Recovery of Voluntary and Evoked Muscle Performance Following Intermittent-Sprint Exercise in the Heat

Rob Duffield, Monique King, and Melissa Skein

Purpose: This study investigated the effects of hot conditions on the acute recovery of voluntary and evoked muscle performance and physiological responses following intermittent exercise. Methods: Seven youth male and six female team-sport athletes performed two sessions separated by 7 d, involving a 30-min exercise protocol and 60-min passive recovery in either 22°C or 33°C and 40% relative humidity. The exercise protocol involved a 20-s maximal sprint every 5 min, separated by constant-intensity exercise at 100 W on a cycle ergometer. Maximal voluntary contraction (MVC) and a resting evoked twitch (Pf) of the right knee extensors were assessed before and immediately following exercise and again 15, 30, and 60 min postexercise, and capillary blood was obtained at the same time points to measure lactate, pH, and HCO₃. During and following exercise, core temperature, heart rate and rating of perceived exertion (RPE) were also measured. Results: No differences (P = 0.73 to 0.95) in peak power during repeated sprints were present between conditions. Postexercise MVC was reduced (P < .05) in both conditions and a moderate effect size (d = 0.60) indicated a slower percentage MVC recovered by 60 min in the heat (83 ± 10 vs 74 ± 11% recovered). Both heart rate and core temperature were significantly higher (P < .05) during recovery in the heat. Capillary blood values did not differ between conditions at any time point, whereas sessional RPE was higher 60 min postexercise in the heat. Conclusions: The current data suggests that passive recovery in warm temperatures not only delays cardiovascular and thermal recovery, but may also slow the recovery of MVC and RPE.

Keywords: motivation, hyperthermia, performance, postexercise recovery

Prolonged exercise performance in hot conditions is often suppressed owing to elevations in internal body temperature.¹ This reduction in performance is proposed to be a result of either an overloading of cardiovascular and metabolic systems² or a reduction in recruitment of voluntary force either in response³ or avoidance⁴,⁵ of an increased thermal load. Further, increased core temperature (Tcore) resulting from exercise may result in a delay in the recovery of performance and physiological functioning.⁶,⁷ Despite the focus of research on the effects of

The authors are with the School of Human Movement Studies, Charles Sturt University, Bathurst, NSW, Australia.
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exercise performance in the heat, and given the importance of recovery when multiple training/competition bouts are held within close proximity, there is limited research on the effects of hot conditions on postexercise recovery. Given the extent of training and competition bouts conducted by sports in warm environments, often with limited access to cooler environments or cooling interventions, recovery in the heat is an issue that often confronts many athletes. Accordingly, the current study investigated the effects of recovery in hot and mild environmental temperatures on postexercise physiological and performance responses.

While warmer environmental conditions have been shown to improve anaerobic power in single maximal efforts when repeated efforts are required, the effects of warmer environments become ergolytic. Previous research has highlighted that both exercise-induced and passive elevations in core and local tissue temperatures reduce voluntary force production. This reduction in voluntary force is likely due to a reduction in central nervous system (CNS) drive to active musculature as a preventative mechanism to reduce the production of metabolic heat. Further, there is evidence to show that the return to optimal performance is faster when aggressive cooling interventions actively reduce Tcore. While this collection of data are either immediately postexercise or from passive heating, it highlights that the maintenance of a higher Tcore may affect ensuing exercise performance.

Even though previous research outlines that an alleviation of substantial endogenous thermal stress may assist in the return of voluntary force production and exercise performance, surprisingly there is minimal research describing the effects of hot conditions on the acute postexercise recovery. Falk et al have reported that a 60-min recovery in 35°C after the completion of a 5- × 15-s repeat-sprint protocol did not reduce power in an ensuing series of 5- × 15-s efforts, although Tcore did not exceed 38.0°C. Further, Backx et al reported no effect of recovery in 40°C on power output during repeated 30-s Wingate efforts following 60-min recovery, concluding that anaerobic power was not suppressed in the heat. In contrast, Vaile et al reported a 15-min active recovery followed by 40 min of passive recovery in 34°C suppressed the total work performed in an ensuing 15-min cycling time trial, when compared with an initial 15-min time trial. Unlike the Falk et al data, during this recovery period in warm conditions, Tcore was still elevated above 38.0°C after the recovery period. The time course of recovery of Tcore for moderate- to high-intensity exercise has been outlined as at least 60 min in duration in normothermic conditions, yet there is little corresponding muscle performance data reported. Further, previous research outlines that reductions in performance in the heat may not be of peripheral origin, as evoked twitch measures indicate no changes in peak twitch force, yet voluntary force production is reduced. Often in training or competition, repeated exercise bouts are required within close proximity, and in hot conditions a suppression of performance may result, which has led to the implementation of strategies to accelerate postexercise recovery. Finally, despite the proposed physiological explanations for improved recovery being responsible for improved performance, often the perceptual aspects of recovery or readiness to perform are also of importance. Accordingly, the role of perceived exertion is also relevant in field-based training and competition scenarios.

