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ORIGINAL ARTICLE

Does a bout of strength training affect 2,000 m rowing ergometer performance and rowing-specific maximal power 24 h later?

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Abstract Rowers regularly undertake rowing training within 24 h of performing bouts of strength training; however, the effect of this practice has not been investigated. This study evaluated the impact of a bout of high-intensity strength training on 2,000 m rowing ergometer performance and rowing-specific maximal power. Eight highly trained male club rowers performed baseline measures of five separate, static squat jumps (SSJ) and countermovement jumps (CMJ), maximal rowing ergometer power strokes (PS) and a single 2,000 m rowing ergometer test (2,000 m). Subsequently, participants performed a high-intensity strength training session consisting of various multi-joint barbell exercises. The 2,000 m test was repeated at 24 and 48 h post-ST, in addition SSJ, CMJ and PS tests were performed at these time points and also at 2 h post-ST. Muscle soreness, serum creatine kinase (CK) and lactate dehydrogenase (LDH) were assessed pre-ST and 2, 24 and 48 h post-ST. Following the ST, there were significant elevations in muscle soreness (2 and 24 h, $P < 0.01$), CK (2, 24 and 48 h, $P < 0.01$), and LDH (2 h, $P < 0.05$) in comparison to baseline values. There were significant decrements across all time points for SSJ, CMJ and PS, which ranged between 3 and 10% ($P < 0.05$). However, 2,000 m performance and related measurements of heart rate and blood lactate were not significantly affected by ST. In summary, a bout of high-intensity strength training resulted in symptoms of muscle damage and decrements in rowing-specific maximal power, but this did not affect 2,000 m rowing ergometer performance in highly trained rowers.

Keywords Rowing · 2000 m · Strength training · Recovery · Power · Muscle damage

Introduction

Strength training is commonly practised amongst rowers (Ivey et al. 2004; McNeely et al. 2005; Gee et al. 2011). The structure of rowers' strength training commonly features the Olympic lifts, which involve explosive eccentric muscle actions (Chiu and Schilling 2005) and multi-joint strength exercises such as squats, which have been found to result in subsequent acute decrements in aspects of muscle function such as isokinetic and isometric strength, jumping ability and cycling peak power (Raastad and Hallen 2000; Byrne and Eston 2002a, b; French et al. 2008). In addition, research suggests that rowers perform strength training with a loading between 85 and 95% of their one repetition maximum (McNeely et al. 2005; Gee et al. 2011). Heavy load resistance training such as this has been shown to produce more pronounced and longer lasting decrements in parameters of muscle function, including muscle power, maximal voluntary contraction, peak torque and electrically evoked force, than moderate load resistance training (Raastad and Hallen 2000; Linnamo et al. 2005; Paschalis et al. 2005a). Despite the strenuous nature of the strength training performed by rowers, there is a lack of research investigating

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the impact of acute strength training on rowing or endurance performance in general. Scott et al. (2003) are the only authors to assess the impact of a bout of strength training, featuring free-weight barbell exercises, on subsequent endurance exercise. They found that participants reported significantly higher ratings of perceived exertion during a 30-min submaximal run performed 24–30 h after the strength training session in comparison to a baseline trial. The participants in the Scott et al. (2003) study were described as 'physically active', taking part in >3 running sessions a week. Using such participants rather than athletes, who train specifically to compete in a particular sport or event, limits the applicability of the findings obtained in relation to the athletic setting (Marcora and Bosio 2007). Various studies have assessed the impact of muscle damaging exercise challenges (commonly, a series of jumps or prolonged downhill running) on subsequent cycling or running endurance performance. This research has generally involved either assessment of physiological responses during submaximal exercise (Gleeson et al. 1995; Calbet et al. 2001; Braun and Dutto 2003; Scott et al. 2003; Paschalis et al. 2005b; Chen et al. 2007, 2008) or incremental tests to volitional exhaustion (Gleeson et al. 1998; Davies et al. 2008, 2009). However, the use of these endurance protocols has been questioned on the basis that they possess low ecological validity since the featured protocols do not simulate or model the demands imposed throughout a typical endurance cycling or running event (Schabort et al. 1998; Atkinson and Nevill 2001). In terms of athletic performance, a more reliable and externally valid means of assessing endurance performance involves protocols in which athletes are required to complete a fixed amount of work or to cover a given distance in the shortest possible time (time trials), or to complete a maximal amount of work in a specific time period (Schabort et al. 1998; Atkinson and Nevill 2001; Hopkins et al. 2001). Marcora and Bosio (2007) and Twist and Eston (2009), reported ~4% decreases in the distance run in 30 min and the distance cycled in 5 min, respectively, following muscle damaging protocols involving plyometric jumps. Despite the exercise tests being more applicable to the athletic setting than those previously discussed, the participants in these studies were not trained endurance athletes. In light of this issue, Marcora and Bosio (2007) cautioned that their results could not confidently be applied to high-level athletes, since this population might be less susceptible to exercise-induced muscle damage due to the repeated bout effect (McHugh 2003). The repeated bout effect refers to unfamiliar muscle damaging eccentric exercise performed on more than one occasion with symptoms of muscle damage reducing over time (Nosaka et al. 2001). Authors have found this effect to occur with repeated bouts of resistance training (Nosaka et al. 2001; Lavender and Nosaka 2008), jump training (Marginson et al. 2005; Miyama and Nosaka 2007) and downhill running (Rowlands et al. 2001). A more complete understanding of the effects of acute strength training on endurance performance is important, particularly for endurance-based sports where strength training is routinely performed. This is because bouts of strength training result in subsequent decrements in sports-specific muscle function, notably power producing ability (Raastad and Hallen 2000; Byrne and Eston 2002a, b; French et al. 2008; Gee et al. 2008). Findings from a recent questionnaire (Gee et al. 2011) found that approximately 90% of rowing coaches programme rowing training on the same day or up to 36 h after strength training. This finding indicates a belief amongst coaches that rowers are able to perform high-load strength training and subsequently perform meaningful rowing training in close proximity to one another. However, we have recently observed that a strength training session, similar to that habitually performed by rowers, led to a decrease in 250 m rowing sprint performance at 24 h with accompanying symptoms of muscle damage and decreases in jump height (Gee et al. 2008). Since short duration rowing tests have been shown to correlate with 2,000 m rowing performance (r = 0.87–0.88, Ingham et al. 2002; Riechman et al. 2002), there is the potential that 2,000 m rowing performance would be negatively affected by prior strength training undertaken in close proximity. Therefore, the aim of this study was to determine the effect of a bout of high-intensity strength training on 2,000 m rowing ergometer performance and rowing-specific maximal power. We hypothesised that concurrent performance decrements would occur in 2,000 m rowing ergometer performance and rowing-specific maximal power following a bout of high-intensity strength training.

