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ABSTRACT

CALDWELL, L. K., W. J. KRAEMER, E. M. POST, J. S. VOLEK, B. C. FOCHT, R. U. NEWTON, K. HÄKKINEN, and C. M. MARESH. Acute Floatation-REST Improves Perceived Recovery After a High-Intensity Resistance Exercise Stress in Trained Men. *Med. Sci. Sports Exerc.*, Vol. 54, No. 8, pp. 1371–1381, 2022. **Purpose:** The aim of the present investigation was to determine whether a 1-h floatation-restricted environmental stimulation therapy (floatation-REST) session could augment recovery from high-intensity resistance exercise (6 × 10 back squats, 2-min rest) known to induce significant metabolic, adrenergic, and mechanical stress. **Methods:** Eleven healthy resistance-trained males (age, 22.5 ± 2.3 yr; height, 176.4 ± 6.0 cm; weight, 85.7 ± 6.2 kg; back squat one-repetition maximum, 153.1 ± 20.1 kg; strength-to-weight ratio, 1.8 ± 0.2) completed the within-subject, crossover controlled study design. Participants completed two exercise testing blocks separated by a 2-wk washout. In one block, the high-intensity resistance exercise protocol was followed by a 1-h floatation-REST session, whereas 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-h recovery window. **Results:** Floatation-REST significantly attenuated muscle soreness across recovery ($P = 0.035$) with greatest treatment difference immediately after the intervention ($P = 0.002$, effect size (ES) = 1.3). Significant differences in norepinephrine ($P = 0.028$, ES = 0.81) and testosterone ($P = 0.028$, ES = 0.81) immediately after treatment revealed the 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:** Because no adverse effects and significant and meaningful benefits were observed, floatation-REST may prove a valuable intervention for managing soreness and enhancing performance readiness after exercise. **Key Words:** FLOAT, SORENESS, FATIGUE, MUSCLE DAMAGE, TESTOSTERONE

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 an 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 time frame between successive exercise bouts, acute interventions (e.g., massage, cryotherapy, nutritional supplements) are commonly utilized in an attempt to alleviate postexercise fatigue, minimize soreness, and accelerate skeletal muscle repair and remodeling (3).

Floatation-REST is a high-salinity environment that 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

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visualization before 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 $60°\cdot s^{-1}$ in untrained individuals. In this study, investigators reported reductions in lactate and perceived pain after a 1-h floatation-REST session compared with a passive recovery control (8). Although 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. Although 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 because 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). After 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). Although 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. Because 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 because of the physiological and psychological relaxation provided by the therapy. Owing to the diverse temporal response patterns observed for different physiological systems, we compared a 1-h floatation-REST session with a passive control condition, delivered immediately after resistance exercise. Measures of metabolic stress, neuroendocrine signaling, structural damage, inflammation, muscular performance, and psychological perception were assessed over the first 48 h of recovery.

METHODS

Experimental Approach to the Problem

The present investigation utilized a within-subject, 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 used involving six sets of 10 barbell back squats at 80% one-repetition maximum (RM) with 2-min 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 postexercise recovery process in trained men.

Experimental Methods

Participants. Eleven healthy resistance-trained males (age, 22.5 ± 2.3 yr; height, 176.4 ± 6.0 cm; weight, 85.7 ± 6.2 kg; back squat one-repetition maximum (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 6 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. Before 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.*** After enrollment and familiarization, participants completed two exercise testing blocks separated by a 2-wk washout period (Fig. 1A). Each block consisted of three consecutive laboratory visits (i.e., exercise visit, +24-h recovery visit, +48-h 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-h floatation session, whereas 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 after exercise (IPE), after 1-h recovery intervention (P1R), 24 h after exercise (+24), and 48 h after exercise (+48) (Fig. 1B).

