Author(s): Caldwell, Lydia K.; Kraemer, William J.; Post, Emily M.; Volek, Jeff S.; Focht, Brian C.; Newton, Robert U.; Häkkinen, Keijo; Maresh, Carl M.

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Lydia K. Caldwell[1,2], William J. Kraemer[2,4], Emily M. Post[2,3], Jeff S. Volek[2], Brian C. Focht[2], Robert U. Newton[4], Keijo Häkkinen[5], and Carl M. Maresh[2]

[1]Department of Kinesiology, Health Promotion and Recreation, University of North Texas, Denton, TX; [2]Department of Human Sciences, The Ohio State University, Columbus, OH; [3]Department of Exercise Science, Ohio Dominican University, Columbus, OH; [4]Exercise Medicine Research Institute, Edith Cowan University, Joondalup WA 6027, AUSTRALIA; [5]Neuromuscular Research Center, Biology of Physical Activity, Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, FINLAND

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[1]Department of Kinesiology, Health Promotion and Recreation, University of North Texas, Denton, TX; [2]Department of Human Sciences, The Ohio State University, Columbus, OH; [3]Department of Exercise Science, Ohio Dominican University, Columbus, OH; [4]Exercise Medicine Research Institute, Edith Cowan University, Joondalup WA 6027, AUSTRALIA; [5]Neuromuscular Research Center, Biology of Physical Activity, Faculty of Sport and Health Sciences, University of Jyväskylä, Jyväskylä, FINLAND

Address for Correspondence:

William J. Kraemer, PhD, Professor, Department of Human Sciences, The Ohio State University, A054 PAES Building, 305 Annie and John Glenn Avenue, Columbus, OH 43210; Phone: 860-208-5189; E-mail: drwilliamkraemer@gmail.com

ABSTRACT

Purpose: The aim of the present investigation was to determine whether a one-hour floatation-REST session could augment recovery from high-intensity resistance exercise (6 x10 back squats, 2 minutes rest) known to induce significant metabolic, adrenergic, and mechanical stress. **Methods:** Eleven healthy resistance-trained males (age: 22.5± 2.3 years; height: 176.4±6.0 cm; weight: 85.7±6.2 kg, back squat 1RM: 153.1±20.1 kg; strength to weight ratio: 1.8±0.2) completed the within-subjects, cross-over controlled study design. Participants completed two exercise testing blocks separated by a two-week washout. In one block, the high-intensity resistance exercise protocol was followed by a one-hour floatation-REST session, while recovery in the alternate block consisted of a passive sensory-stimulating control. Markers of metabolic stress, neuroendocrine signaling, structural damage, inflammation, and perceptions of soreness, mood state and fatigue were assessed over a 48-hour recovery window. **Results:** Floatation-REST significantly attenuated muscle soreness across recovery (p = 0.035) with greatest treatment difference immediately following the intervention (p = 0.002, ES = 1.3). Significant differences in norepinephrine (p =0.028, ES = 0.81) and testosterone (p =0.028, ES = 0.81) immediately following treatment revealed modification of neuroendocrine signaling pathways which were accompanied by greater improvements in mood disturbance (p = 0.029, ES = 0.81) and fatigue (p = 0.001, ES = 1.04). **Conclusions:** As no adverse effects and significant and meaningful benefits were observed, floatation-REST may prove a valuable intervention for managing soreness and enhancing performance readiness following exercise.

Key Words: FLOAT, SORENESS, FATIGUE, MUSCLE DAMAGE, TESTOSTERONE

INTRODUCTION

Recently, there has been an explosion of anecdotal claims surrounding the recovery potential of a novel modality called floatation-restricted environmental stimulation therapy (floatation-REST). Recovery from exercise stress continues to be one of the most important topics impacting individuals interested in optimizing performance. Without careful management, the cumulative load of competition and training can lead to nonfunctional overreaching and increased risk of injury and illness (1, 2). Appropriate recovery can restore physiological and psychological functions; however, with typical training sequences providing only a brief timeframe between successive exercise bouts, acute interventions (e.g., massage, cryotherapy, nutritional supplements) are commonly utilized in an attempt to alleviate post-exercise fatigue, minimize soreness and accelerate skeletal muscle repair and remodeling (3).

Floatation-REST is a high-salinity environment which attenuates afferent nervous system signaling to promote relaxation of the body and mind (4). The intervention has become increasingly popular among high-performance populations seeking to accelerate recovery and enhance performance readiness. Use by professional sports teams and endorsements by individual athletes has bolstered mainstream popularity despite limited research. The few studies investigating the use of floatation-REST in sport performance have primarily focused on the use of floatation-REST in mental preparation and visualization prior to competition (5-7). Interestingly, Morgan and colleagues (8) were the first to investigate the use of floatation-REST in recovery from acute exercise stress consisting of 50 maximal isokinetic eccentric knee extensions and flexions at $60^{\circ} \cdot \sec^{-1}$ in untrained individuals. In this study, investigators reported reductions in lactate and perceived pain following a one-hour floatation-REST session compared

to a passive recovery control (8). While this study provided some initial insights into the recovery process, translation of these findings to high performance populations—where the therapy is predominantly being used—is problematic. While single-joint eccentric protocols are effective at eliciting structural damage, they do not reflect the array of physiological demands typical of resistance training programs used to enhance strength and power performances. Most trained individuals engage in dynamic, whole-body resistance-exercise routines with much higher metabolic intensity. The training status of the participant pool is also an important consideration as trained individuals have already developed highly adaptive mechanisms to respond to the stress of exercise (9, 10).

To effectively assess the recovery potential of floatation-REST in trained individuals, an exercise model is required that incites significant metabolic, adrenergic, and mechanical stress. High volume, moderate to high-intensity protocols, incorporating short rest intervals and large muscle groups, produce the largest acute hormonal elevations (11). As such, multiset squat protocols of this nature have consistently been used to study the exercise and recovery paradigm in resistance-trained men (12, 13). Following exercise of this nature, accumulation of metabolic byproducts and depletion of energy stores contribute to the onset of muscular fatigue, as well as the perception of nociceptive pain (14). These factors also affect neuromuscular transmission (15) leading to compromises in muscular performance (e.g., strength and power). While metabolic factors typically resolve within the first few hours of exercise cessation, prolonged performance deficits are attributed to structural damage (16) and the resulting inflammatory response (17) required for effective repair and remodeling of muscle tissue. Adding to the complexity of the recovery process are perceptions of soreness, fatigue, and mood disturbance, which interact with

peripheral symptoms to influence performance readiness. As recovery from exercise is a multivariate paradigm with many systems attempting to maintain homeostasis, a broad selection of biomarkers is required to examine what processes may be affected by the use of acute floatation-REST following intense exercise stress.