Methods

Participants

Eight club standard rowers were recruited from Tees Rowing Club (mean ± standard deviation, age: 23.6 ± 6.8 years, weight: 85.4 ± 9.8 kg, height: 1.88 ± 0.06 m, 2,000 m ergometer time: 6:35.2 ± 0:12.4 min:s). The participants had all competed at national level events such as the 'Head of the River Race', the 'Henley Royal Regatta' and the 'National Rowing Championships of Great Britain'. The participants possessed a similar 2,000 m ergometer time to those recruited by Ingham et al. (2007) (2,000 m: 6:34.5 min:s), who were described as 'club standard' rowers. To put the standard of our recruited rowers into context, Ingham et al. (2007) found eight Olympic champion rowers to have a 2,000 m time of 5.53.4 min:s. All participants had at least 1 year of experience in regularly

performing structured strength training and, prior to the study, all participants completed at least 12 weeks (two sessions a week) of supervised Olympic weightlifting-style strength training. During this >12 week period, the participants maintained their habitual rowing training and did not perform any additional strength training. They were informed of the experimental procedures and any potential risks involved and gave their written informed consent to participate in the study. The study was approved by the ethical committee of the School of Social Sciences and Law at Teesside University. The participants were asked to abstain from alcohol 24 h preceding laboratory testing sessions and strength training sessions and caffeine before arriving at the laboratory on each of the testing days. This was confirmed through self-report on the days of testing and strength training.

Experimental protocol

Throughout their involvement in the research, participants maintained their regular rowing training and avoided strength training, apart from the sessions given before the two follow-up trials (24 and 48 h). Participants were asked to abstain from exercise on the day of testing and arrive at the laboratory in a hydrated state. All participants were habituated to tests prior to the first testing session. This involved performing each of the power tests at the start of their supervised strength training sessions in the 4 weeks prior to testing. Participants were asked to abstain from strength training in the 72 h before baseline testing. On the first testing session, body mass and height were measured. Participants then completed a 5 min warm-up on a rowing ergometer, followed by five individual static squat jumps (SSJ) and five individual countermovement jumps (CMJ), interspersed with 30 s recovery between each jump. After the jumps, participants performed five maximal rowing power strokes (PS) on the rowing ergometer. The participants were then instructed to warm up for a further 5 min on the rowing ergometer, after which they performed the 2,000 m test. Heart rate was recorded every 30 s during the test. Immediately after the test was completed, participants provided a rating of perceived exertion (RPE) on how physically demanding they found the test. Capillary blood samples were taken before and at the end of the test, and at 1, 3, 5 and 7 min of recovery for the assessment of [lactate]. Participants returned to the laboratory 4–6 days after the baseline measures. A capillary blood sample was taken from the finger for assessment of creatine kinase (CK) and lactate dehydrogenase (LDH). Participants perceived soreness rating and limb girths were also assessed. The participants then performed the strength training session (ST). Two hours following the ST, capillary blood was drawn for assessment of CK and LDH, and soreness and girth measurements were repeated. In addition, at this time point the anaerobic (SSJ, CMJ, PS) power tests were again completed. Participants were then randomly assigned to perform follow-up measures at either 24 or 48 h after the ST. For follow-up measures, capillary blood was collected for assessment of baseline [lactate], CK and LDH, and perceived soreness and limb girths were also assessed. The participants then repeated the testing protocol described for the first testing session. At 4–6 days following completion of the first follow-up trial, participants repeated the ST and then performed follow-up testing after 24 or 48 h in a counterbalanced manner. The study followed a within-participant design, since the same group of participants performed all three 2,000 m trials. See Fig. 1 for a schematic diagram describing the experimental design.

Experimental test battery

Static squat jump and countermovement jump tests

The Just Jump measurement system (Just Jump, Probotics, Huntsville, AL, USA) was used for assessment of jump performance. Five independent trials of both the SSJ and CMJ were conducted with 30 s between each jump, the highest jump for each being recorded for data analysis. The participants positioned themselves in the centre of the Just Jump contact mat and were instructed to place their hands on the iliac crest. The SSJ test began from an erect standing position, from which participants were told to squat down to a position where their thighs were at a 90° angle in relation to the lower leg. Participants held this position for 3 s and then were instructed to jump vertically for maximal height. The CMJ test began from an erect standing position with participants maintaining their hands on the iliac crest. The participants squatted to their perceived optimal depth and immediately ascended to jump vertically for maximal height. The SSJ and CMJ tests have been commonly used to assess functional performance following muscle damaging exercise (Raastad and Hallen 2000; Byrne and Eston 2002a; Marginson et al. 2005; Skurvydas et al. 2006; French et al. 2008) and are regularly used to monitor power in a wide variety of sports (Bret et al. 2002; Apostolidis et al. 2004; Di Cagno et al. 2008; Requena et al. 2009).