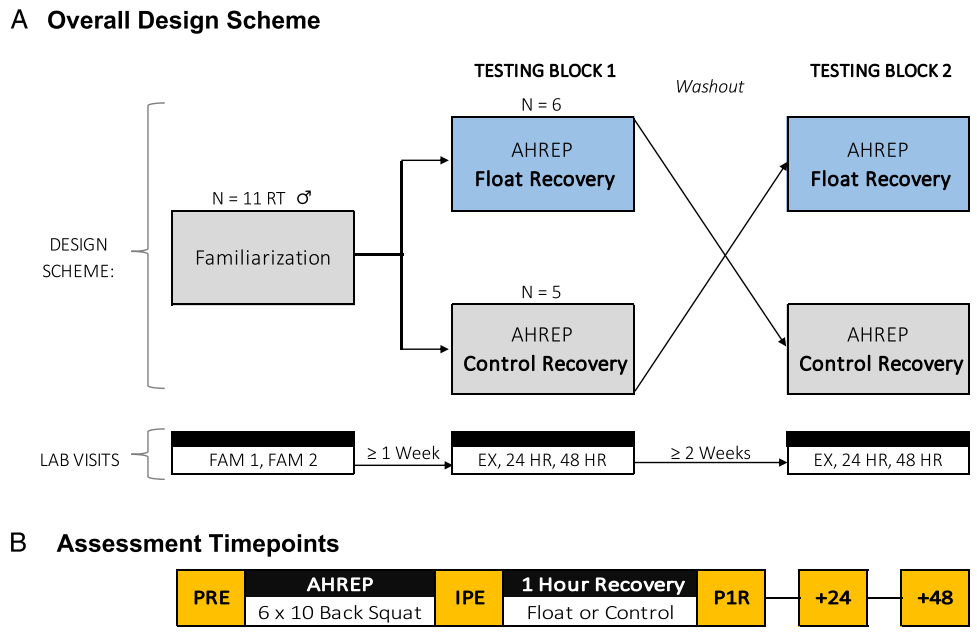

FIGURE 1—A, Overview of the experimental design. B, Assessment time points within each testing block. FAM 1, familiarization visit 1; FAM 2, familiarization visit 2; EX, exercise visit; 24 HR, 24-h recovery visit; 48 HR, 48-h recovery visit.

Familiarization visits. The purpose of the first visit was to expose participants to floatation therapy. After an introduction to the float room and explanation of all procedures, participants completed a 1-h 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 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 1RM for the back squat (18). After 1RM determination, all participants received a second 1-h 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 h. Additional restrictions included 12 h without caffeine, 24 h without alcohol/medications, and 72 h without exercise (except for laboratory testing). At the beginning of each visit, hydration status was assessed via urine-specific gravity using a handheld refractometer (Reichert, New York, NY). Participants were required to demonstrate a minimum level of hydration (urine-specific gravity ≤1.020) before engaging in experimental testing. Participants remained fasted throughout the entire visit but were encouraged to consume water *ad libitum*. In addition, a 3-d diet log was recorded during the first testing block. Participants were instructed to record all food and beverages consumed, beginning 24 h before the exercise visit. This 3-d 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 2-min rest intervals. Starting weight was set at 80% 1RM. 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 before 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 were recorded for each set. Heart rate was monitored continuously using a Polar H10 chest strap (Polar Electro Inc., Lake Success, NY). Rating of perceived exertion 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 × 78 × 88-inch fiberglass tank contains approximately 300 gallons of water, creating a depth of about 10 inches. The water is saturated with greater than 1200 lb of USP grade Epsom salt ($MgSO_4$) and maintained at a specific gravity of ~1.26 $g \cdot cm^{-3}$. In-tank heaters provide a steady thermal environment 34.44°C (94°F), 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. In addition, 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 laboratory 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 1-h 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 before returning to the laboratory 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 2000*g* for 15 min (4°C) and plasma harvested while serum was clotted at room temperature for 30 min before centrifugation. Resulting serum and plasma were aliquoted into appropriate storage tubes and stored at −80°C. Samples underwent a single freeze–thaw cycle before assay. Serum was obtained for analysis of cortisol, total testosterone, myoglobin, creatine kinase, interleukin (IL) 6, and tumor necrosis factor α (TNF-α). Heparinized plasma was obtained for analysis of norepinephrine and epinephrine, whereas 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-α 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-s 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 time point to reflect how a participant feels "right now, in the present moment." Perceived pain and muscle soreness were assessed using visual analog scale with end point labels corresponding to "None" on the far left and "As bad as it could be" on the far right. The sensory subscale from the Short-Form McGill Pain Questionnaire (21) was completed to provide information on the quality of perceived pain. The Positive and Negative Affect Schedule was used to assess positive and negative affect (22), whereas the Profile of Mood States—Short Form 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 for each measure. The 0–100 Percent of Maximum Possible score was calculated using the equation ((Observed score − minimum possible score)/(maximum possible score − minimum possible score)) × 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 SD were calculated for each variable and differences assessed with parametric statistics. Repeated-measures ANOVA was used to assess differences in the testing blocks. A 2 × 6 (treatment–set) ANOVA was used to assess mean differences during the AHREP, a 2 × 3 (treatment–time) ANOVA was used to assess changes in lactate, and a 2 × 5 (treatment–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 least significant difference. Statistical significance for all analyses was set *a priori* at $P \leq 0.05$. In addition, 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 1-h floatation-REST session administered immediately after high-intensity resistance exercise would improve recovery processes in healthy resistance-trained men. Because recovery is a multifaceted phenomenon, we investigated several critical recovery domains including metabolic stress, neuroendocrine signaling, structural damage, inflammation,

