training-load management, and individualized monitoring rather than treating it as a standalone recovery solution.

Conclusion

In the current sample of trained combat sport athletes, contrast heat-cold showering showed short-term relative improvements versus thermoneutral showering in resting autonomic indices and perceived recovery after four weeks. These effects were not maintained after the two-week wash-out. No clear intervention-specific effects were observed for morning salivary cortisol or PORH-derived cutaneous microvascular outcomes. Because participants were not blinded, training continued under real-world conditions, the comparator was an active thermoneutral shower, and no performance outcomes were measured, the findings should be interpreted as transient autonomic and perceptual responses rather than definitive evidence of durable multisystem recovery adaptation or performance benefit.

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The authors report no actual or potential conflicts of interest. While the datasets generated and analyzed in this study are not publicly available, they can be obtained from the corresponding author upon request. All experimental procedures were conducted in compliance with the relevant legal and ethical standards of the country where the study was carried out. The authors declare that no Generative AI or AI-assisted technologies were used in the writing of this manuscript.

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Key points

- Four weeks of post-training contrast heat-cold showering improved resting autonomic regulation in trained combat sport athletes, reducing resting heart rate and increasing vagally mediated HRV indices compared with thermoneutral showering.
- The intervention enhanced perceived recovery after the 4-week period, but autonomic and perceptual benefits were not maintained after the 2-week wash-out, suggesting mainly transient recovery effects.
- No clear intervention-specific effects were observed for morning salivary cortisol or PORH-derived microvascular reactivity, indicating limited evidence for endocrine or microvascular adaptation beyond training-related changes.

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