To gain insight into the recovery process, the purpose of the present investigation was to examine the effect of acute floatation-REST on recovery from strenuous, high-intensity resistance exercise in trained men. In general, we hypothesized that floatation-REST will enhance the recovery process due to the physiological and psychological relaxation provided by the therapy. Owing to the diverse temporal response patterns observed for different physiological systems, we compared a one-hour floatation-REST session to a passive control condition, delivered immediately following resistance exercise. Measures of metabolic stress, neuroendocrine signaling, structural damage, inflammation, muscular performance, and psychological perception were assessed over the first 48-hours of recovery.

METHODS

Experimental Approach to the Problem

The present investigation utilized a within-subjects, crossover-controlled research design to evaluate the impact of acute floatation-REST on recovery from high-intensity resistance exercise. A classic multiset stress model was employed involving six sets of 10 barbell back squats at 80% one-repetition maximum (RM) with 2 minutes rest between sets. The dynamic stimulus induced substantial homeostatic perturbations, disrupting the functional and structural integrity of the involved muscle mass, and creating a significant metabolic and adrenergic response pattern

challenging both physiological and psychological recovery. The model thereby provided a valid method of examining the effectiveness of floatation therapy in augmenting the post-exercise recovery process in trained men.

Experimental Methods

Participants

Eleven healthy resistance-trained males (age: 22.5± 2.3 years; height: 176.4±6.0 cm; weight: 85.7±6.2 kg, back squat 1RM: 153.1±20.1 kg; strength to weight ratio: 1.8±0.2) completed the study. Participants confirmed resistance training history of at least two times per week for the last six months with regular incorporation of the barbell back squat and an ability to squat at least 150% body weight. All participants were cleared of injuries and medical complications that could confound study results, including the use of medications and hormonal substances that may alter the acute response to exercise. Prior to enrollment, participants were objectively informed of the study procedures as well as associated benefits and risks. All participants provided written informed consent in accordance with The Ohio State University's Institutional Review Board for protection of human subjects.

Procedures

Experimental Progression

Following enrollment and familiarization, participants completed two exercise testing blocks separated by a two-week washout period (Figure 1A). Each block consisted of three consecutive laboratory visits (i.e., exercise visit, +24 HR recovery visit, +48 HR recovery visit). The testing blocks were identical except for the recovery intervention occurring during the exercise

visit. In one block, the acute heavy resistance exercise protocol (AHREP) was followed by a 1-hour floatation session, while the alternate block involved the AHREP followed by a seated control condition. Treatment order was counterbalanced by enrollment. Markers of metabolic stress, structural damage, inflammation, muscular performance, and psychological perception were measured at five time points during each testing block: before exercise (PRE), immediately post-exercise (IPE), post 1-hour recovery intervention (P1R), twenty-four hours post-exercise (+24) and forty-eight hours post-exercise (+48) (Figure 1B).

Familiarization Visits

The purpose of the first visit (FAM 1) was to expose participants to floatation therapy. After an introduction to the float room and explanation of all procedures, participants completed a 1-hour float session. To conclude the visit, participants engaged in a conversational debrief with the investigator. The debrief was used to discuss the floatation experience and ensure any barriers to full relaxation were resolved before the onset of testing.

The second visit (FAM 2) was used for familiarization with the testing protocols. A full run-through of the exercise testing visit was conducted with explanations and demonstrations from the research team. During the exercise portion of this visit, participants completed a one-repetition maximum (1RM) for the back squat (18). Following 1RM determination, all participants received a second 1-hour float exposure.

Study Design Controls

All testing visits began at 6 AM. Participants reported to the laboratory for each visit after an overnight fast of 8 hours. Additional restrictions included 12 hours without caffeine, 24 hours without alcohol/medications, and 72 hours without exercise (except for laboratory testing). At the beginning of each visit, hydration status was assessed via urine specific gravity (USG) using a handheld refractometer (Reichert, New York, NY, USA). Participants were required to demonstrate a minimum level of hydration (USG $\leq$ 1.020) prior to engaging in experimental testing. Participants remained fasted throughout the entire visit but were encouraged to consume water ad libitum. Additionally, a 3-day diet log was recorded during the first testing block. Participants were instructed to record all food and beverages consumed, beginning 24 hours prior to the exercise visit. This 3-day diet log was then replicated during the second testing block—matching both food intake and timing. Furthermore, during each testing block, participants were prohibited from engaging in any recovery methods other than the prescribed laboratory treatment.

The Acute Heavy Resistance Exercise Protocol

The AHREP consisted of 6 sets of 10 repetitions of the barbell back squat with two-minute rest intervals. Starting weight was set at 80% 1 RM. If spotter assistance was required to complete a set, the barbell weight was reduced by 10% for the following set. If the set was completed successfully, the weight was maintained. A standardized dynamic warm-up was performed prior to exercise test initiation as well as a brief warm-up with the barbell—providing a gradual increase to the working weight. Peak heart rate and rating of perceived exertion (RPE) were recorded for each set. Heart rate was monitored continuously using a Polar H10 chest strap (Polar Electro Inc., Lake Success, NY). RPE was assessed immediately after the last repetition of a set using the CR-

10 with free magnitude estimation (i.e., ratings from 0 to 10 and higher if perceptual stress was greater) (19).

Floatation Recovery Intervention

All floatation sessions took place in a customized laboratory float room located in the Department of Human Sciences/Kinesiology laboratories at The Ohio State University. The room contains a private shower with a Deluxe Quest Float Suite (Superior Float Tanks, Norfolk, VA). The 94 x 78 x 88-inch fiberglass tank contains approximately 300 gallons of water, creating a depth of about 10 inches. The water is saturated with greater than 1,200 pounds of USP grade Epsom salt (MgSO4) and maintained at a specific gravity of ~1.26 g/cm^3. In-tank heaters provide a steady thermal environment 34.44ºC (94 ºF), approximating that of skin temperature. Before entering the floatation tank, participants showered with sulfate-free shampoo and body wash. After showering, minor skin irritations were covered with Vaseline and earplugs were inserted. Next, participants entered the float tank and reclined on the back with arms placed at the sides. The session was conducted in the absence of light and sound. Additionally, participants floated nude to further reduce somatosensory sensation. At the completion of the hour session, a dim light came on within the tank and a gentle voice alerted the participant that the session was over. The participant then exited the tank and showered before returning to the lab for P1R measurements.