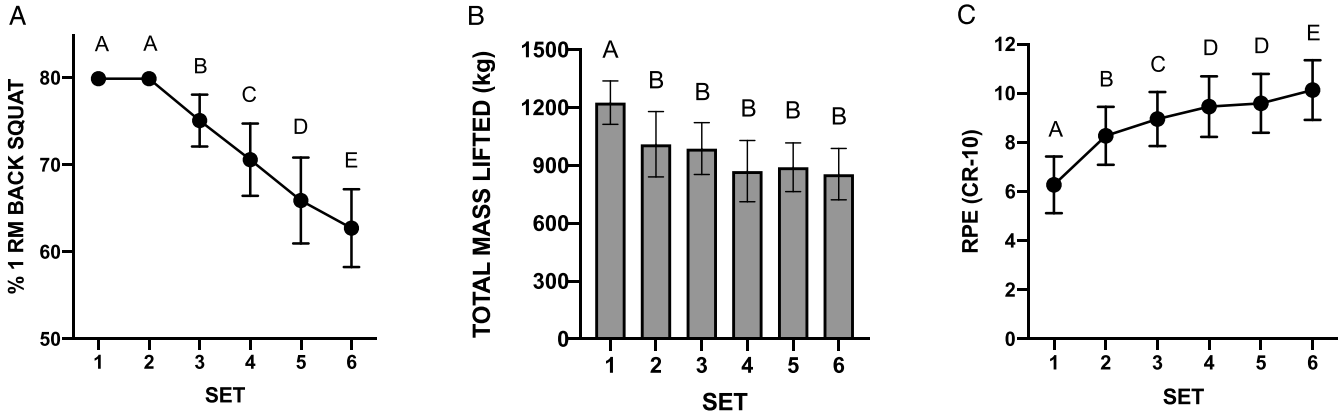

FIGURE 2—Change in (A) barbell load and (B) total mass lifted (barbell load × unassisted repetitions) and (C) perceived exertion during the AHREP. Data represent the main effect for time and are expressed as means ±95% confidence intervals. Values not sharing a common letter were significantly different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

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. Although structural damage and inflammation were not altered by floatation-REST, differences in testosterone and norepinephrine immediately after 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 maximum. 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 SD are presented in Table 1. The responses were transient, with substantial recovery occurring over the 1-h treatment window. Although epinephrine and cortisol both returned to preexercise values by P1R, norepinephrine revealed a significant treatment–time interaction —returning to baseline after floatation-REST but remaining elevated after the sensory stimulating control ($P = 0.028$, ES = 0.81). Testosterone also demonstrated a significant treatment–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 with floatation-REST ($P = 0.028$, ES = 0.81).

Biomarkers of structural damage and inflammation were significantly elevated after 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-h recovery period. Main effects for time are displayed for the SJ and CMJ as percent change from PRE: −25% (IPE) −14% (PIR), −8% (+24), and −3% (+48) for SJ (jump height); −17% (IPE), −12% (PIR), −6% (+24), and −3% (+48) for SJ (peak power); −24% (IPE), −15% (PIR), −6% (+24), and −7% (+48) for CMJ (jump height); and −15% (IPE), −11% (PIR), −4% (+24), and −3% (+48) for CMJ (peak power).

Pain and soreness were significantly elevated at all postexercise time points (Fig. 4). The degree of soreness, however, was significantly reduced after recovery with floatation-REST. The greatest treatment difference was observed directly after the intervention ($P = 0.002$, ES = 1.3), with intensity of postexercise soreness rated 36% lower than control. Although there were no treatment differences in pain intensity, the sensory subscale from the Short-Form McGill Pain Questionnaire 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

TABLE 1. Metabolic, sympathetic, and adrenergic responses to AHREP and acute recovery.

Biomarker		PRE	IPE	P1R	+24	+48
Lactate (nmol·L^{-1})	Control	0.97 (0.13)^{A}	12.40 (1.62)^{B}	1.54 (0.34)^{C}		
	Float	1.02 (0.20)^{A}	13.22 (1.67)^{B}	1.55 (0.30)^{C}		
Epinephrine (nmol·L^{-1})	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}
	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 (nmol·L^{-1})	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}
	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 (nmol·L^{-1})	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}
	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 (nmol·L^{-1})	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}
	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). Simple effects for time are also provided for each recovery condition; values not sharing a common letter were significantly different ($P \leq 0.05$).
*Significant treatment–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 ($P \leq 0.05$).