Power strokes

Maximal stroke power was assessed with an air-braked rowing ergometer (Concept 2 Model D, Concept 2 Ltd, Wilford, Notts, UK) with a drag factor of 140. Participants initially rowed submaximally for 1 min, at which point they were instructed to perform two build up strokes, which were followed by the first of five consecutive maximal effort PS. All participants were required to hold a rate of 30

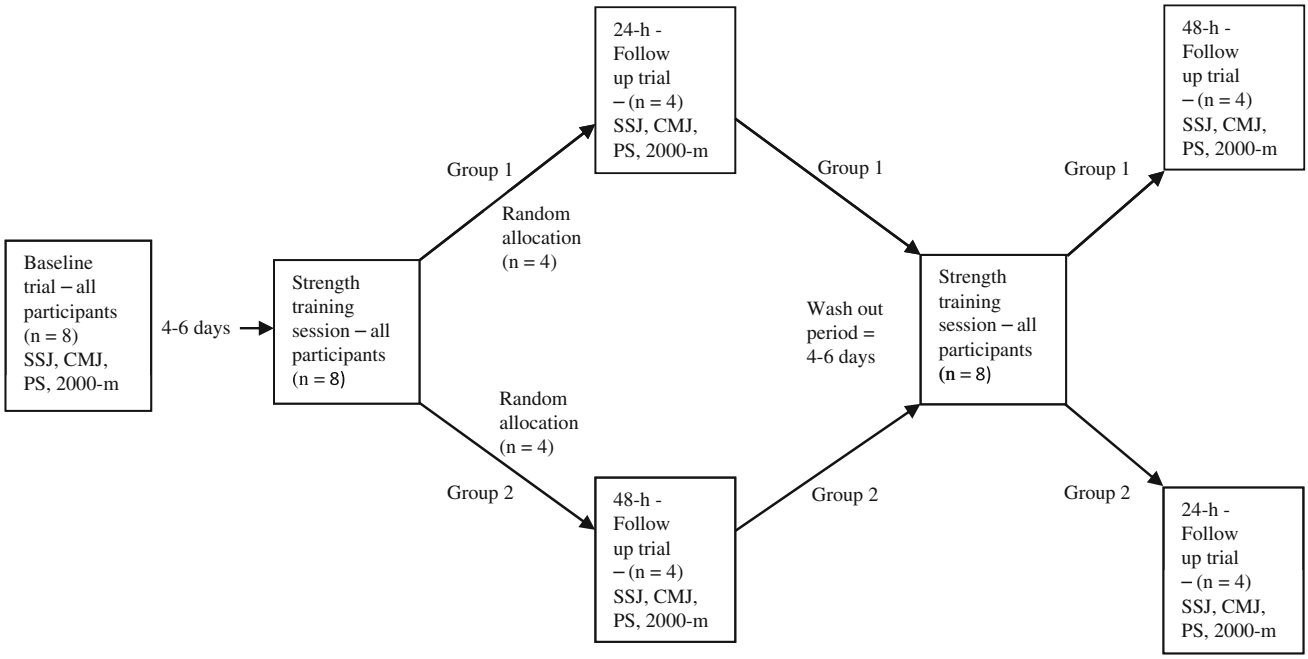

Fig. 1 Schematic diagram describing the experimental design

strokes min^{-1} during the PS, as described previously (Ingham et al. 2002).

2,000 m rowing ergometer test

The test was performed on an air-braked rowing ergometer (Concept 2 Model D, Concept 2 Ltd, Wilford, Notts, UK) with a drag factor of 140. Before the initiation of the test, participants rowed submaximally for 5 min which acted as a warm-up. During the test, participants were given feedback from the rowing ergometer screen, which displayed the distance in meters, time in minutes and seconds, 500 m split time in minutes and seconds and stroke rate per minute. This feedback was typical to that regularly experienced by the group of participants when performing rowing ergometer training and testing. Heart rate was recorded using the Polar monitoring system (Polar Electro, Kempele, Finland). Participants wore a chest strap transmitter interfaced via short range telemetry with a wrist unit, which displayed the heart rate in beats per minute. A member of the experimental team held this unit and recorded the displayed value every 30 s during the test. Participants reported their rating of perceived exertion (RPE) (6–20 scale; Borg 1970) immediately after the test was completed. Capillary blood samples for the assessment of [lactate] were drawn at the completion of the test and at 1, 3, 5 and 7 min of recovery.

Rating of perceived soreness

Rating of perceived muscle soreness was assessed via a visual analogue scale (VAS), previously used in literature (Avery et al. 2003; Spiering et al. 2007). This scale was a 10 cm long horizontal line anchored at either end with a small vertical line. Each anchored point was labelled as either 'No pain/soreness' (on the leftmost point of the scale representing a rating of zero) or 'Pain/soreness as bad as it could be' (on the rightmost point of the scale representing a rating of ten), respectively. Participants were instructed to mark their level of subjective pain using a vertical line along the continuum. The distance of the participants' mark on the scale in relation to the leftmost point of the scale was measured in centimetre and this distance represented their soreness rating.

Limb girths

Limb girth measurements were taken from the mid-thigh, mid-calf and upper arm using a standard tape measure in adherence with procedures produced by Lohman et al. (1988).

Strength training session

Ten days prior to commencement of the experimental protocol, the participants' one repetition maximum (1RM) was assessed on various strength training exercises. In preparation for the strength training session (ST), participants performed a warm-up, which involved performing exercises that mimicked those to be performed in the session with a 20 kg bar. The participants then completed the ST as described in Table 1. This session featured Olympic weightlifting exercises (the clean and the snatch) and classical strength training exercises (the squat, Romanian deadlift,

Table 1 Strength training session and mean ± standard deviation of 1-RM achieved by the participants on the exercises featured

Exercise	Sets × reps	% 1-RM/weight used	1-RM achieved (kg)
Snatch	4 × 5	85%	60 ± 5
Clean	4 × 5	85%	82.5 ± 7.5
Back squat	4 × 5	85%	110 ± 15
Romanian deadlift	3 × 8	75% of squat 1-RM	–
Bench press	3 × 5	85%	80 ± 7.5
Bench pull	3 × 5	85%	82.5 ± 5
Weighted sit-ups	3 × 15	15 kg	–

Mean ± standard deviation rounded to nearest 2.5kg increment.

bench press, bench pull and weighted sit-ups). Two minutes rest was allowed between each set. Verbal encouragement was given to the participants during the performance of the featured exercises. These exercises are performed routinely by rowers (Ivey et al. 2004; McNeely et al. 2005; Gee et al. 2011) and the participants regularly performed the featured exercises in their training. In their supervised training period before the initiation of the study, the participants generally followed a similar loading, rest period, set and repetition scheme as was featured in the ST. On a limited number of isolated occasions, participants failed to complete the final repetition of an exercise. In these cases, the barbell load was reduced by 2.5–5 kg (under the discretion of the supervising experimenter) for the next set of the exercise.