Control Recovery Intervention

The control recovery intervention was designed to provide a relaxing yet sensory-stimulating environment to match the timing of the floatation-REST exposure. During the one-hour recovery, participants sat in a reclining phlebotomy chair in a well-lit room and watched

episodes of *Planet Earth* (BBC Earth) on a tablet computer. In direct contrast to floatation-REST, the television program provided continuous auditory and visual stimulation. The nature documentary was selected to minimize emotional arousal. Episodes were carefully selected and standardized across participants. The episodes depicted diverse geographic landscapes and wildlife scenes, while deliberately excluding more violent content that may provoke a heightened physiological response and impede relaxation (20). While watching the episodes, participants were instructed to engage the footrest on the reclining chair to provide support to the lower body and minimize movement. Just as in the float condition, at the end of the hour recovery session, participants showered prior to returning to the lab for P1R measurements.

Blood Collection and Biochemical Analysis

Blood samples were obtained five times during each testing block: PRE, IPE, P1R, +24, and +48. At each time point, participants sat in a partially reclined phlebotomy chair while blood was collected by a trained phlebotomist from an antecubital vein with a 21-gauge needle (BD Vacutainer Safety-Lok Blood Collection Set, Becton Dickinson and Company, Franklin Lakes, NJ). Blood was immediately centrifuged at 2,000 x *g* for 15 minutes (4°C) and plasma harvested while serum was clotted at room temperature for 30 minutes prior to centrifugation. Resulting serum and plasma were aliquoted into appropriate storage tubes and stored at -80°C. Samples underwent a single freeze-thaw cycle prior to assay. Serum was obtained for analysis of cortisol, total testosterone, myoglobin, creatine kinase, IL-6, and TNF-alpha. Heparinized plasma was obtained for analysis of norepinephrine and epinephrine, while K_2EDTA plasma was obtained for analysis of lactate.

Cortisol, myoglobin, and total testosterone were measured via solid-phase, competitive chemiluminescent immunoassay using an Immulite 1000 analyzer (Siemens Healthcare Diagnostics, Inc., Deerfield, IL). IL-6 and TNF-alpha were multiplexed using an electrochemiluminescence method and read using the Meso QuickPlex SQ 120 (Meso Scale Discovery, Rockville, MD). Creatine kinase was analyzed using the Dimension Xpand Clinical Chemistry System (Siemens Medical Diagnostics, Decatur, GA). Norepinephrine and epinephrine were determined via high-performance liquid chromatography (Quest Diagnostics Nichols Institute, Chantilly VA) and lactate was analyzed using a ChemWell automated chemistry analyzer (Awareness Technology, Inc., Palm City, FL). For all biochemical analyses the intra-assay coefficient of variation was less than 9% (range 2.8-8.7%).

Performance Jumps

Lower body power was assessed using a series of single squat (SJ) and countermovement (CMJ) jumps. Three trials were performed for each jump. Jumps were performed in alternating order, SJ then CMJ, with 30 seconds rest between jumps. Verbal commands were standardized across trials. Hands were placed on the hips throughout the entire motion. Power data were collected and analyzed using an AMTI force plate (Advanced Mechanical Technology Inc, Watertown, MA) with Accupower 2.0 software. Only the trial with the highest power output was analyzed for each jump.

Psychometric Assessments

Self-report measures were completed electronically at each timepoint to reflect how a participant feels "right now, in the present moment." Perceived pain and muscle soreness were

assessed using visual analogue scale (VAS) with endpoint labels corresponding to "None" on the far left and "As bad as it could be" on the far right. The sensory subscale from the SF-MPQ (21) was completed to provide information on the quality of perceived pain. The PANAS was used to assess positive and negative affect (22) while the POMS-SF was used to examine changes in mood (23). To facilitate interpretation, scores from the self-report metrics were converted to standardized units representing the Percent of Maximum Possible (POMP) for each measure. The 0-100 POMP score was calculated using the equation ((Observed score – minimum possible score)/ (maximum possible score – minimum possible score)) x 100 (24).

Statistical Analyses

Data were analyzed using SPSS version 25 (IBM, Armonk, NY). Gaussian distribution was confirmed and when necessary, a $\log_{10}$ transformation was used. Means and standard deviations were calculated for each variable and differences assessed with parametric statistics. Repeated measures analysis of variance (ANOVA) was used to assess differences in the testing blocks. A 2x6 (Treatment x Set) ANOVA was used to assess mean differences during the AHREP; A 2x3 (Treatment x Time) ANOVA was used to assess changes in lactate; and a 2x5 (Treatment x Time) ANOVA was used for all other variables. Violations of sphericity were adjusted with the Greenhouse-Geisser correction. In the event of a significant F test, pairwise comparisons were further evaluated using Fisher's LSD. Statistical significance for all analyses was set a priori at p ≤ 0.05. Additionally, effect sizes (ES) were calculated to assess magnitude of change between the recovery conditions (ES = [M1-M2] / SD_{dif}), with values of 0.2, 0.5 and 0.8 considered small, medium and large, respectively (25).

RESULTS

The primary objective of this investigation was to determine whether a one-hour floatation-REST session administered immediately after high-intensity resistance exercise would improve recovery processes in healthy resistance-trained men. As recovery is a multifaceted phenomenon, we investigated several critical recovery domains including metabolic stress, neuroendocrine signaling, structural damage, inflammation, muscular performance, and psychological perception. The acute therapy successfully targeted perceptual markers of exercise stress, with the most pronounced improvements occurring in muscle soreness and fatigue. While structural damage and inflammation were not altered by floatation-REST, differences in testosterone and norepinephrine immediately post-recovery indicated significant mediation of neuroendocrine signaling pathways.

The AHREP provided a potent mechanical stimulus with no differences noted between testing blocks. For each set, the average peak heart rate was between 84 and 88% age-predicted max. As displayed in Figure 2, perceived exertion increased over time, despite concurrent reductions in barbell loading.

The exercise protocol induced significant sympathetic, adrenergic, and metabolic responses. Means and standard deviations are presented in Table 1. The responses were transient, with substantial recovery occurring over the 1-hour treatment window. While epinephrine and cortisol both returned to pre-exercise values by P1R, norepinephrine revealed a significant treatment x time interaction—returning to baseline following floatation-REST but remaining elevated following the sensory stimulating control ($p = 0.028$; ES = 0.81). Testosterone also demonstrated a significant treatment x time interaction. Both recovery conditions revealed a

similar temporal response pattern, however, the degree of testosterone decline at P1R was significantly greater in the control condition compared to floatation-REST (p = 0.028; ES = 0.81).