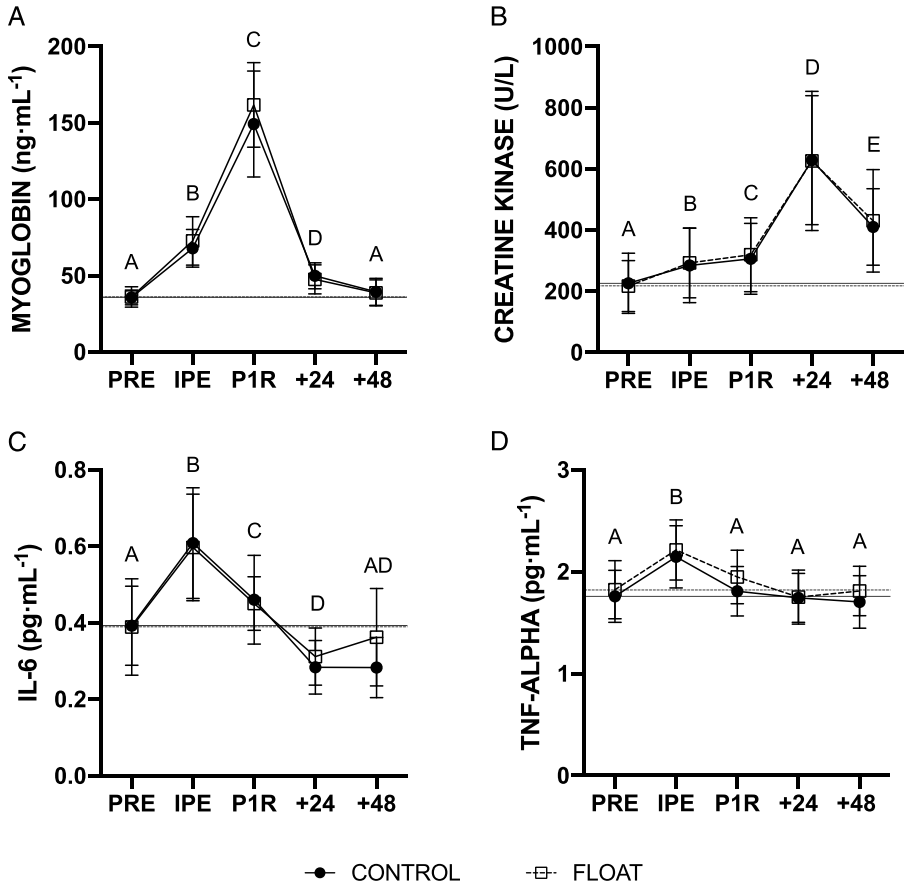

FIGURE 3—Changes in biomarkers of structural damage and inflammation after the AHREP and acute recovery intervention. Data represent the main effect for time and are presented as mean ± 95% CI. Values not sharing a common letter were significantly different ($P \leq 0.05$).

after the control treatment compared with 45% after floatation-REST. Other noticeable differences were the appearance of more severe pain characteristics (e.g., splitting, shooting, stabbing) at +24 after the control treatment despite similar treatment profiles between the recovery conditions at +48.

Psychometric data from the Positive and Negative Affect Schedule and the Profile of Mood States 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 SD). Although it was anticipated that PRE and IPE values would be similar between testing blocks, participants seemed to respond less favorably after exercise in the floatation-REST testing block. After the AHREP, participants experienced increased negative affect ($P = 0.003$, ES = 1.26), with trends toward 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 after recovery (P1R, +24, +48). To