Blood analysis

Prior to collection of all blood samples, the finger of each participant was prepared using an alcohol-based mediwipe. A 25 µl capillary blood sample was collected for the assessment of blood [lactate]. The YSI 2300 STAT Plus™ (YSI Inc. Yellow Springs, OH, USA), which has detection limits between 0 and 30 mmol/L, was used for analysis. The analyser ran a self-calibration programme, which was repeated during every 15 min of use. To determine plasma CK and LDH activity, a capillary blood sample of 70 µl was collected. This sample was then centrifuged at 2,000 rpm for 8 min, and 10 µl of plasma supernatant was drawn from the capillary tube with a with a pipette. The supernatant was then dispensed onto designated test slides and the VITROS® DT60 II Chemistry System (Ortho-Clinical Diagnostics, Rochester, NY, USA), which had been calibrated prior to use, was used for analysis.

Statistical analysis

Data are presented as mean ± standard deviation, unless stated otherwise. Due to the large inter-participant variability in serum CK and LDH levels (Nosaka and Clarkson 1996, Xue and Yeung 1994), recorded values were log transformed using a spreadsheet produced by Hopkins et al. (2009) and subsequent statistical analysis was conducted on the transformed data. Absolute means for CK and LDH values are presented in "Results". For all other measures, raw data values were used for statistical analysis. Changes in assessed measures were analysed using repeated measures ANOVA tests. The alpha level for significance was set at $P < 0.05$ for all data. Assumptions of sphericity were assessed using Mauchly's test of sphericity. If a significant main effect across time was shown, then post hoc differences across trials were analysed with the use of LSD correction. Where significant differences were shown in the markers of muscle damage and performance tests, then Pearson product moment correlations were conducted on the changes in the data. Pearson product moment correlations were also performed between 2,000 m performance and SSJ, CMJ and PS at baseline, 24 and 48 h. In addition, the smallest practical effect of change was calculated for measures exhibiting significant changes, since defining the smallest practical effect allows for qualification of the probability of occurrence of a worthwhile effect (Rowlands et al. 2008). Smallest practical effect was calculated for each dependent variable from the product of 0.2 (which represents the smallest standardised (Cohen) change in mean) times the between-participant standard deviation for baseline values of all the participants. From using the smallest practical effect value, magnitude and inference of the change in each dependent variable was then analysed according to procedures developed by Hopkins (2007). From these procedures, 90% confidence intervals (CI) for the changes in dependent variables from pre- to post- intervention are calculated. In addition, practical inferences of harm or benefit caused to each dependent variable from the independent variable (intervention) were drawn using the approach identified by Batterham and Hopkins (2006). These inferences were based on percentage boundaries, which indicate the chances in percent of harm or benefit occurring to a dependent variable as a consequence of the intervention: 0–0.5% indicated most unlikely; 0.5–5% indicated very unlikely; 5–25% indicated unlikely; 25–75% indicated possibly; 75–95% indicated likely; 95–99.5% indicated very likely; and >99.5% indicated most likely (Hopkins 2007).

Results

Markers of muscle damage

There was a significant main effect across time for perceived muscle soreness ($F_{3,6} = 5.06$, $P = 0.010$). Perceived soreness rating was significantly raised above baseline at 2