Biomarkers of structural damage and inflammation were significantly elevated following exercise, however no treatment differences were observed in any of the response profiles. The main effect of time is displayed in Figure 3. Likewise, the AHREP resulted in compromised jump performance, irrespective of recovery condition. The most substantial reductions in jump height and peak power were observed IPE with incremental improvements occurring over the 48-hour recovery period. Main effects for time are displayed for the SJ and CMJ as percent change from PRE— SJ (jump height) IPE: -25%, PIR: -14%, +24: -8%, +48: -3%; SJ (peak power) IPE: -17%, PIR: -12%, +24: -6%, +48: -3%; CMJ (jump height) IPE: -24%, PIR: -15%, +24: -6%, +48: -7%. CMJ (peak power) IPE: -15%, PIR: -11%, +24: -4%, +48: -3%.

Pain and soreness were significantly elevated at all post-exercise time points (Figure 4). The degree of soreness, however, was significantly reduced following recovery with floatation-REST. The greatest treatment difference was observed directly following the intervention (p = 0.002, ES = 1.3), with intensity of post-exercise soreness rated 36% lower than control. While there were no treatment differences in pain intensity, the sensory subscale from the SF-MPQ revealed differences in the way participants experienced pain. Largest differences were noted immediately after recovery with 73% of participants describing pain as aching and heavy following control compared to 45% after floatation-REST. Other noticeable differences were the appearance of more severe pain characteristics (e.g., splitting, shooting, stabbing) at +24 following the control treatment despite similar treatment profiles between the recovery conditions at +48.

Psychometric data from the PANAS and POMS are presented in Figure 5 (see Tables S1 and S2, Supplemental Digital Contents 1 and 2, http://links.lww.com/MSS/C540, http://links.lww.com/MSS/C541, for means and standard deviations). While it was anticipated that PRE and IPE values would be similar between testing blocks, participants seemed to respond less favorably post-exercise in the floatation-REST testing block. Following the AHREP, participants experienced increased negative affect (p = 0.003; ES = 1.26), with trends towards higher mood disturbance (p = 0.072; ES = 0.64) and increased fatigue (p = 0.079; ES = 0.62). Interestingly, these treatment differences were not observed following recovery (P1R, +24, +48). To further evaluate the impact of the recovery intervention on these variables, change scores were constructed relative to PRE and compared between treatment conditions. Floatation-REST led to significantly greater improvements in negative affect (p = 0.020, ES = 0.87) total mood disturbance (p = 0.029, ES = 0.81) and experienced fatigue (p = 0.008, ES = 1.04) over the one-hour recovery intervention (PIR – IPE).

DISCUSSION

The primary purpose of this study was to investigate what aspects of the recovery process—if any—were affected when floatation-REST was delivered directly following high-intensity exercise in resistance-trained men. The utility of this novel modality was evaluated with respect to several essential domains involved with short-term recovery processes. The most compelling findings were an attenuation of perceived muscle soreness and immediate improvements in mood state and fatigue compared to the sensory-stimulating control. These data suggest a positive impact of floatation-REST in the first 48 hours of exercise recovery.

Recovery depends upon the nature of the stressor and the physiological systems which respond to the homeostatic threat. The exercise protocol in this study produced a dramatic sympathetic and adrenergic stress response. Serum cortisol increased 44% during exercise but returned to baseline values following recovery with both floatation-REST and control. The lack of treatment effect was surprising considering previous investigations of floatation-REST—conducted in the absence of exercise—have consistently demonstrated reductions in basal cortisol of approximately 20% without significant change in control groups (26, 27). The ability of floatation-REST to effectively reduce cortisol at rest but impart no benefit following a direct challenge of the hypothalamic-pituitary-adrenal (HPA) axis—as delivered in this study—may be explained by the highly sensitive negative feedback system observed in trained men (28). While the participants in the current study demonstrated healthy patterns of HPA arousal and subsequent inhibition following exercise, it remains unknown how floatation-REST may benefit athletes displaying signs of HPA dysfunction as is common in chronic stress and states of overtraining (29).

While the tropic signaling of the HPA axis demonstrated potent negative feedback mechanisms that may limit the effect of floatation-REST on this system immediately following exercise, sympathetic nervous system stress appeared to be more sensitive to the reduced sensory environment. In the current study, floatation-REST significantly reduced sympathetic-adrenal-medullary activity following exercise. While plasma epinephrine and norepinephrine were drastically elevated during the resistance exercise protocol, both catecholamines returned to pre-exercise concentrations following the one-hour floatation-REST session. In contrast, norepinephrine remained significantly elevated immediately following recovery with the sensory-

stimulating control. Taken together, these findings suggest that acute floatation-REST used in immediate recovery from intense exercise has a differential influence on nervous system and endocrine mediated stress signaling.

Immediate treatment differences were also noted in the hypothalamic-pituitary-gonadal axis. This pathway plays a vital role in recovery from exercise with multitudes of genomic and non-genomic signaling pathways (30). As expected, the high-volume, short rest resistance exercise protocol resulted in significant elevations in total testosterone, which fell below baseline values into recovery. Interestingly, testosterone was significantly higher immediately after floatation-REST compared to control. The differential response pattern may have important implications for muscle repair and remodeling as the steroid hormone has both anabolic and anti-catabolic effects (30). Elevations in testosterone may be attributed to reduced hepatic clearance, decreased plasma volume, or increased secretory behavior (31). While the mechanism in this context is unclear, elevated concentrations of testosterone enhance the probability of hormone-receptor interactions and therefore, initiation of anabolic signaling pathways.

Anabolic signaling is augmented by increased blood flow following intense exercise (e.g., post-exercise hyperemia, histaminergic vasodilation) (32). Floatation-REST also promotes vasodilation; however, it is unclear whether the acute intervention provides additional hemodynamic benefit in this immediate post-exercise timeframe. It may be that floatation-REST has more pronounced effects on tissues other than skeletal muscle, which already receive local vasodilatory modulation. An increase in blood flow to the testes, for example, could explain the greater availability of testosterone following recovery with floatation-REST. In contrast, other

popular recovery modalities (e.g., cryotherapy, cold water immersion) promote vasoconstriction, thereby decreasing tissue exposure to potent anabolic signals (12) which may contribute to compromised training adaptions when these types of recovery interventions are used regularly. This hypothesis is strongly supported by the findings of Roberts et al. (33) who demonstrated attenuated gains in strength and muscle mass following an eight-week resistance training program when cold-water immersion was used in recovery. Muscle biopsies revealed suppression of satellite cells as well as a decreased kinase activity in the mTOR signaling pathway compared to an active recovery control (33). Unlike the popular cold-therapies, floatation-REST provides an environment that harnesses the natural post-exercise recovery response by promoting continued vasodilation throughout the recovery treatment.