Despite evidence of reduced performance in hot conditions and acceptance of the importance of postexercise recovery procedures, there is minimal research...
investigating the effect of hot compared with mild environmental temperatures on exercise and recovery. Therefore the current study aimed to investigate the effects of hot environmental conditions on recovery of muscle performance (voluntary and evoked), core temperature, heart rate, and perceived exertion.

Methods

Participants

Thirteen trained club-standard, team-sport athletes including six females and seven adolescent males were recruited as participants. Female participants had a mean ± SD age of 21 ± 1.5 y, height of 164.1 ± 5.0 cm, and mass of 58.2 ± 9.4 kg, and the adolescent male participants had a mean ± SD age of 17 ± 1.5 y, height of 177.3 ± 6.2 cm, and mass of 69.9 ± 6.3 kg. All participants and, if appropriate, the guardian or parent of a participant gave verbal and written consent to participate in this study and ethics approval was granted by the Institutional Human Ethics Committee.

Overview

An initial session was performed to ensure familiarity with all testing environments, measures, and procedures. Participants performed two randomized testing sessions separated by 7 d, at the same time of day and where appropriate, within the same phase of the menstrual cycle. All female data involved both sessions being conducted within the same phase of menstrual cycle with no intake of oral contraceptives. These test sessions were identical except for the environmental temperature, which included a hot condition (H) in 33 ± 0.4°C and a control condition (C) in 22 ± 0.4°C, respectively, with standardized relative humidity (40 ± 3%). The testing sessions consisted of a 30-min high-intensity, intermittent-sprint cycling protocol followed by a 60-min immediate postexercise passive (seated) recovery period. Physiological and performance measures were recorded pre-, during, and postexercise and further repeated at 15, 30, and 60 min postexercise. The exercise protocol and all testing procedures (apart from assessment of voluntary and evoked muscle performance) were conducted in a climate chamber set to the respective testing temperatures. The assessments of voluntary and evoked muscle performance were conducted outside the climate chamber temperature at normothermic laboratory temperatures (22 ± 0.5°C). Participants were expediently moved to and from climate chamber to the testing dynamometer outside the climate chamber for ~3 to 5 min on each respective test of muscle function.

In the 24 h before all testing sessions, participants were required to avoid strenuous exercise and alcohol, while avoiding food and drink, especially caffeine, in the 3 h before testing. Participants were prehydrated 2 h before exercise with 300 mL of water and recorded all activity, food, and fluid consumption in a diary during the first testing session and replicated these activities for the remaining session. Before any testing measures or exercise protocol, participants performed a standardized warm-up of 5 min at 70 W on a cycle ergometer in 22°C (828E Monark, Stockholm, Sweden). Participants did not engage in any stretch-
ing activities to ensure evoked twitch properties were not affected by alterations to musculotendinous stiffness as a result of stretching.

Exercise Protocol

Participants performed a 30-min high-intensity, intermittent-sprint cycling protocol consisting of a maximal 20-s sprint every 5 min interspersed with an electronically braked constant-intensity exercise at 100 W (LODE Excalibur Sport, Groningen, The Netherlands). This protocol is an adaptation of previous intermittent-sprint protocols with acceptable coefficient of variation (CV = 4%, r = .97). Peak power (W) and time to peak power (s) were recorded for each respective sprint effort. Between each maximal sprint effort, participants cycled at a constant power output of 100 W, which was controlled by the electronically braked cycle ergometer and allowed self-selection of RPM. In the 30 s before each sprint, participants were required to cycle at 40 RPM to ensure a consistent starting RPM (below this value the cycle ergometer program paused). During the 60-min postexercise recovery period, participants remained seated in the climate chamber unless required for assessment of muscle function at 15, 30, and 60 min postexercise. No fluid or food was consumed during this 60-min recovery.