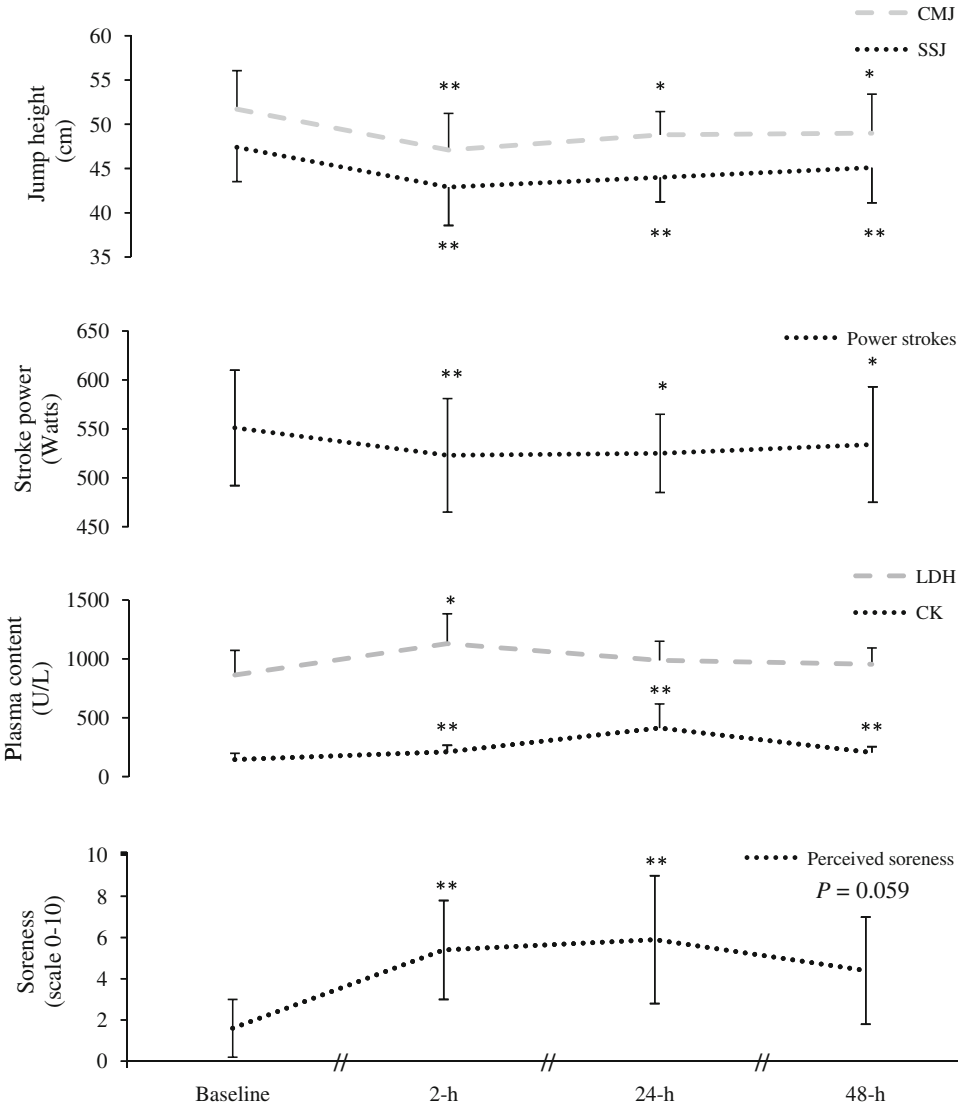

Fig. 2 The *upper panel* describes change in static squat jump and countermovement jump height following high-intensity strength training (n = 8). The *second panel* describes change in rowing stroke power following high-intensity strength training (n = 8). The *third panel* describes changes in creatine kinase (CK) and lactate dehydrogenase (LDH) activity following high-intensity strength training (CK n = 8; LDH n = 7). The *lower panel* change in soreness (scale: 0–10) following high-intensity strength training (n = 7). For all panels, **significantly higher than baseline ($P < 0.01$). *Significantly higher than baseline ($P < 0.05$)

and 24 h, while a trend for increased soreness existed at 48 h (see Fig. 2). There was a significant main effect across time for log transformed CK values ($F_{3,7}$ = 12.05, P = 0.000). Values were significantly raised above baseline (145 ± 54 U/L) at all time points (2 h: 210 ± 57 U/L, 24 h: 413 ± 205 U/L, 48 h: 205 ± 50 U/L) (see Fig. 2). Practical inferences indicated that CK levels and perceived soreness ratings were 'very' to 'most' likely to increase at all assessed time points. There was a significant main effect across time for log-transformed LDH activity ($F_{3,6}$ = 3.205, P = 0.048). A significant rise in LDH occurred at 2 h post-ST in relation to baseline (1,130 ± 253 U/L vs. 863 ± 210 U/L) with the practical inference that the ST was 'very likely' to result in raised LDH levels at this time point. There were no significant changes in limb girths at any time point with measurements at each site: arm (32.3–32.7 cm across trials), calf (36.8–37 cm) and thigh (55.3–56.1 cm) remaining consistent throughout trials.

Exercise test measures

Anaerobic power tests

There were significant main effects over time for SSJ height ($F_{3,7}$ = 11.96, P = 0.000) and CMJ height ($F_{3,7}$ = 8.83, P = 0.001). Baseline values for SSJ and CMJ were 47.4 ± 3.9 cm (90% CI: 44.8–49.9 cm) and 51.7 ± 4.4 cm (90% CI: 48.8–54.6 cm), respectively. Jump height significantly decreased at 2 h [SSJ: 42.9 ± 4.3 cm (90% CI: 40.0–45.8 cm), CMJ: 47.1 ± 4.1 cm (90% CI: 44.4–49.9 cm)], 24 h [SSJ: 44.0 ± 2.8 cm (90% CI: 42.1–45.8 cm), CMJ: 48.8 ± 2.6 cm (90% CI: 47.0–50.5 cm)] and 48 h [SSJ: 45.1 ± 4.0 cm (90% CI: 42.4–47.7 cm), CMJ: 49.0 ± 4.4 cm (90% CI: 46.1–52.0 cm)] following ST (see Fig. 2). It was inferred that decreases in SSJ and CMJ height were 'very likely' to occur at all time points. There were significant correlations between changes in CMJ and CK from

Table 2 Changes in 2,000 m rowing ergometer performance following high-intensity strength training

	Baseline	24 h	48 h
Completion time (min:s) ($n = 8$)	6:38.6 ± 11.9	6:40.8 ± 9.3	6:40.0 ± 9.1
Mean heart rate ($b.min^{-1}$) ($n = 8$)	181 ± 8	179 ± 8	181 ± 9
Peak heart rate ($b.min^{-1}$) ($n = 8$)	189 ± 8	188 ± 8	190 ± 8
Baseline blood lactate (mmol L^{-1}) ($n = 6$[a])	1.5 ± 0.3	1.9 ± 0.7	1.9 ± 0.3*
Peak blood lactate (mmol L^{-1}) ($n = 6$[a])	12.6 ± 1.7	13.2 ± 2.5	13.1 ± 3.2
Change in blood lactate (mmol L^{-1}) ($n = 6$[a])	11.2 ± 1.5	11.4 ± 2.6	11.2 ± 3.2
RPE (6–20 Scale) ($n = 8$)	19 ± 2	18 ± 2	18 ± 2

Values are expressed as mean ± standard deviation

* Significantly different from baseline ($P = 0.05$)

[a] Valid measurements could not be obtained from two of the participants

baseline to 48 h ($r = 0.66$, $P = 0.037$), and changes in SSJ and soreness from baseline to 2 h ($r = -0.68$, $P = 0.048$). There were no other significant correlations between changes in jump performance and markers of muscle damage. There was a significant main effect over time for PS to change following ST ($F_{3,7} = 3.66$, $P = 0.029$). In relation to baseline peak power output (PPO) during the PS [551 ± 59 watts (w) (90% CI: 511–590 w)] significant decreases in PPO occurred at 2 h [523 ± 58 w (90% CI: 485–562 w)], 24 h [525 ± 40 w (90% CI: 498–552 w)] and 48 h [534 ± 59 w, (90% CI: 494–574 w)]. (see Fig. 2). The practical inference was that the ST was 'very likely' harmful to stroke power at 2 h, 'likely' harmful to stroke power at 24 h, and 'possibly' harmful to stroke power at 48 h.

2,000 m rowing ergometer test

Baseline 2,000 m rowing time was 99.1% of the participants' personal best performance for the test. There were no changes in performance time for the 2,000 m rowing ergometer test across trials, with the changes in the mean between baseline and both 24 h (2.2 s) and 48 h (1.4 s) being inside the calculated smallest practical effect for the group of participants (2.4 s). Resting [lactate] was found to be significantly higher at 48 h compared to baseline ($P < 0.05$), although no differences existed in either peak or change in [lactate]. There were no other significant differences in physiological measures across trials (see Table 2). There was a significant correlation between 2,000 m time and PS power at baseline ($r = -0.81$, $P = 0.015$) and 48 h ($r = -0.77$, $P = 0.024$), but no significant correlations between 2,000 m time and jump height at any time point.

