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Section III – Sports Training

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Investigating the Effects of Typical Rowing Strength Training Practices on Strength and Power Development and 2,000 m Rowing Performance

by
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This study aimed to determine the effects of a short-term, strength training intervention, typically undertaken by club-standard rowers, on 2,000 m rowing performance and strength and power development. Twenty-eight male rowers were randomly assigned to intervention or control groups. All participants performed baseline testing involving assessments of muscle soreness, creatine kinase activity (CK), maximal voluntary contraction (leg-extensors) (MVC), static-squat jumps (SSJ), counter-movement jumps (CMJ), maximal rowing power strokes (PS) and a 2,000 m rowing ergometer time-trial (2,000 m) with accompanying respiratory-exchange and electromyography (EMG) analysis. Intervention group participants subsequently performed three identical strength training (ST) sessions, in the space of five days, repeating all assessments 24 h following the final ST. The control group completed the same testing procedure but with no ST. Following ST, the intervention group experienced significant elevations in soreness and CK activity, and decrements in MVC, SSJ, CMJ and PS ($p < 0.01$). However, 2,000 m rowing performance, pacing strategy and gas exchange were unchanged across trials in either condition. Following ST, significant increases occurred for EMG ($p < 0.05$), and there were non-significant trends for decreased blood lactate and anaerobic energy liberation ($p = 0.063 – 0.086$). In summary, club-standard rowers, following an intensive period of strength training, maintained their 2,000 m rowing performance despite suffering symptoms of muscle damage and disruption to muscle function. This disruption likely reflected the presence of acute residual fatigue, potentially in type II muscle fibres as strength and power development were affected.

Key words: *recovery, muscle function, muscle damage, resistance training, endurance performance.*

Introduction

Strength training forms an integral part of the structured regimen of elite rowers accounting for 10-20% of total training time (Gee et al., 2011b; Guellich et al., 2009). Descriptive research suggests that during the competitive season rowers perform strength training with loading between 85 and 95% of their one repetition maximum (1 RM) (Gee et al., 2011b; McNeely et al., 2005). In support of this practice, rowing specific concurrent training research has demonstrated the effectiveness of the addition of eight week high load strength training (~70-90% 1 RM) protocols for eliciting greater improvements in rowing performance compared to rowing only or lower load (< 70% 1 RM) strength training (Ebben et al., 2004; Izquierdo-Gabarren et al., 2010). For athletes who concurrently train for both strength and endurance, three weekly strength

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training sessions have been recommended (Garcia-Pallares and Izquierdo, 2011). Accordingly, rowers tend to perform two to three strength training sessions a week with the final session often occurring 24- to 48 h before a high-quality rowing session or even a competition (Gee et al., 2011a). However, it has been shown that high volume strength training can lead to the development of 'residual fatigue' and sub-optimal adaptations in rowing performance, compared to lower volume strength training (Izquierdo-Gabarren et al., 2010). Hence, to illicit optimal adaptations and performance, the prescription of strength training for rowers requires careful implementation and monitoring.

Previously many authors have investigated the effect of acute singular bouts of strength and power training on muscle function within cohorts of competitive athletes (Gee et al., 2011a; Gee et al., 2012; Raastad and Hallén, 2000). These authors have recorded decreases in various aspects of muscle function including peak power, jump height, sprint time, dynamic strength and symptoms of transient muscle damage following acute strength and plyometric training. Conversely, recent research has shown a post-potentiation effect of acute strength training upon dynamic strength, jump and sprint ability assessments performed 6 h following (Cook et al., 2014). This observed morning to afternoon performance simulative effect was attributed to hormonal priming via acute strength training and related to within-day circadian rhythm, yet longer lasting effects (24 h >) were not investigated. However, in practice athletes perform multiple bouts of strength training on a weekly basis, which could present a collaborative fatigue effect (Gee et al., 2011b; Izquierdo-Gabarren et al., 2010). Therefore, to investigate influence of high-load strength training on athletes, analysis of effects on muscle function of multiple sessions performed across a series of days is more applicable than investigating effects following a single bout.

Several researchers have investigated the effects of short-term strength training (over 4-7 days) on various aspects of muscle function, although within non-athlete populations. Cohorts of non-athlete male participants were shown to demonstrate decreases in running sprint ability, isokinetic strength and 1 RM bench press as well as 1 RM squat performance after 4-5 sessions of multi-joint strength training over 4-5 days (Fry et al., 1994; Kraemer et al., 2006). However, it is questionable whether these effects are transferrable to an endurance trained population, as previously, trained rowers have shown greater physical robustness than non-athlete groups following acute singular bouts of strength training (Gee et al., 2011a; Gee et al., 2012; Scott et al., 2003). The distinct parameters of strength training prescription for rowers, including frequency, exercise selection and loading have been previously identified through descriptive research (Gee et al., 2011b; McNeely et al., 2005). However, it is still unclear what effect a typical weekly regimen of high-load strength training would have on rowing performance. Such information would be pertinent to both the acute and longitudinal programming of strength training into the training regimen of rowing athletes.