Training adaptions may also be affected by alterations in inflammatory signaling. The modest elevations observed for systemic inflammation in this study suggest an adaptive immune response in this group of trained men, which may limit the capacity for treatment improvement. While unique temporal response patterns were observed for TNF-alpha and IL-6, there were no treatment differences between recovery conditions. TNF-alpha and IL-6 both peaked immediately following exercise, but quickly recovered to baseline values. The transient responses were similar to those reported by Townsend et al. (34); however the concentrations of circulating hormone were considerably lower than seen in other exercise investigations (13, 35). High levels of physical activity have been associated with systemic reductions in IL-6 as well as upregulation of IL-6 receptor content within the skeletal muscle (36, 37). These adaptations suggest that training increases IL-6 sensitivity, supporting homeostatic regulation in the early inflammatory phase with positive implications for both substrate availability and reduced cellular damage (38). In some

respects, the lack of treatment effect seen in this study may be considered favorable as the one-hour floatation-REST session did not negatively impact the tightly regulated pro-inflammatory phase necessary for initiating the muscle repair process (39).

In the present study, skeletal muscle damage was assessed indirectly through the appearance of myofibrillar proteins in circulation and reductions in explosive power. Given that the AHREP provided an equivalent mechanical (e.g., set load, total mass lifted) and biochemical stimulus (e.g., lactate, catecholamines, cortisol, testosterone) and no alterations were displayed in systemic markers of catabolism (e.g., cortisol, TNF-alpha), it may not be surprising that there were also no treatment differences in structural damage or functional performance. Clearly, the volume and intensity of the resistance exercise protocol used in this study disrupted myofibrillar structure even in highly trained men accustomed to high-force muscle contractions. The degree of damage may have influenced the ability of floatation-REST to mediate performance changes in recovery. Similar to our findings, Morgan et al. (2013) found no treatment differences in functional performance when floatation-REST was compared to a passive control condition following maximal resistance exercise (8). However, when floatation-REST was used in recovery from a simulated team sport competition stress—involving primarily running and jumping tasks—the intervention resulted in improvements in both strength and sprint performance (40). Differences in performance findings between these studies are likely attributed to differences in the underlying mechanism responsible for the performance decrement (fatigue, soreness, structural damage, etc.).

While floatation-REST conferred no benefit for structural damage or muscular performance in this study, the reduced sensory environment led to significant attenuation of muscle

soreness in the first 48-hours of exercise recovery. Floatation-REST has consistently demonstrated efficacy as a pain management intervention outside of exercise recovery with several studies revealing significant improvements in pain symptomology compared to non-treatment controls (41-44). Many of these investigations involved patient populations experiencing stress-related muscle tension and pain while employing a more conventional floatation-REST treatment scheme of 2-3 sessions a week for up to six weeks. In contrast, the exercise recovery literature has demonstrated improvements in pain and soreness after acute exposure, revealing the strength of this treatment effect and the ability of floatation-REST to deliver immediate somatic relief (8, 40).

There is some evidence to suggest that improvements in pain and soreness with floatation-REST may be mediated by baseline intensity. Studies with pain intensity ratings of less than 30 on a 100-point scale (42, 43) have failed to demonstrate treatment improvements, while those with a higher degree of baseline pain (45, 46) reported reductions as 18% and 30%. This may explain why in the present study, floatation-REST significantly improved ratings of muscle soreness but had limited impact on the lower ratings of perceived pain. Recently, the first neuroimaging study on floatation-REST was published and revealed reductions in resting state functional connectivity between brain regions associated with the default-mode network and somatomotor cortices, leading authors to postulate that floatation-REST may act by reducing self-reflective processes regarding the current state of the body (47). This suggests that floatation-REST may act through nervous system modulation allowing for a more positive perception of local environments.

Subjective perceptions of mood and general wellbeing are also susceptible to nervous system modulation. The present study revealed significant reductions in negative affect, total mood

disturbance, and fatigue when floatation-REST was applied in recovery from intense exercise stress. These findings support previous investigations of floatation-REST, which have consistently demonstrated improvements in self-report metrics after a single acute exposure (48, 49). Similar perceptual benefits have been noted in studies involving lengthier intervention schemes (42, 43, 45) which suggests improvements may not merely be an effect of novelty bias or expectancy.

The optimization of sport performance relies on the management of both physiological and psychological factors. While the mechanism remains unclear, the psychological benefit of floatation-REST is well documented in the existing body of float research. Mental state affects many factors including concentration, motivation, and emotion regulation, which together can influence both the decision-making capacity of an individual as well as the effort brought to performance. Several investigations have demonstrated that relaxation and changes in mindset can significantly improve competitive performance when floatation-REST is utilized prior to competition. These investigations demonstrated a variety of performance enhancements in comparison to control groups including higher competition scores in young gymnasts (5), greater number of first service winners in collegiate tennis players (50) and improved free throw percentage in recreational basketball players (6). The ability of floatation-REST to improve psychological perceptions is an important finding with profound implications for performance readiness and resiliency.

CONCLUSIONS

In summary, our findings from this investigation are that floatation-REST differentially mediates the observed recovery domains following demanding resistance exercise in highly trained

men. The most beneficial treatment effects occurred in areas of perceived recovery with significant improvements demonstrated for muscle soreness, mood state and fatigue. Acute post-exercise compensations (hyperemia and negative feedback of the HPA axis), as well as the training status of participants, may have contributed to the limited treatment effect observed for structural damage, inflammation, and functional performance. However, significant, and salient alterations in norepinephrine and total testosterone following floatation-REST treatment suggest meaningful modification of neuroendocrine signaling pathways, which may mediate positive recovery processes.

As we are just beginning the systematic evaluation of this modality in exercise recovery, more research is needed and future work may benefit from examining whether similar results are obtained when floatation-REST is used regularly or at a different stage in the recovery process. It is important to consider the sample size when evaluating the results of this study as only large effect sizes would be detectable in a sample of this size. Interestingly, the lack of effect on damage markers and inflammatory signaling may suggest the suitability of floatation-REST in the daily training environment, as the reduced sensory treatment improved perceptions of readiness but did not appear to interfere with tissue repair and remodeling. As no adverse effects were observed in any of the recovery domains, floatation-REST may be considered a safe and valuable intervention for promoting relaxation and managing post-exercise soreness and fatigue.

Disclosures

No conflicts of interest, financial or otherwise, are declared by the authors.

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FIGURE LEGENDS

Figure 1. (A) Overview of the experimental design (B) assessment timepoints within each testing block. AHREP, acute heavy resistance exercise protocol; FAM 1, familiarization visit 1; FAM 2, familiarization visit 2; EX, exercise visit; 24 HR, twenty-four-hour recovery visit, 48 HR, forty-eight-hour recovery visit; PRE, before exercise; IPE, immediately post-exercise; P1R, post 1-hour recovery intervention; +24, twenty-four hours post-exercise; 48, forty-eight hours post exercise.