Discussion

This is the first study to investigate the impact of a bout of high-intensity strength training on event-specific performance in trained endurance athletes. The results showed that following strength training, 2,000 m rowing ergometer performance was not significantly altered despite significant decreases in rowing-specific maximal power. The increases in perceived muscle soreness, plasma CK and LDH provide evidence that muscle damage was present following the ST. The CK values were significantly raised in relation to baseline at all time points (up to 48 h) following the ST. This is a similar response to strength training bouts featuring free-weight multi-joint exercises such as squats, deadlifts and lunges (Raastad and Hallen 2000; Hoffman et al. 2010). Lactate dehydrogenase levels were also significantly increased, but only at 2 h post-ST, which is in agreement with Machado et al. (2010) who also observed raised LDH levels shortly after the completion of whole-body strength training. Perceived soreness rating was significantly elevated at 2 and 24 h following ST, and it was inferred that soreness was 'very likely' to be increased at 48 h. These findings are similar to responses following other strength training protocols (French et al. 2008; Hoffman et al. 2010; Scott et al. 2003). Following the ST, there were significant reductions in rowing stroke power and jump height, which persisted for 48 h. The percentage decreases in performance for the anaerobic power tests across the assessed time points were 10, 7, 5% for SSJ, which were similar to the decrements of 9, 6, 5% for CMJ, while the decrements in PS were smaller (5, 5, 3%). However, the 90% confidence intervals demonstrate that decrements in performance on these tests vary widely between participants. This indicates that when rowers perform power testing/training sessions, a wide variation in the acute responses (<48 h) is likely following intense strength training across a crew. The decrements in jump height in our study were smaller than have been recorded following protocols of high volume barbell squats and plyometric jumps (9–17%; Marginson et al. 2005; Skurvydas et al. 2006; French et al. 2008). The larger decrements in jump height in these studies are not surprising, since the squat and plyometric protocols employed were more 'leg-dominant' than the whole-body strength training used in the present study. The greater overall recruitment of the lower body musculature would translate to exaggerated damage in these muscles, which are primarily used for the performance of SSJ and CMJ (Crowther et al. 2007). Interestingly, in comparison to our ST, higher intensity but lower volume bouts of multi-joint strength training have resulted in a smaller decrement in SSJ performance in trained participants (Raastad and Hallen 2000) and lower CK levels in rowers (Kokalas et al. 2004). These findings indicate that

the volume of strength training is a key determinant of the extent of the subsequent impairment of muscle function. Performance time for the 2,000 m ergometer test and related measures of heart rate, RPE and lactate were unaffected following strength training. This finding is contrary to that reported by other authors who have investigated short-term endurance performance (<8 min) following muscle damaging exercise. Twist and Eston (2009) reported 5 min cycling time trial performance to decrease 48 h following 100 countermovement jumps and Davies et al. (2008, 2009) have shown significantly shorter times to exhaustion in maximal cycling ramp tests performed 48 h after high volume barbell squats. The muscle damaging exercise protocols used by Twist and Eston (2009) and Davies et al. (2008, 2009) highly concentrated on the lower body musculature and the performance test modality was cycling exercise. In our study, the ST was whole body focused and so was the performance test (2,000 m on row ergometer). Interestingly, a greater rise in perceived soreness was experienced by the participants involved in the studies by Twist and Eston (2009) and Davies et al. (2008, 2009) compared to the ratings attributed by our participants in response to the ST. This may have been due to the concentration on lower limb activity and/or because their participants had not participated in resistance training for 6 months prior to involvement. Marcora and Bosio (2007) have previously questioned the validity of applying results obtained from novice participants to the athletic setting. Athletes regularly participating in resistance training (as in our study), or any exercise featuring stressful eccentric actions, would have a level of protection from such exercise due to the repeated bout effect, which makes comparison with 'novice' trainers less meaningful (McHugh 2003). The observed decreases in jump height and stroke power and the increases in markers of muscle damage indicate that at the time points when the 2,000 m test was performed, rowers exhibited muscle damage and were in a state of strain or transient over-reaching. However, this state did not seem to influence 2,000 m ergometer performance. These findings are in agreement with the results of Mäestu et al. (2005), who assessed 2,000 m ergometer performance following a 3-week intensive period of training. During this 3-week period, rowers increased their training volume by 100%, which resulted in decreases in the T/C ratio, an indicator of over-reaching (Vervoorn et al. 1992). However, despite this, 2,000 m performance was not significantly altered following the intensive training period. Our findings indicate that the current practice of scheduling endurance rowing training sessions in close proximity to bouts of strength training (Gee et al. 2011) is justified. Previous literature has shown that type II fibres may be more susceptible to damage from eccentric exercise compared to type I fibres (Jones et al. 1986; Asp et al. 1998). Aerobic metabolism is primarily responsible for energy provision during 2,000 m rowing; thus, performance is highly dependent on type I muscle fibres (Hagerman et al. 1978), whereas the maximal power tests would be more dependent on recruitment of type II fibres (Potteiger et al. 1999). Therefore, the specific muscle function required to carry out the 2,000 m row may have allowed performance to be maintained to a greater extent than that required for the power tests. This notion is supported by the consistent finding in literature that muscle strength/power has been affected by muscle damaging exercise to a greater extent than endurance performance when both have been assessed consecutively (Paschalis et al. 2005b; Chen et al. 2007, 2008; Marcora and Bosio 2007; Davies et al. 2008; Twist and Eston 2009).