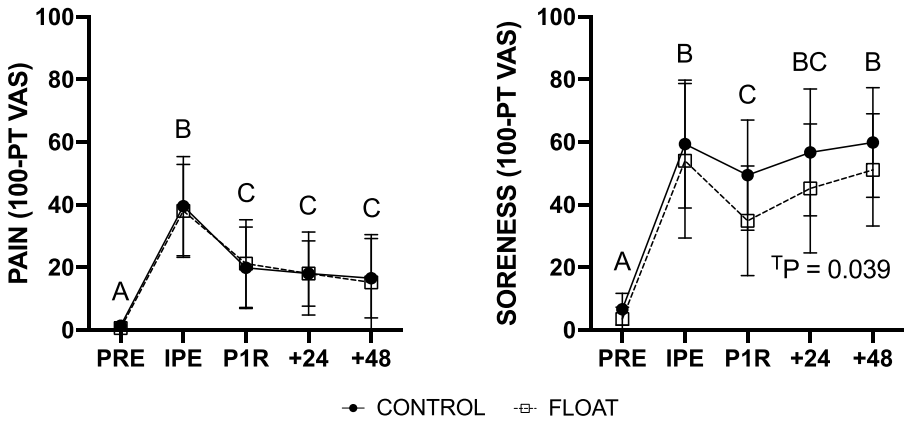

FIGURE 4—Change in visual analog scale pain and soreness scores. Data are presented as means ±95% CI. Values not sharing a common letter represent differences across time ($P < 0.05$). ^{T}P indicates a significant treatment effect.

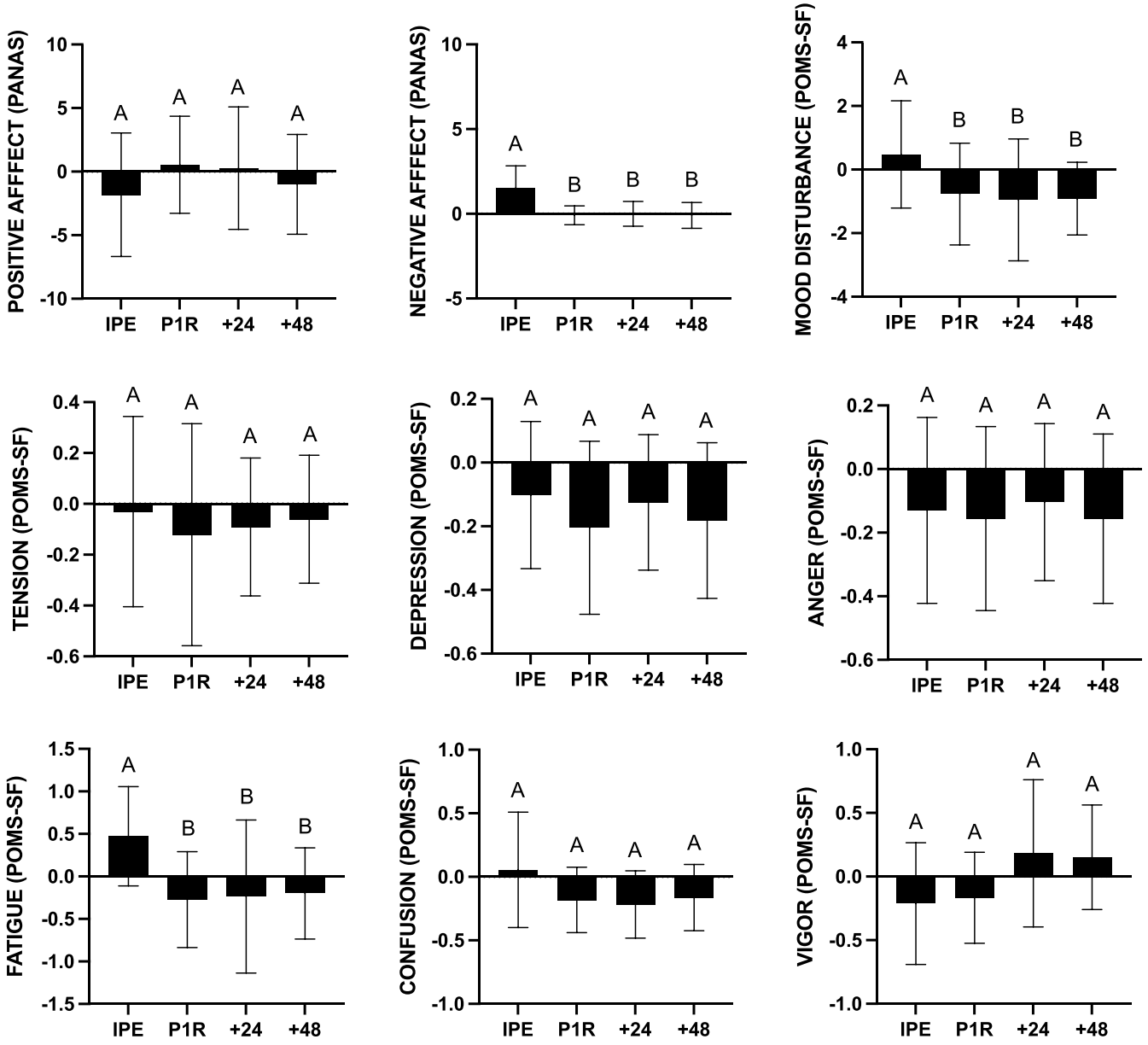

FIGURE 5—Treatment differences in affect, mood, and fatigue. Data represent treatment change scores (float − control) for each PRE-adjusted time point. Data are presented as means ±95% CI. Values not sharing a common letter were significantly different ($P \leq 0.05$).