Maximal Voluntary Contraction and Evoked Twitch Potential Measures

Preexercise and then again immediately, 15, 30, and 60 min postexercise, maximal voluntary and evoked peak force of the right knee extensors (KE) were assessed using an isokinetic dynamometer (Kin-Com model 125, Chattanooga Group Inc, Hixon, TN) linked to a PXI data acquisition system (Model no. 1042Q, National Instruments, Austin, TX). Due to logistical constraints of the size and location of the dynamometer, assessment of MVC was performed outside the climate chamber in thermoneutral temperatures (~22°C). This methodological limitation will be discussed later. Participants were seated upright with the hip flexed at 75° and the knee flexed at 90° (0° being full extension), which were determined using a goniometer and were secured to the dynamometer via waist and shoulder straps. The axis of rotation of the dynamometer was aligned with the lateral epicondyle of the femur with the lower leg attached to the lever arm 1cm above the lateral malleolus of the ankle. Seating and lower limb positions were recorded and standardized for all measures for each respective participant for all sessions. Evoked muscle activation was achieved by percutaneous stimulation using two 90- × 50-mm adhesive gel electrodes positioned on the anterior surface of the right thigh; one 4 cm above the superior border of the patella and the other 1 cm below the inguinal fold. The current applied was delivered by a Digitimer DS7AH stimulator (Digitimer Ltd, Welwyn Garden City, Hertfordshire, England) using a single square-wave pulse with a width of 200 μs (400 V with a current of 150 to 800 mA), driven by a custom designed instrument using Labview v8.0 software (National Instruments, Austin, TX). Initially, the current was manually applied in incremental steps until peak twitch force was attained. Following this, stimulus intensity was increased by a further 25% to ensure supramaximal stimulation was achieved. Evoked twitch testing was performed before
voluntary performance testing and involved six pulses separated by 10 s delivered to the nerve with the subject at complete rest. This was determined based on the absence of any load placed on the force transducer other than that due to the effect of gravity on the lower leg. For analysis, force data were exported into spreadsheet software and corrected for the effect of gravity on the lower leg offline. Following this, twitch force-time curves were averaged over all evoked contractions with the mean used to determine the peak twitch force (Pf) defined as the highest isometric force value achieved during the evoked contraction.24

Immediately following evoked twitch testing, participants completed a series of isometric MVC efforts of the right KE. Isometric assessments were performed using the same assessment apparatus and participant position as previously described for evoked twitch testing. The angle of isometric assessment was assessed at 90° from full extension and was standardized for all sessions for each respective participant. Testing consisted of a minimum of four trials where participants were instructed to exert maximal effort throughout each contraction. A minimum rest period of 30 s separated each trial, and testing continued until the final three trials had values within approximately ±5% of each other and reported data are the mean of the three highest trials. Strong verbal encouragement was provided during all voluntary tests and participants received continuous visual feedback of performance from a graphic display on a computer monitor.

**Physiological and Perceptual Measures**

Four hours before each testing session, participants ingested a telemetric temperature capsule (VitalSense, MiniMitter, Oregon, USA) to ensure movement of the capsule past the stomach into the GI tract by the commencement of testing. This thermosensitive capsule transmitted internal core temperature to an associated hand-held receiver (VitalSense, MiniMitter, Oregon, USA). Tcore and heart rate (RS200, Polar Electo Oy, Kempele, Finland) were measured before; every 5 min during (before each respective sprint); and immediately, 15, 30, and 60 min postexercise. A 100-μL sample of capillary blood was obtained from a hyperemic earlobe to measure anaerobic metabolic markers of lactate [La−], pH, and bicarbonate (HCO3) (ABL825, Radiometer, Copenhagen, Denmark) preexercise and immediately, 15, 30, and 60 min postexercise. Nude mass was measured on a set of calibrated weigh scales (HW150K, A & D, Tokyo, Japan) preexercise and following the 60-min recovery period to estimate changes in fluid loss. A measure of the rate of perceived exertion (RPE) was obtained based on the CR-10 Borg scale, with participants rating how hard the exercise felt from a scale of 0 (Nothing at all) to 10 (Maximal) during exercise and a sessional rating 60 min postexercise.25

**Statistical Analysis**

Data are reported as mean ± standard deviation (SD). A two-way repeated-measures ANOVA was used to determine significant differences between the two conditions (H vs. C) with significance set at P < .05. Effect size (ES) was calculated (Cohen’s d) to determine the magnitude of effect of exercise and recovery in the heat. An ES of <0.2 is classified as a “trivial,” 0.2 to 0.4 as a “small,” 0.5 to 0.7 as
a “moderate,” and >0.8 as a “large” effect. As there were no significant differences ($P < .05$) between male and female conditions, data are reported as combined gender groupings for subsequent results and analyses.