Conclusion

The findings from this study indicate that high-intensity strength training resulted in symptoms of muscle damage and decrements in rowing-specific power that last 48 h, but has no impact on short-term endurance performance (~6–7 min). Since the muscle damaging exercise protocol was familiar to the participants, it is likely that the repeated bout effect protected participants from exaggerated decrements in functional performance that have been shown to result following novel exercise challenges. Our findings provide important considerations for those responsible for the planning and monitoring of training in rowers, notably that it could be predicted that rowers would perform suboptimally when engaging in primary 'anaerobic' physical tests or power training sessions up to 48 h following high-intensity strength training. However, performance in longer, more 'aerobic' sport-specific tests or training sessions would not be significantly affected 24 h after high-intensity strength training. Our findings indicate that the current practice of scheduling endurance rowing training sessions in close proximity to bouts of strength training is justified.

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References

Apostolidis N, Nassis GP, Bolatoglou T, Geladas ND (2004) Physiological and technical characteristics of elite young basketball players. J Sports Med Phys Fitness 44:157–163

Asp S, Daugaard JR, Kristiansen S, Kiensand B, Richter EA (1998) Exercise metabolism in human skeletal muscle exposed to prior eccentric exercise. J Physiol 509:305–313

Atkinson G, Nevill AM (2001) Selected issues in design and analysis of sport performance research. J Sports Sci 19:811–827

Avery NG, Kaiser JL, Sharman MJ, Scheett TP, Barnes DM, Gómez AL, Kraemer WJ, Volek JS (2003) Effects of vitamin E supplementation on recovery from repeated bouts of resistance exercise. J Str Cond Res 17:801–809

Batterham AM, Hopkins WG (2006) Making meaningful inferences about magnitudes. Int J Sports Physiol Perform 1:50–57

Borg G (1970) Perceived exertion as an indicator of somatic stress. Scand J Rehabil Med 2:92–98

Braun WA, Dutto DJ (2003) The effects of a single bout of downhill running and ensuing delayed onset of muscle soreness on running economy performed 48 h later. Eur J Appl Physiol 90:29–34

Bret C, Rahmani A, Dufour AB, Messonnier L, Lacour JR (2002) Leg strength and stiffness as ability factors in 100 m sprint running. J Sports Med Phys Fitness 42:274–281

Byrne C, Eston R (2002a) The effect of exercise-induced muscle damage on isometric and dynamic knee extensor strength and vertical jump performance. J Sports Sci 20:417–425

Byrne C, Eston R (2002b) Maximal-intensity isometric and dynamic exercise performance after eccentric muscle actions. J Sports Sci 20:951–959

Calbet JAL, Chavare J, Dorado D (2001) Running economy and delayed onset muscle soreness. J Sports Med Phys Fitness 41:18–26

Chen TC, Nosaka K, Tu JH (2007) Changes in running economy following downhill running. J Sports Sci 25:55–63

Chen TC, Nosaka K, Wu CC (2008) The rate of increase in rating of perceived exertion predicts the duration of exercise to fatigue at a fixed power output in different environmental conditions. Eur J Appl Physiol 103:569–577

Chiu LZF, Schilling BK (2005) A primer on weighlifting: from sport to sports training. Strength Cond J 27:42–48

Crowther RG, Spinks WL, Leicht AS, Spinks CD (2007) Kinematic responses to plyometric exercises conducted on compliant and non-compliant surfaces. J Str Cond Res 21:460–465

Davies RC, Eston RG, Poole DC, Rowlands AV, Dimenna F, Wilkerson DP, Twist C, Jones AM (2008) The effect of eccentric exercise-induced muscle damage on the dynamics of muscle oxygenation and pulmonary oxygen uptake. J Appl Physiol 105:1387–1388

Davies RC, Rowlands AV, Eston RG (2009) Effect of exercise-induced muscle damage on ventilatory and perceived exertion responses to moderate and severe intensity cycle exercise. Eur J Appl Physiol 107:11–19

Di Cagno A, Baldari C, Battaglia C, Brasili P, Merni F, Piazza M, Toselli S, Ventrella AR, Guidetti L (2008) Leaping ability and body composition in rhythmic gymnasts for talent identification. J Sports Med Phys Fitness 48:341–346

French DN, Thompson KG, Garland SW, Barnes CA, Portas MD, Hood PE, Wilkes G (2008) The effects of contrast bathing and compression therapy on muscular performance. Med Sci Sports Exerc 40:1297–1306

Gee T, Olsen P, Thompson K, Golby J, Garland S, White D (2008) Recovery of rowing sprint performance after high-intensity strength training. J Sports Sci 26 (Suppl 2): S66

Gee TI, Olsen PD, Berger NJ, Golby J, Thompson KG (2011) Strength and conditioning practices in rowing. J Str Cond Res 25:668–682

Gleeson M, Blannin AK, Zhu B, Brooks S, Cave R (1995) Cardiorespiratory, hormonal and haematological responses to submaximal cycling performed 2 days after eccentric or concentric exercise bouts. J Sports Sci 13:471–479

Gleeson M, Blannin AK, Walsh NP, Field CN, Pritchard JC (1998) Effect of exercise-induced muscle damage on the blood lactate response to incremental exercise in humans. Eur J Appl Physiol Occup Physiol 77:292–295

Hagerman FC, Connors MC, Gault JA, Hagerman GR, Polinski WJ (1978) Energy expenditure during simulated rowing. J Appl Physiol 45:87–93

Hoffman JR, Ratamess NA, Tranchina CP, Rashti SL, Kang J, Faigenbaum AD (2010) Effect of a proprietary protein supplement on recovery indices following resistance exercise in strength/power athletes. Amino Acids 38:771–778

Hopkins WG (2007) A spreadsheet for deriving a confidence interval, mechanistic inference and clinical inference from a *p* value. Sportscience 11:16–20

Hopkins WG, Schabort EJ, Hawley JA (2001) Reliability of power in physical performance tests. Sports Med 31:211–234

Hopkins WG, Marshall SW, Batterham AM, Hanin J (2009) Progressive statistics for studies in sports medicine and exercise science. Med Sci Sports Exerc 41:3–12

Ingham SA, Whyte GP, Jones K, Nevill AM (2002) Determinants of 2, 000 m rowing ergometer performance in elite rowers. Eur J Appl Physiol 88:243–246

Ingham SA, Carter H, Whyte GP, Doust JH (2007) Comparison of the oxygen uptake kinetics of club and Olympic champion rowers. Med Sci Sports Exerc 39:865–871