The purpose of this study was to determine the effects of a short-term, high-load strength training intervention, typically undertaken by club-standard rowers, on 2,000 m rowing ergometer performance and power development. It was hypothesized that 2,000 m rowing ergometer performance would be unaffected in trained rowers, despite the occurrence of symptoms of muscle damage and decrements in muscle function following a series of three strength training sessions performed over five days.

Material and Methods

Participants

Twenty-eight well-trained club-standard male rowers were randomly assigned to two groups: a strength training intervention group (STG) (n: 14, mean ± SD, age: 21 ±3.2 years, body mass: 79.9 ±7.3 kg, body height: 1.83 ±0.05 m, 2,000 m ergometer time: 6:34.1 ±0:08.5 min:s) and a control group (CG) (n: 14, age: 22.3 ±4.8 years, body mass: 84.1 ±8.5 kg, body height: 1.85 ±0.05 m, 2,000 m ergometer time: 6:35.7 ±0:11.2 min:s). The participants had all competed at national level events such as the 'Henley Royal Regatta', the 'National Rowing Championships of Great Britain' and the 'British Universities and Colleges Sports Rowing Championships'. The participants had 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 the recruited rowers into the context, Ingham et al. (2007) found eight Olympic champion rowers to have a 2,000 m time of 5.53.4 min:s. At the time of the study, participants were within the pre-season phase of training which encompassed one to two boat/ergometer rowing training sessions being performed daily (Gee et al., 2011b). All participants had at least one year of experience of regularly performed structured strength training, and all maintained a consistent strength training frequency of two to three sessions per week. Participants provided written informed consent to participate in the study, which was approved by the ethics committee of the School of Life Sciences at Northumbria University and in line with the Declaration of Helsinki for research with human volunteers.

Procedures

The study followed a within and between group randomised controlled design to determine the effects of a protocol involving three strength training sessions performed in five days on 2,000 m rowing ergometer performance and muscle function. Prior to the study, all participants performed a familiarisation testing session involving all the assessments featured in the experimental protocol. For the baseline trial, participants were asked to arrive at the laboratory having abstained from exercise on the day of testing and strength training for 72 h. Initially participants' perceived muscle soreness was measured by a 10 cm visual analogue scale, used previously (Avery et al., 2003), and serum creatine kinase (CK) was determined via a 30 μl capillary blood sample using the Reflotron® Plus (Roche, Grenzach-Wyhlen, Germany). Participants then performed a 5 min warm-up on a rowing ergometer (Concept 2 Model C, Concept 2 Ltd, Wilford, Notts, UK). Thereafter, a protocol of strength and power tests involving assessment of maximal voluntary contraction force of the leg extensors at 90° (MVC) using a strain gauge (MIE Medical Research Ltd, Leeds, UK), three individual static-squat jumps (SSJ) and counter-movement jumps (CMJ) using an optoelectronic sensor (Optojump Next, Microgate, Bolzano, Italy), and five maximal rowing power strokes (PS) performed at a rate of 30 strokes·min^{-1}, as described previously (Ingham et al., 2002), was completed.

Participants then performed a 2,000 m ergometer test (2,000 m) with accompanying respiratory-exchange and EMG analysis. Before the test, as a warm up, participants rowed sub-maximally for 5 min. During each trial, the only feedback given to participants was their stroke rate and distance remaining. Expired breath-by-breath respiratory gas exchange parameters ($\dot{V}O_2$ and $\dot{V}CO_2$) were measured using an automated online metabolic cart (Cortex, Metalyzer, Leipzig, Germany). Participants reported their rating of perceived exertion (RPE) [6-20 scale] immediately after the test was completed. Capillary blood samples (20 μl) for assessment of blood lactate ([LA$^-$]) were drawn at completion of the test and at 1, 3, 5 and 7 min of recovery and analysed using the Biosen C_Line Sport (2 channel) lactate and glucose analyzer (EKF Diagnostic, Barleben, Germany). Contributions of the aerobic (P_{aer}) and anaerobic metabolism (P_{anaer}) to total power (P_{tot}) during each 500 m stage were calculated according to methods previously described (de Koning et al., 1999) using previously established exercise efficiency for trained rowers performing 2,000 m ergometer testing (Hagerman et al., 1978).

Surface EMG was recorded from seven anatomical sites: gastrocnemius (GA), biceps femoris (BF), gluteus maximus (GM), erector spinae (ES), vastus medialis (VM), rectus abdominis (RA) and latissimus dorsi (LD), and measured during PS and the 2,000 m test. Preparation and placement were performed in accordance with SENIAM guidelines (Hermans et al., 2000). Surface EMG was collected at a sampling frequency of 1000 Hz and amplified (gain: 1000) using a 16 channel wireless telemetric system (Myon RFTD-E16, Myon AG, Baar, Switzerland) interfaced with a multifunction data acquisition module (USB-6210, National instruments, Austin, Texas, USA). Data were recorded within commercially available software (MyoResearch XP, Noraxon, Scottsdale, Arizona, USA) prior to being exported for analysis within alternative software (LabChart 7, AD Instruments, Oxford, UK). The raw EMG data were high-pass filtered with a cut off frequency of 15 Hz and the filtered data were full-wave rectified. Mean rectified EMG recorded during each 500 m stage of the 2,000 m test was normalised against the mean rectified EMG recorded during the PS.