## Results

### Exercise Performance

Peak power on repeated 20-s sprint efforts did not differ ($P = .73$ to 0.95) and showed trivial ES ($d < 0.2$) between respective conditions (Figure 1). Further, time to peak power was not significantly different between conditions ($P = .40$) and displayed trivial ES ($d < 0.2$) ($7.9 \pm 1.5$ vs. $7.8 \pm 1.5$ s for C and H conditions, respectively).

### Voluntary and Evoked Twitch Performance

MVC in both conditions were significantly reduced immediately ($P = .02$) and until 60 min ($P = .04$) postexercise when compared with respective preexercise measures. However, whereas postexercise MVC data for H and C conditions were not significantly different between conditions ($P = .25$ to 0.65), there was a moderate effect ($d = 0.60$) for a 9% slower recovery of MVC in the H condition 60 min postexercise ($74 \pm 11$ vs. $83 \pm 10\%$ for H and C, respectively; Figure 2). Similar to MVC results, evoked Pf was significantly ($P = .01$) reduced postexercise in both conditions. However, there were no significant differences ($P = .75$ to 0.95) and trivial ES ($d < 0.1$) between H and C conditions during the recovery period (Figure 2). There were no significant condition $\times$ time interactions for either MVC ($P = .45$) or Pf ($P = .78$).

### Physiological and Perceptual Variables

The decrease in nude mass was significantly ($P = .03$) higher in the H condition ($0.82 \pm 0.24$ vs. $0.53 \pm 0.21$ kg for H and C conditions, respectively). Both $T_{core}$ and heart rate respectively showed a condition $\times$ time interaction; however, significant differences were apparent predominantly during the recovery phase ($P = .01$ to 0.05). $T_{core}$ was significantly increased postexercise in both conditions ($P = .02$) and, although there were no differences between conditions during the exercise protocol ($P = .15$ to 0.25), $T_{core}$ was significantly higher ($P = 0.01$ to 0.03) in the H condition postexercise at all times during the recovery (Figure 3). Similarly, heart rate was also significantly increased postexercise ($P = 0.02$) in both conditions and was also significantly higher ($P = .03$) in the H condition in the 60-min recovery period (Figure 3). Capillary blood measures of $[\text{La}^-]$, pH, and $\text{HCO}_3$ (Table 1) were significantly different in pre- to immediately postexercise in all conditions ($P = .01$ to 0.05), but had returned to baseline values by 60 min. Further, there were no differences ($P = 0.44$ to 0.88) between respective conditions and trivial ES ($d < 0.2$) for all blood variables. Finally, RPE values did not differ significantly ($P = .08$ to 0.42), but did display large ES ($d > 0.9$) between conditions during exercise and were significantly higher in the 60-min postexercise sessional rating in the H condition ($P = .03$; Figure 4).
Figure 1 — Mean ± SD peak power (W) for respective sprint performances during hot (H) and control (C) trials. There were no significant differences between trials ($P > 0.05$).
The current data suggests that passive recovery in the heat (33°C) with moderate elevations in $T_{\text{core}}$ (38.5°C) had no effect on Pf but may have had a moderate effect of slowing the recovery of voluntary force, in addition to elevating recovery measures of $T_{\text{core}}$ and heart rate. The 30-min exercise protocol resulted in a moderate increase in peak $T_{\text{core}}$, yet neither condition surpassed 39.0°C. The external conditions did not result in any difference between H and C conditions in exercise performance, which has been reported previously for similar types of exercise.22,26 Previous research has highlighted that power output during single maximal efforts

**Figure 2** — Mean ± SD (A) maximal voluntary contraction (MVC) (N) and (B) peak evoked twitch force (Pf) (N) pre- and postexercise and during the 60-min recovery (R) period in hot (H) and control (C) conditions. #Represents a moderate effect size between conditions for recovery from preexercise.