Figure 2. Change in (A) barbell load (B) total mass lifted [barbell load x unassisted repetitions] and (C) perceived exertion during the AHREP. Data represent the main effect for time and are expressed as means ± 95% confidence intervals. Values not sharing a common letter were significantly different (P ≤ 0.05).

Figure 3. Changes in biomarkers of structural damage and inflammation following the AHREP and acute recovery intervention. Data represent the main effect for time and are presented as mean ± 95% CI. Values not sharing a common letter were significantly different (P ≤ 0.05).

Figure 4. Change in VAS pain and soreness scores. Data are presented as means ± 95% CI. Values not sharing a common letter represent differences across time (P < 0.05). [T]P indicates a significant treatment effect.

Figure 5. Treatment differences in affect, mood, and fatigue. Data represent treatment change scores (Float - Control) for each PRE adjusted timepoint. Data are presented as means ± 95% CI. Values not sharing a common letter were significantly different (P ≤ 0.05).
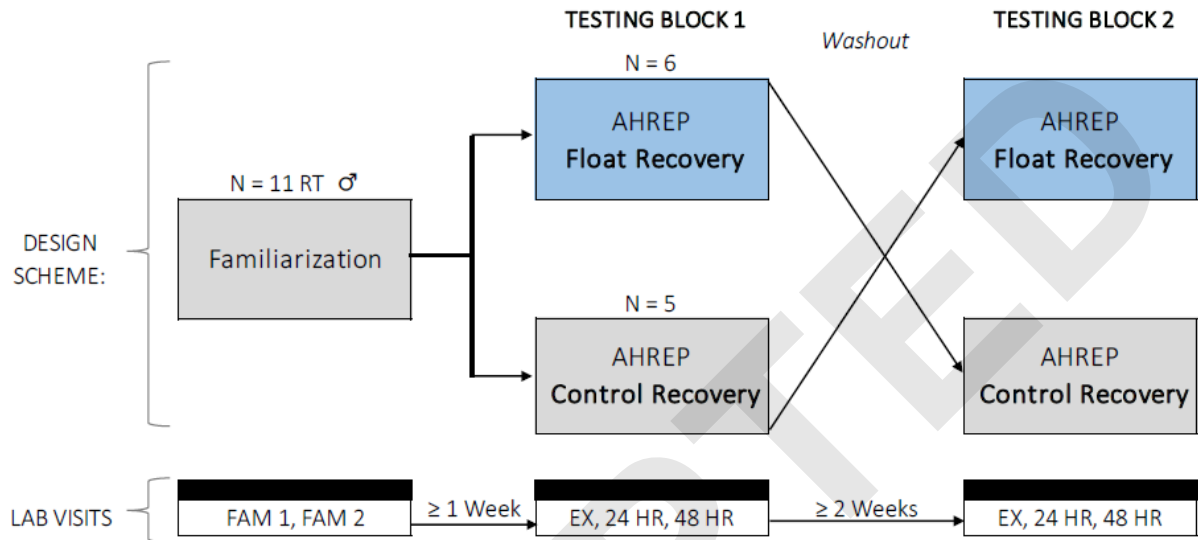
SUPPLEMENTAL DIGITAL CONTENT

SDC 1: SDC_Table S1.pdf – Change in affect between control and float interventions following AHREP.

SDC 2: SDC_Table S2.pdf – POMS-SF: Change in mood between control and float recovery interventions following AHREP.

Figure 1

A) Overall Design Scheme
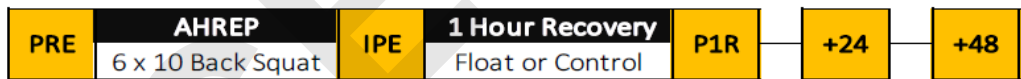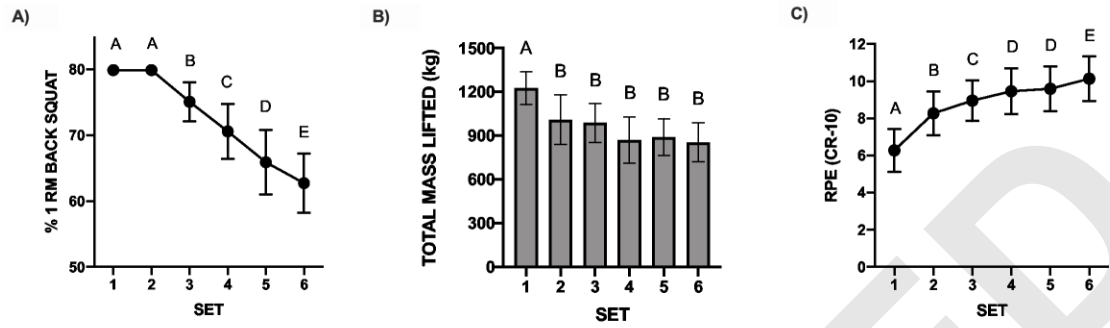