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 1-h 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 after 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 with the sensory-stimulating control. These data suggest a positive impact of floatation-REST in the first 48 h of exercise recovery.

Recovery depends on the nature of the stressor and the physiological systems that 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 after recovery with both floatation-REST and control. The lack of treatment effect was surprising considering that 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 after 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). Although the participants in the current study demonstrated healthy patterns of HPA arousal and subsequent inhibition after 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).

Although the tropic signaling of the HPA axis demonstrated potent negative feedback mechanisms that may limit the effect of floatation-REST on this system immediately after exercise, sympathetic nervous system stress seemed to be more sensitive to the reduced sensory environment. In the current study, floatation-REST significantly reduced sympathetic–adrenal–medullary activity after exercise. Although plasma epinephrine and norepinephrine were drastically elevated during the resistance exercise protocol, both catecholamines returned to preexercise concentrations after the 1-h floatation-REST session. In contrast, norepinephrine remained significantly elevated immediately after 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 nongenomic 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 with control. The differential response pattern may have important implications for muscle repair and remodeling as the steroid hormone has both anabolic and anticatabolic effects (30). Elevations in testosterone may be attributed to reduced hepatic clearance, decreased plasma volume, or increased secretory behavior (31). Although 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 after intense exercise (e.g., postexercise hyperemia, histaminergic vasodilation) (32). Floatation-REST also promotes vasodilation; however, it is unclear whether the acute intervention provides additional hemodynamic benefit in this immediate postexercise time frame. 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 after 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 after an 8-wk 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 with an active recovery control (33). Unlike the popular cold therapies, floatation-REST provides an environment that harnesses the natural postexercise 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. Although unique temporal response patterns were observed for TNF-α and IL-6, there were no treatment differences between recovery conditions. TNF-α and IL-6 both peaked immediately after 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 and 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 1-h floatation-REST session did not negatively impact the tightly regulated proinflammatory 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-α), 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. (8) found no treatment differences in functional performance when floatation-REST was compared with a passive control condition after maximal resistance exercise. 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.).

Although 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 h 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 with nontreatment controls (41–44). Many of these investigations involved patient populations experiencing stress-related muscle tension and pain while using a more conventional floatation-REST treatment scheme of 2–3 sessions a week for up to 6 wk. 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, whereas 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 well-being 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 that 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. Although 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 and 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 before 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 after 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 postexercise 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 after floatation-REST treatment suggest meaningful modification of neuroendocrine signaling pathways, which may mediate positive recovery processes.

Because 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 because only large ES 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 seem to interfere with tissue repair and remodeling. Because 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 postexercise soreness and fatigue.