Ivey P, Oakley J, Hagerman P (2004) Strength training for the preparatory phase in collegiate women's rowing. Strength Cond J 26:10–15

Jones DA, Newham DJ, Round JM, Tolfree SE (1986) Experimental human muscle damage: morphological changes in relation to other indices of damage. J Physiol 375:435–448

Kokalas N, Tsalis G, Tsigilis N, Mougios V (2004) Hormonal responses to three training protocols in rowing. Eur J Appl Physiol 92:128–132

Lavender AP, Nosaka K (2008) A light load eccentric exercise confers protection against a subsequent bout of more demanding eccentric exercise. J Sci Med Sport 11:291–298

Linnamo V, Pakarinen A, Komi PV, Kraemer WJ, Häkkinen K (2005) Acute hormonal responses to submaximal and maximal heavy resistance and explosive exercises in men and women. J Str Cond Res 19:566–571

Lohman TG, Roche AF, Martorell R (1988) Anthropometric Standardization Reference Manual, Human Kinetics. Champaign, Illinois

Machado M, Koch AJ, Willardson JM, Pereira LS, Cardoso MI, Motta MK, Pereira R, Monteiro AN (2010) Effect of varying rest intervals between sets of assistance exercise on creatine kinase and lactate dehydrogenase responses. J Str Cond Res (in press)

Mäestu J, Jürimäe J, Jürimäe T (2005) Monitoring of performance and training in rowing. Sports Med 35:597–617

Marcora SM, Bosio A (2007) Effect of exercise-induced muscle damage on endurance running performance. Scand J Med Sci Sports 17:662–671

Marginson V, Rowlands AV, Gleeson NP, Eston RG (2005) Comparison of the symptoms of exercise-induced muscle damage after an initial and repeated bout of plyometric exercise in men and boys. J Appl Physiol 99:1174–1181

McHugh MP (2003) Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise. Scand J Med Sci Sports 13:88–97

McNeely E, Sandler D, Bamel S (2005) Strength and power goals for competitive rowers. Strength Cond J 27:10–15

Miyama M, Nosaka K (2007) Protection against muscle damage following fifty drop jumps conferred by ten drop jumps. J Str Cond Res 21:1087–1092

Nosaka K, Clarkson PM (1996) Variability in serum creatine kinase response after eccentric exercise of the elbow flexors. Int J Sports Med 17:120–127

Nosaka K, Sakamoto K, Newton M, Sacco P (2001) How long does the protective effect on eccentric exercise-induced muscle damage last? Med Sci Sports Exerc 33:1490–1495

Paschalis V, Koutedakis Y, Jamurtas AZ, Mougios V, Baltzopoulos V (2005a) Equal volumes of high and low intensity of eccentric

exercise in relation to muscle damage and performance. J Str Cond Res 19:184–188

Paschalis V, Koutedakis Y, Baltzopoulos V, Mougious V, Jamurtas AZ, Theoharis V (2005b) The effects of muscle damage on running economy in healthy males. Int J Sports Med 26:827–831

Potteiger JA, Lockwood RH, Haub MD, Dolezal BA, Alumzaini KS, Schroeder JM, Zebas CJ (1999) Muscle power and fiber characteristic following 8 weeks of plyometric training. J Str Cond Res 13:275–279

Raastad T, Hallen J (2000) Recovery of skeletal muscle contractibility after high- and moderate-intensity strength exercise. Eur J Appl Physiol 82:206–214

Requena B, Gonzàlez-Badillo JJ, de Villareal ES, Ereline J, García I, Gapeyeva H, Pääsuke M (2009) Functional performance, maximal strength, and power characteristics in isometric and dynamic actions of lower extremities in soccer players. J Str Cond Res 23:1391–1401

Riechman SE, Zoeller RF, Balasekaran G, Goss FL, Robertson RJ (2002) Prediction of 2000 m indoor rowing performance using a 30 s sprint and maximal oxygen uptake. J Sports Sci 20:681–687

Rowlands AV, Eston RG, Tilzey C (2001) Effect of stride length manipulation on symptoms of exercise-induced muscle damage and the repeated bout effect. J Sports Sci 19:333–340

Rowlands DS, Rossler K, Thorp RM, Graham DF, Timmons BW, Stannard SR, Tarnopolsky MA (2008) Effect of dietary protein content during recovery from high-intensity cycling on subsequent performance and markers of stress, inflammation, and muscle damage in well-trained men. Appl Physiol Nutr Metab 33:39–51

Schabort EJ, Hawley JA, Hopkins WG, Mujika I, Noakes TD (1998) A new reliable laboratory test of endurance performance for road cyclists. Med Sci Sports Exerc 30:1744–1750

Scott KE, Rozenek R, Russo AC, Crussemeyer JA, Lacourse MG (2003) Effects of delayed onset muscle soreness on selected physiological responses to submaximal running. J Str Cond Res 17:652–658

Skurvydas A, Streckis V, Mickeviciene D, Kamandulis S, Stanislovaitis A, Mamkus G (2006) Effect of age on metabolic fatigue and on indirect symptoms of skeletal muscle damage after stretch-shortening exercise. J Sports Med Phys Fitness 46:431–441

Spiering BA, Kraemer WJ, Vingren JL, Hatfield DL, Fragala MS, Ho JY, Maresh CM, Anderson JM, Volek JS (2007) Responses of criterion variables to different supplemental doses of L-carnitine L -tartrate. J Str Cond Res 21:259–264

Twist C, Eston RG (2009) The effect of exercise-induced muscle damage on perceived exertion and cycling endurance performance. Eur J Appl Physiol 105:559–567

Vervoorn C, Vermulst LJ, Boelens-Quist AM, Koppeschaar HP, Erich WB, Thijessen JH, de Vries WR (1992) Seasonal changes in performance and free testosterone: cortisol ratio of elite female rowers. Eur J Appl Physiol Occup Physiol 64:14–21

Xue Q, Yeung ES (1994) Variability of intracellular lactate dehydrogenase isoenzymes in single human erythrocytes. Anal Chem 66:1175–1178