**Discussion**

The current data suggests that passive recovery in the heat (33°C) with moderate elevations in $T_{\text{core}}$ (38.5°C) had no effect on Pf but may have had a moderate effect of slowing the recovery of voluntary force, in addition to elevating recovery measures of $T_{\text{core}}$ and heart rate. The 30-min exercise protocol resulted in a moderate increase in peak $T_{\text{core}}$, yet neither condition surpassed 39.0°C. The external conditions did not result in any difference between H and C conditions in exercise performance, which has been reported previously for similar types of exercise.22,26 Previous research has highlighted that power output during single maximal efforts
may be increased in warmer environmental conditions; however, repeated efforts may result in no difference or a greater reduction in performance in the heat.\textsuperscript{10,17} As expected, the warmer environmental temperature did result in an increase in heart rate and postexercise $T_{\text{core}}$, likely as a result of the combined effects of peripheral vasodilation to ensure adequate evaporative heat loss and maintenance of muscle blood flow.\textsuperscript{2} Despite similarities in exercise performance between conditions, $T_{\text{core}}$, heart rate, and sessional RPE were elevated postexercise and during the recovery period in the heat. Moreover, passive recovery in the heat, with an elevated $T_{\text{core}}$, heart rate, and RPE also may show a slower recovery of MVC.

Previous studies have reported associations between reductions in MVC with higher peak $T_{\text{core}}$, generally above 39.0°C.\textsuperscript{11,12} Further, Saboisky et al.\textsuperscript{27} have also reported selective reductions in postexercise MVC in exercised as opposed to nonexercised muscle groups with $T_{\text{core}}$ also above 39.0°C. Moreover, Todd et al.\textsuperscript{28} have shown a reduction in sustained MVC in passive hyperthermia that was not

**Figure 3** — Mean ± SD (A) Core temperature ($^\circ$C) and (B) heart rate (b·min$^{-1}$) during a 30-min intermittent-sprint exercise protocol and 60-min postexercise recovery (R) in hot (H) and control (C) conditions. *Represents significant difference between conditions ($P < 0.05$).
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explained by decreased motor cortex excitability. Collectively, these data indicate the reduction in voluntary force due to high internal temperatures, despite minimal change in contractile function, and thus highlight the role of a selective reduction in central activation of heat-generating musculature.3,5 Despite these findings, to date the research has entailed either nonexercise induced (passive) heating or has not followed the postexercise time course of recovery of voluntary force production. Somewhat in contrast to the collection of previously mentioned studies, Falk et al17 reported that peak power on repeated 5-s efforts was not altered after a 60-min recovery in 34°C. However, unlike the collection of studies where T_{core} was in excess of 39°C and postexercise performance was reduced,7,11,12,28 Falk et al17 reported T_{core} below 38.0°C. The effects of hot conditions in the current study on performance recovery indicated a moderate effect and were not as explicit as those reported by Martin et al11 and Thomas et al,12 yet were more evident than cycle ergometer peak power measures reported by Falk et al.17 This may be explained by the peak T_{core} observed in the current study, which was of moderate strain at 38.5°C, and is between the respective sets of data outlined previously; and this may be a reason as to why only moderate effect of a slower recovery of MVC was present. Accordingly, it may be that a higher T_{core} may exacerbate these differences.11,12,28 Collectively, these data may further indicate that the reduction in voluntary force when exercising or recovering in the heat is dependent upon the extent of the rise in T_{core}.29,30

Despite a similar rate of recovery in Pf between conditions, a moderate effect for a slower recovery of MVC was observed. Given the similarity of exercise workload and anaerobic metabolic markers between conditions, it is evident the exercise bout and ensuing metabolic acidosis of the different environments had limited influence on the altered rate of force recovery. In addition, the similarity in Pf between conditions further highlights the limited disparity in contractile

Table 1  Mean ± SD capillary blood lactate [La−] (mmol·L−1), pH (AU), and bicarbonate (HCO3) (mmol·L−1) pre- and postexercise and during the 60-min recovery (R) period for hot (H) and control (C) conditions

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre</th>
<th>Post</th>
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<th>R30</th>
<th>R60</th>
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<td>[La−]</td>
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<tr>
<td>H</td>
<td>1.1 ± 0.4</td>
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</tr>
<tr>
<td>C</td>
<td>1.0 ± 0.3</td>
<td>12.8 ± 2.7*</td>
<td>6.4 ± 2.2*</td>
<td>3.7 ± 1.4*</td>
<td>2.0 ± 1.4</td>
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<td>pH</td>
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<tr>
<td>H</td>
<td>7.42 ± 0.01</td>
<td>7.25 ± 0.04*</td>
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<tr>
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<td>HCO3</td>
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<tr>
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<td>19.5 ± 2.7*</td>
<td>21.6 ± 2.2*</td>
<td>22.9 ± 1.2</td>
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Note: No significant differences (P > .05) and trivial ES (d < 0.2) between conditions.