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REFERENCES

1. Meeusen R, Duclos M, Foster C, et al. Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. *Med Sci Sports Exerc*. 2013;45(1):186–205.
2. Soligard T, Schwellnus M, Alonso JM, et al. How much is too much? (Part 1) International Olympic Committee consensus statement on load in sport and risk of injury. *Br J Sports Med*. 2016;50(17): 1030–41.
3. Dupuy O, Douzi W, Theurot D, Bosquet L, Dugue B. An evidence-based approach for choosing post-exercise recovery techniques to reduce markers of muscle damage, soreness, fatigue, and inflammation: a systematic review with meta-analysis. *Front Physiol*. 2018;9:403.
4. van Dierendonck D, Te Nijenhuis J. Flotation restricted environmental stimulation therapy (REST) as a stress-management tool: a meta-analysis. *Psychol Health*. 2005;20(3):405–12.
5. Lee AB, Hewitt J. Using visual imagery in a flotation tank to improve gymnastics performance and reduce physical symptoms. *Int J Sp Psy*. 1987;18:223–30.
6. Suedfeld P, Bruno T. Flotation REST and imagery in the improvement of athletic performance. *J Sport Exerc Psychol*. 1990;12(1):82–5.
7. Wagaman JD. Flotation REST and imagery in the improvement of collegiate basketball performance. *Percept Mot Skills*. 1991;72(1): 119–22.
8. Morgan PM, Salacinski AJ, Stults-Kolehmainen MA. The acute effects of flotation restricted environmental stimulation technique on recovery from maximal eccentric exercise. *J Strength Cond Res*. 2013;27(12):3467–74.
9. Ihalainen JK, Ahtiainen JP, Walker S, et al. Resistance training status modifies inflammatory response to explosive and hypertrophic resistance exercise bouts. *J Physiol Biochem*. 2017;73(4):595–604.
10. Baldwin J, Snow RJ, Febbraio MA. Effect of training status and relative exercise intensity on physiological responses in men. *Med Sci Sports Exerc*. 2000;32(9):1648–54.
11. Spiering BA, Kraemer WJ, Anderson JM, et al. Resistance exercise biology: manipulation of resistance exercise programme variables determines the responses of cellular and molecular signalling pathways. *Sports Med*. 2008;38(7):527–40.
12. Earp JE, Hatfield DL, Sherman A, Lee EC, Kraemer WJ. Cold-water immersion blunts and delays increases in circulating testosterone and cytokines post-resistance exercise. *Eur J Appl Physiol*. 2019;119(8): 1901–7.
13. Levers K, Dalton R, Galvan E, et al. Effects of powdered Montmorency tart cherry supplementation on an acute bout of intense lower body strength exercise in resistance trained males. *J Int Soc Sports Nutr*. 2015;12:41.
14. Zajac A, Chalimoniuk M, Maszczyk A, Golas A, Lngfort J. Central and peripheral fatigue during resistance exercise—a critical review. *J Hum Kinet*. 2015;49:159–69.
15. Allen DG, Lamb GD, Westerblad H. Skeletal muscle fatigue: cellular mechanisms. *Physiol Rev*. 2008;88(1):287–332.
16. Gibala MJ, Interisano SA, Tarnopolsky MA, et al. Myofibrillar disruption following acute concentric and eccentric resistance exercise in strength-trained men. *Can J Physiol Pharmacol*. 2000;78(8): 656–61.
17. Paulsen G, Mikkelsen UR, Raastad T, Peake JM. Leucocytes, cytokines and satellite cells: what role do they play in muscle damage and regeneration following eccentric exercise? *Exerc Immunol Rev*. 2012;18:42–97.
18. Kraemer WJ, Ratamess NA, Fry AC, French DN. Strength training: development and evaluation of methodology. In: Maud PJ, Foster C, editors. *Physiological Assessment of Human Fitness*. Champaign (IL): Human Kinetics Publishers; 2006. pp. 119–50.
19. Noble BJ, Borg GA, Jacobs I, Ceci R, Kaiser P. A category-ratio perceived exertion scale: relationship to blood and muscle lactates and heart rate. *Med Sci Sports Exerc*. 1983;15(6):523–8.
20. Feinstein JS, Khalsa SS, Yeh H, et al. The elicitation of relaxation and interoceptive awareness using floatation therapy in individuals with high anxiety sensitivity. *Biol Psychiatry Cogn Neurosci Neuroimaging*. 2018;3(6):555–62.
21. Melzack R. The Short-Form McGill Pain Questionnaire. *Pain*. 1987; 30(2):191–7.
22. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: the PANAS scales. *J Pers Soc Psychol*. 1988;54(6):1063–70.
23. Shacham S. A shortened version of the Profile of Mood States. *J Pers Assess*. 1983;47(3):305–6.
24. Cohen P, Cohen J, Aiken LS, West SG. The problem of units and the circumstance for POMP. *Multivariate Behav Res*. 1999;34(3):315–46.
25. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. Hillsdale (NJ): L. Erlbaum Associates; 1988.
26. Turner JW Jr, Fine T, Ewy G, Sershon P, Freundlich T. The presence or absence of light during flotation restricted environmental stimulation: effects on plasma cortisol, blood pressure, and mood. *Biofeedback Self Regul*. 1989;14(4):291–300.
27. Turner JW Jr, Fine TH. Restricting environmental stimulation influences levels and variability of plasma cortisol. *J Appl Physiol (1985)*. 1991;70(5):2010–3.
28. Zschucke E, Renneberg B, Dimeo F, Wustenberg T, Strohle A. The stress-buffering effect of acute exercise: evidence for HPA axis negative feedback. *Psychoneuroendocrinology*. 2015;51:414–25.
29. Angeli A, Minetto M, Dovio A, Paccotti P. The overtraining syndrome in athletes: a stress-related disorder. *J Endocrinol Invest*. 2004;27(6):603–12.
30. Kraemer WJ, Ratamess NA, Nindl BC. Recovery responses of testosterone, growth hormone, and IGF-1 after resistance exercise. *J Appl Physiol (1985)*. 2017;122(3):549–58.
31. Kraemer WJ, Ratamess NA. Hormonal responses and adaptations to resistance exercise and training. *Sports Med*. 2005;35(4):339–61.
32. Romero SA, Minson CT, Halliwill JR. The cardiovascular system after exercise. *J Appl Physiol (1985)*. 2017;122(4):925–32.
33. Roberts LA, Raastad T, Markworth JF, et al. Post-exercise cold water immersion attenuates acute anabolic signalling and long-term adaptations in muscle to strength training. *J Physiol*. 2015;593(18):4285–301.
34. Townsend JR, Hoffman JR, Fragala MS, et al. TNF-alpha and TNFR1 responses to recovery therapies following acute resistance exercise. *Front Physiol*. 2015;6:48.
35. Wilson LJ, Dimitriou L, Hills FA, Gondek MB, Cockburn E. Whole body cryotherapy, cold water immersion, or a placebo following resistance exercise: a case of mind over matter? *Eur J Appl Physiol*. 2019;119(1):135–47.
36. Keller C, Steensberg A, Hansen AK, Fischer CP, Plomgaard P, Pedersen BK. Effect of exercise, training, and glycogen availability on IL-6 receptor expression in human skeletal muscle. *J Appl Physiol (1985)*. 2005;99(6):2075–9.
37. Fischer CP, Berntsen A, Perstrup LB, Eskildsen P, Pedersen BK. Plasma levels of interleukin-6 and C-reactive protein are associated with physical inactivity independent of obesity. *Scand J Med Sci Sports*. 2007;17(5):580–7.
38. Peake JM, Neubauer O, Della Gatta PA, Nosaka K. Muscle damage and inflammation during recovery from exercise. *J Appl Physiol (1985)*. 2017;122(3):559–70.
39. Chazaud B. Inflammation during skeletal muscle regeneration and tissue remodeling: application to exercise-induced muscle damage management. *Immunol Cell Biol*. 2016;94(2):140–5.
40. Broderick V, Uiga L, Driller M. Flotation-restricted environmental stimulation therapy improves sleep and performance recovery in athletes. *Perform Enhanc Health*. 2019;7(1–2):100149.
41. Bood SÅ, Sundequist U, Kjellgren A, et al. Eliciting the relaxation response with the help of flotation-rest (restricted environmental stimulation technique) in patients with stress-related ailments. *Int J Stress Manage*. 2006;13(2):154–75.