B) Assessment Timepoints

Figure 2
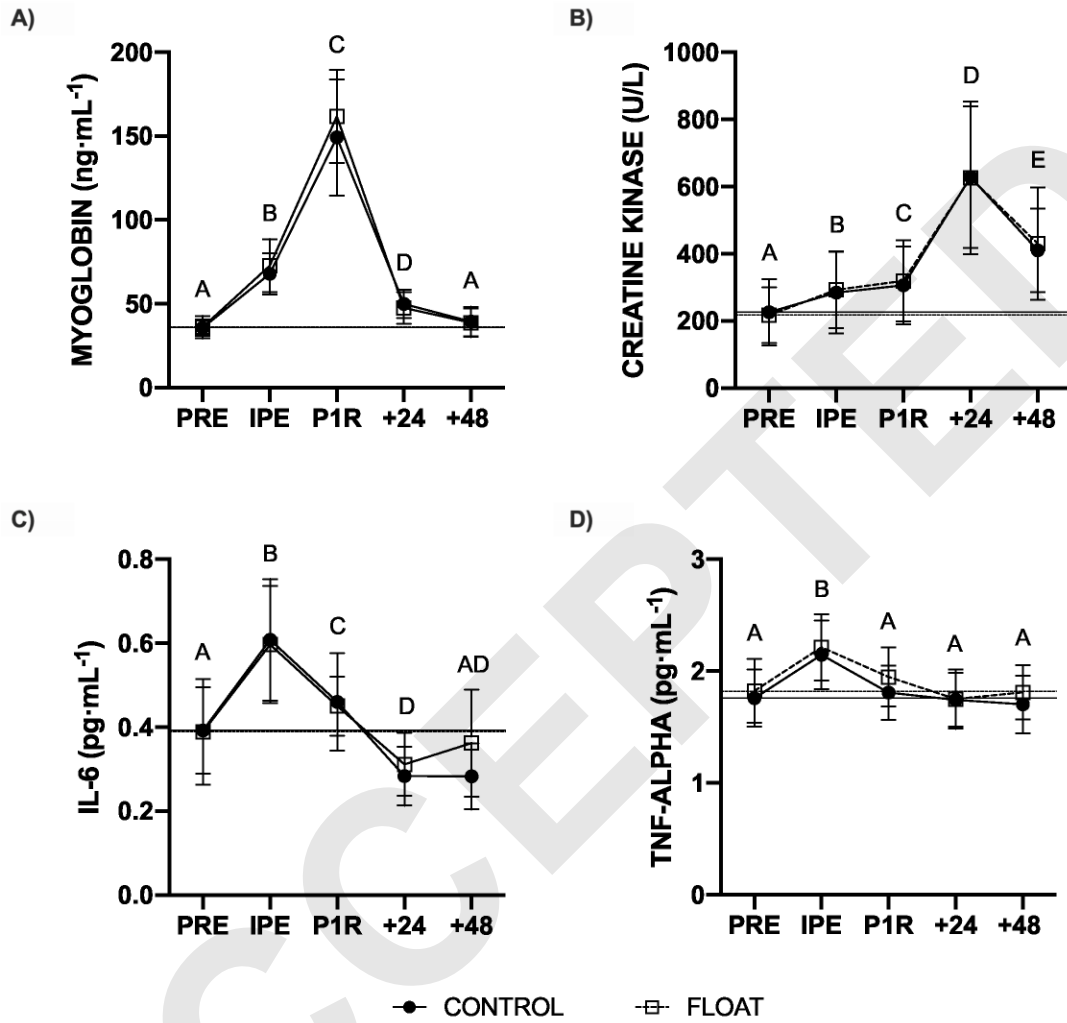
Figure 3

Figure 4
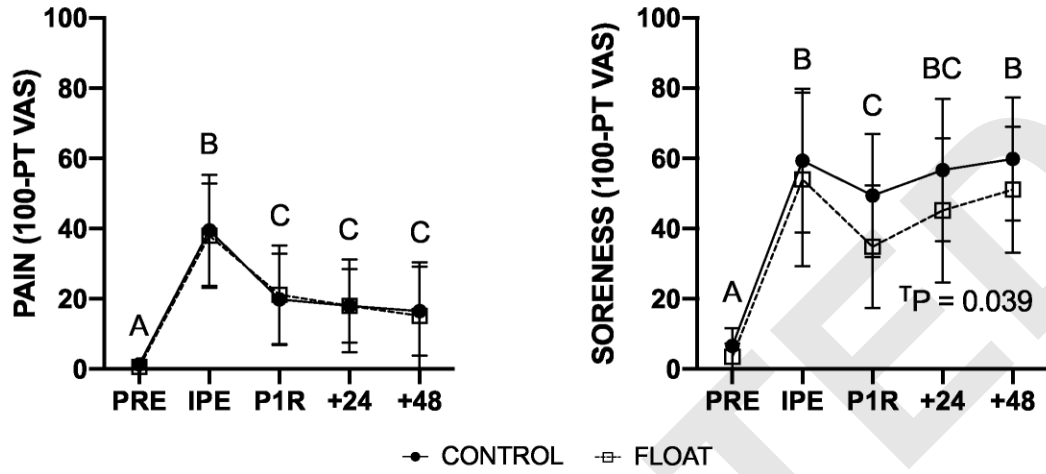
Figure 5
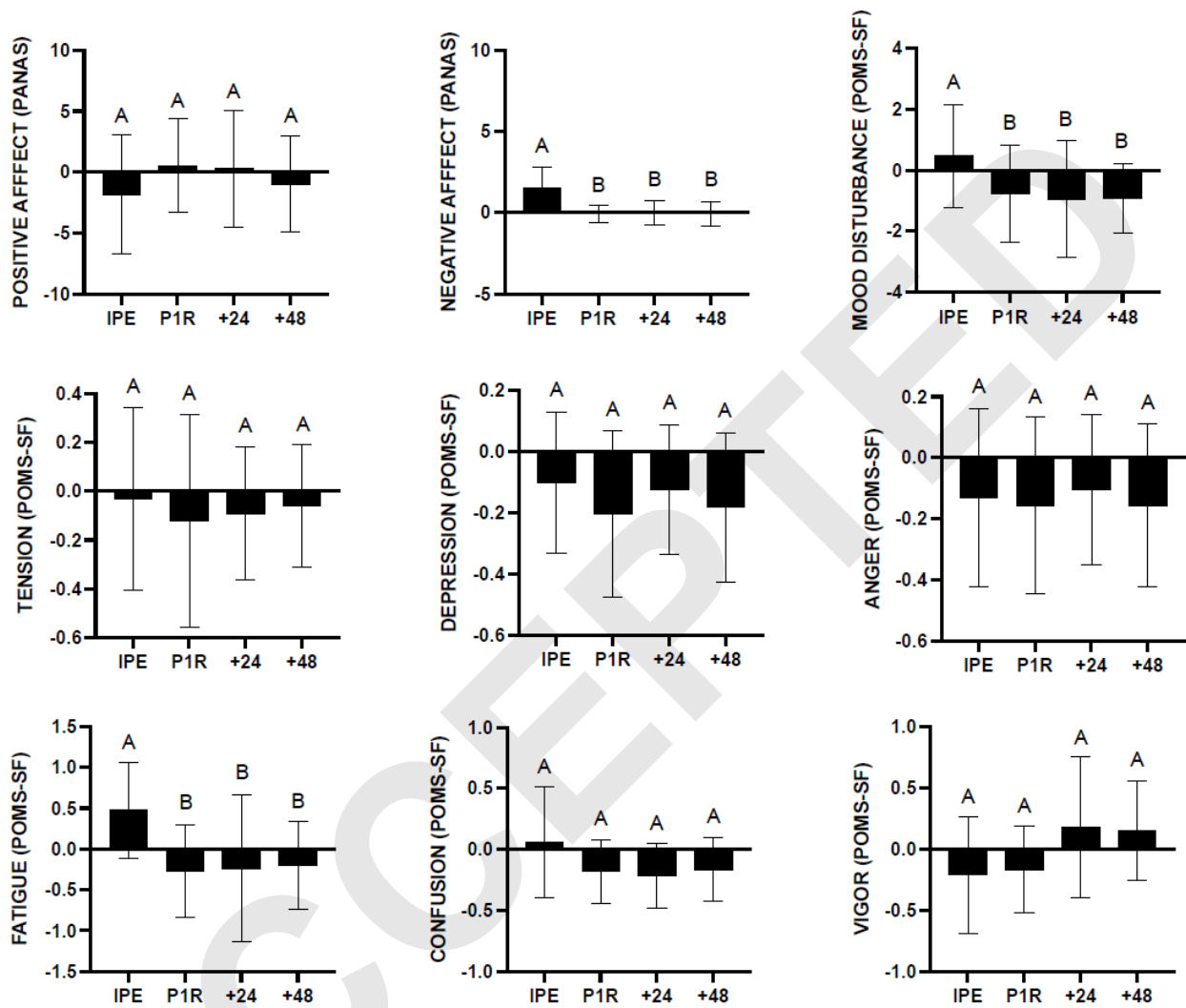
Table 1. Metabolic, sympathetic, and adrenergic responses to AHREP and acute recovery