*Represents significantly different (P < .05) to Pre measures within each condition.
Figure 4 — Mean ± SD rate of perceived exertion (RPE) during a 30-min intermittent-sprint exercise protocol and 60-min postexercise recovery (R) in hot (H) and control (C) conditions. *Represents significant difference between conditions (P < .05). #Represents large effect size (d > 0.8).
function due to recovery in the heat. The moderate effect for a slowing of the rate of recovery in MVC is possibly due to a higher $T_{core}$ and/or RPE respectively, suggesting that the act of recovery in the heat may limit external motivation to perform. Previous research has highlighted an increase in perception of fatigue with increased $T_{core}$, and resultant reduction in performance in the heat. However, Morrison et al and Todd et al have, respectively, reported the suppression of voluntary activation in the heat independent of perceptual fatigue ratings, concluding elevated $T_{core}$ reduces neuromuscular function as opposed to merely a reduction in motivation owing to a higher perception of effort. However, recently Racinais et al have reported that the reduction in voluntary activation with passive heating occurs at a supraspinal level as well as a reduction in neuromuscular junction transmission. Further, St. Clair Gibson et al highlight that RPE is not linearly correlated with physical or physiological factors but is instead more closely related to the present psychological state of the athlete, in conjunction with previous experiences in similar settings. Alternatively, Tucker et al reported that exercise in the heat is adjusted based on subjective RPE from afferent feedback regarding heat storage rate. In the current study, $T_{core}$ was not above 38.5°C and a moderate effect for a slower recovery of MVC seemed evident, independent of contractile function. Whether this effect was due to reduced motivation based on perception of environmental conditions or a reduction in supraspinal activation is unknown. However, methodological limitations must be considered in that because of the size and location of the dynamometer, measurements of MVC and Pf were performed in thermoneutral environments outside the climate chamber. Accordingly, the time (4 to 5 min) to move and record measures from participants represents a substantial period of the early recovery phase spent in 22°C for both conditions. While this constraint was unavoidable, this mini-cooling exposure during the hot condition may have resulted in transient changes in core or skin temperatures. While it is unclear as to whether this may have influenced the findings, $T_{core}$ was not unduly altered by the next measurement time point. In addition, it can be argued that MVC and Pf measures may also be temperature dependent and should be measured in comparable environmental temperatures to avoid the effect of temperature-related factors on tendon compliance.

Lower postexercise heart rate and $T_{core}$ responses were evident in the mild environmental temperatures, even though the rate of recovery was similar. Halson et al and Vaile et al have, respectively, reported that cold-water immersion reduces the cardiovascular and thermal load when recovering in the heat. Accordingly, it is possible that the reduced cardiovascular load contributed to the small difference in recovery of MVC in mild temperatures. However, previous research has observed reduced voluntary activation following passive heating, independent of cardiovascular or physiological strain, the differences in cardiovascular load may not be related to the recovery of MVC in the heat. Although it is possible that an increased perception of cardiac load could also contribute to higher sensations of postexercise exertion. Thus, in the same way that an elevated $T_{core}$ is proposed to reduce motivation for volitional force production, the elevated heart rate response could also decrease perception of recovery and reduce motivation to perform. However, the relationship between RPE and heart rate is not causal, as unchanged RPE values have been reported for increased heart rate responses induced by beta blocker use during cycling exercise.
In summary, performing exercise and recovery in warm-to-hot environmental conditions may act to slow the recovery of voluntary force when measured in thermoneutral conditions. A slightly slower recovery of MVC in the current context is likely to be from a motivational origin, as Pf did not differ between conditions and no change in contractile properties were evident. In addition, sessional RPE was significantly higher postexercise in the heat and may indicate the increased perceptual strain and/or a reduction in motivation to perform. Further, hot conditions also slowed the recovery of T\textsubscript{core} and heart rate, with minimal effect on blood-based variables of anaerobic metabolism, further highlighting likely motivational rather than metabolic mechanisms. Therefore, these data indicate that even moderately higher T\textsubscript{core} in hot environments during recovery may slow the recovery of voluntary force.

**Practical Applications**

Warm environmental conditions that result in a greater increase and slower recovery of core temperature may slow the recovery of voluntary force production. This slower recovery may be a result of a reduced motivation for voluntary force production owing to higher core temperature and/or increased perceived exertion. Accordingly, the avoidance of recovery in the heat or the use cooling interventions to aggressively reduce cardiovascular, thermal, and perceived stress following exercise in the heat may allow for a faster acute recovery of performance for ensuing bouts of exercise. This may particularly be the case when the environmental conditions are extreme or the increase in core temperature is great, particularly over 39.0°C.

**References**