42. Kjellgren A, Westman J. Beneficial effects of treatment with sensory isolation in flotation-tank as a preventive health-care intervention—a randomized controlled pilot trial. *BMC Complement Altern Med*. 2014;14:417.
43. Kjellgren A, Sundequist U, Norlander T, Archer T. Effects of flotation-REST on muscle tension pain. *Pain Res Manag*. 2001;6(4):181–9.
44. Landstrom A, Bood SÃ, Kjellgren A, Norlander T. Treating stress-related pain in a clinical sample with flotation-REST: a further report on improvements in pain assessed by the pain area inventory (PAI). *Soc Behav Pers*. 2007;35(9):1279–80.
45. Bood SÅ, Sundequist U, Kjellgren A, Nordstram G, Norlander T. Effects of flotation REST (restricted environmental stimulation technique) on stress related muscle pain: are 33 flotation sessions more effective than 12 session? *Soc Behav Pers*. 2007;35(2):143–56.
46. Bood SÅ, Sundequist U, Kjellgren A, Nordstrom G, Norlander T. Effects of flotation-restricted environmental stimulation technique on stress-related muscle pain: what makes the difference in therapy—attention-placebo or the relaxation response? *Pain Res Manag*. 2005;10(4):201–9.
47. Al Zoubi O, Misaki M, Bodurka J, et al. Taking the body off the mind: decreased functional connectivity between somatomotor and default-mode networks following floatation-REST. *Hum Brain Mapp*. 2021;42(10):3216–27.
48. Suedfeld P, Ballard EJ, Murphy M. Water immersion and flotation: from stress experiment to stress treatment. *J Environ Psychol*. 1983;3(2):147–55.
49. Feinstein JS, Khalsa SS, Yeh HW, et al. Examining the short-term anxiolytic and antidepressant effect of floatation-REST. *PLoS One*. 2018;13(2):e0190292.
50. McAleney PJ, Barabasz A, Barabasz M. Effects of flotation restricted environmental stimulation on intercollegiate tennis performance. *Percept Mot Skills*. 1990;71(3):1023–8.