BIOMARKER		PRE	IPE	P1R	+24	+48
Lactate	Control	0.97(0.13)[A]	12.40 (1.62)[B]	1.54 (0.34)[C]		
(nmol·L-1)	Float	1.02 (0.20)[A]	13.22(1.67)[B]	1.55 (0.30)[C]		
Epinephrine	Control	0.20 (0.10)[A]	1.40 (0.65)[B]	0.25 (0.09)[A]	0.22 (0.13)[A]	0.23 (0.14)[A]
(nmol·L-1)	Float	0.21 (0.14)[A]	1.58 (0.60)[B]	0.29 (0.15)[A]	0.16 (0.05)[A]	0.20 (0.07)[A]
Norepinephrine	Control	1.37 (0.41)[A]	13.23 (3.46)[B]	1.97 (0.57)[C]	1.67 (0.59)[A]	1.41 (0.33)[A]
(nmol·L-1)	Float	1.47 (0.35)[A]	13.99 (5.45)[B]	1.52 (0.37)[A*]	1.48 (0.54)[A]	1.79 (0.43)[C*]
Cortisol	Control	429.03 (143.65)[A]	597.91 (54.00)[B]	403.969 (93.00)[A]	407.15 (103.24)[A]	410.56 (75.43)[A]
(nmol·L-1)	Float	403.74 (106.78)[A]	600.17 (50.96)[B]	405.97 (70.95)[A]	368.2 (114.46)[A]	403.41 (89.53)[A]
Testosterone	Control	16.68 (4.14)[A]	20.28 (3.70)[B]	14.67 (2.46)[C]	16.33 (3.61)[AC]	17.22 (3.90)[A]
(nmol·L-1)	Float	17.01 (3.58)[A]	20.58 (3.63)[B]	15.61 (3.33)[C*]	16.06 (3.66)[AC]	16.40 (3.58)[A]

Data are presented as Mean (SD). The * indicates significant treatment x time interaction ($P \leq 0.05$).
Simple effects for time are also provided for each recovery condition; values not sharing a common letter were significantly different ($P \leq 0.05$).

Table S1. PANAS: Change in affect between control and float interventions following AHREP

		PRE	IP	1R	+24	+48
Positive	Control	48.2 (22.9)[A]	51.4 (26.6)[A]	48.4 (24.9)[A]	45.7 (26.2)[A]	45.7 (27.9)[A]
	Float	44.8 (22.1)[A]	43.4 (22.4)[A]	46.4 (19.9)[A]	43.0 (23.3)[A]	39.8 (29.0)[A]
Negative	Control	3.6 (3.0)[A]	2.5 (3.9)[AB]	1.1 (2.6)[B]	0.5 ± (1.0)[B]	1.6 ± (3.9)[AB]
	Float	3.5 (3.2)[A]	6.6 (5.8)[B*]	1.4 (2.6)[AC]	0.2 ± (0.8)[C]	1.4 ± (2.3)[AC]

Data are presented as Mean (SD). The * indicates significant treatment x time interaction (P ≤ 0.05). Simple effects for time are also provided for each recovery condition; values not sharing a common letter were significantly different (P ≤ 0.05).

Table S2. POMS-SF: Change in mood between control and float recovery interventions following AHREP

		PRE	IP	1R	+24	+48
TMD	Control	13.3 (4.9)[A]	21.1 (8.5)[B]	19.0 (6.9)[B]	17.5 (6.5)[B]	16.3 (7.2) [AB]
	Float	14.9 (7.1)[A]	26.8 (10.8)[B]	19.5 (7.8)[A]	17.3 (6.2)[A]	16.2 (6.5) [A]
Tension	Control	7.6 (6.7)[A]	7.6 (8.3)[AB]	1.9 (2.9)[C]	2.7 (3.4)[ABC]	2.3 (2.9)[AC]
	Float	8.8 (14.3)[A]	11.0 (7.0)[AB]	3.0 (5.3)[C]	4.5 (5.4)[AC]	4.9 (6.1)[AC]
Depression	Control	0.0 (0.0)[A]	2.3 (4.4)[B]	0.6 (1.9)[A]	0.0 ± (0.0)[A]	0.3 (0.9)[A]
	Float	1.9 (10.2)[A]	4.6 (4.9)[B]	0.3 (0.9)[A]	1.7 ± (3.2)[A]	0.6 (1.3)[A]
Anger	Control	0.0 (0.0)[A]	1.0 (3.2)[A]	0.0 (0.0)[A]	3.6 (0.0)[B]	4.5 (2.3)[B]
	Float	0.7 (2.3)[A]	1.6 (3.3)[A]	0.0 (0.0)[A]	4.9 (2.9)[B]	4.3 (2.3)[B]
Fatigue	Control	8.5 (10.4)[A]	46.0 (19.5)[B]	42.0 (17.2)[BC]	27.5 (22.2)[CD]	25.0 (21.5)[AD]
	Float	7.5 (15.1)[A]	61.0 (24.9)[B]	38.5 (23.6)[C]	24.5 (20.4)[A]	20.5 (15.8)[A]
Confusion	Control	1.5 (4.1)[A]	8.0 (10.6)[A]	7.5 (10.8)[A]	2.5 (3.4)[A]	2.5 (3.4)[A]
	Float	6.5 (10.5)[A]	15.5 (22.8)[AB]	9.5 (15.8)[AC]	3.5 (5.1)[AC]	5.0 (7.9)[AC]
Vigor	Control	36.3 (26.5)[A]	31.7 (22.7)[A]	32.5 (25.8)[A]	24.2 (29.1)[A]	30.8 (27.3)[A]
	Float	35.8 (26.3)[A]	24.2 (20.8)[A]	26.7 (20.3)[A]	29.2 (26.4)[A]	35.0 (27.5)[A]

Data are presented as Mean (SD). Simple effects for time are provided for each recovery condition; values not sharing a common letter were significantly different (P ≤ 0.05).