The following week, the STG performed a strength training protocol (ST) involving a series of multi-joint strength training exercises, which was completed three times over five days with a day break between the first, second and third sessions (Table 1). Individual loading of exercises was determined from one-repetition maximum (1 RM) which was performed prior to commencement of the experimental protocol. Two minutes of rest were allowed between each set. The chosen exercises are performed routinely by rowers (Gee et al., 2011b) and the participants regularly performed these exercises in their training. The day after the final ST session, the STG performed the follow-up trial, which featured the same battery of tests performed for baseline measures. At this time point, the CG also performed the follow-up trial.

Statistical Analysis

Data are presented as mean (± SD), unless stated otherwise. Testing for data normality and homogeneity of variance was conducted prior to analysis. To assess the effect of the ST on markers of muscle damage, strength / power test performance, 2,000 m time and related physiological variables, two-way ANOVA (group x trial) tests were conducted. Three-way (group x trial x stage) ANOVA tests were conducted to assess the impact of the ST on pacing strategy during 2,000 m. The significance level was set at $p < 0.05$ for all analyses and the LSD correction was used for pairwise comparisons. Effect size (*ES*) was calculated for any non-statistically significant result trends (p = 0.051-0.10) in accordance to procedures suggested by Hopkins (2003) and consequently, these procedures interpretation of observed effect sizes are as follows: trivial < 0.2, small 0.2-0.6, moderate 0.6-1.2, large 1.2-2.0, very large > 2.0 (Hopkins, 2003).

Additionally, inferential statistics were used to quantify the magnitude of the change in measures exhibited post-strength training (Batterham and Hopkins, 2006). This was done by calculating the smallest practical effect for each dependent variable from the product of 0.3 (represents the smallest standardised change in mean for a group of trained participants) multiplied by 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 Batterham and Hopkins (2006).

Results

Markers of muscle damage

Significant trial x group interactions were observed for muscle soreness ($F_{1,13}$ = 8.59, p = 0.007) and CK (log transformed values; $F_{1,13}$ = 18.39, $p < 0.001$), with increases in both variables demonstrated for the STG during the follow-up trial in comparison to baseline ($p < 0.001$), while values for the CON remained unchanged (p = 0.883-0.965) (Table 2). Practical inferences indicated that muscle soreness and CK activity were 'very' to 'most' likely to increase following ST.

Strength and power tests

Following ST there was a significant reduction in MVC in the STG ($F_{1,13}$ = 10.76, p = 0.003), while no changes occurred from baseline to follow up in CON (p = 0.142). The practical inference suggested that decreases in MVC were 'likely' to occur following ST. There were significant trial x group interactions for SSJ height ($F_{1,13}$ = 6.41, p = 0.018) and CMJ height ($F_{1,13}$ = 15.58, p = 0.001). Pairwise comparisons demonstrated there were significant reductions in SSJ and CMJ ($p < 0.001$) following ST (Table 2), while no changes occurred in the control group (SSJ: p = 0.179, CMJ: p = 0.861). The practical inference was that decreases in SSJ and CMJ height were 'most likely' to occur following strength training. There was a significant trial x group interaction for PS ($F_{1,13}$ = 14.41, p = 0.001). For the STG, PS was significantly reduced following ST ($p < 0.001$), while no changes occurred in CON (p = 0.740). The practical inference suggested that decreases in stroke power were 'very likely' to occur following ST.

2,000 m ergometer time trial performance and physiological measures

There were no changes in 2,000 m time ($F_{1,13}$ = 0.91, p = 0.350), mean $\dot{V}O_2$ ($F_{1,13}$ = 3.76, p = 0.065; STG: p = 0.168, CG: p = 0.201) and RPE ($F_{1,13}$ = 0.064, p = 0.802) from baseline to follow-up for either group. There was a trend toward a trial x group interaction effect for peak [LA^-] ($F_{1,13}$ = 3.67, p = 0.066) with pairwise comparisons revealing a trend toward a reduction in the follow-up trial for the STG (p = 0.063, *ES* = 0.40), whereas no changes

occurred for the CON (p = 0.449) (Table 2).

Pacing of the 2,000 m ergometer time trial

There were no differences in mean power output from baseline to follow-up for each 500 m segment of trials, within or between experimental groups ($F_{3,13}$ = 0.75, p = 0.446).

Table 1

The design of a strength training session and mean ± standard deviation of 1 RM achieved by the intervention group participants on the exercises featured

Exercise	Sets x reps	% 1 RM / weight used	1 RM achieved (kg)*
Snatch grip high pull	4x5	85%	60 (7.5)
Clean	4x5	85%	75 (10)
Back squat	4x5	85%	105 (12.5)
Romanian deadlift	3x8	75% of squat 1 RM	-
Bench press	3x5	85%	75 (12.5)
Bench pull	3x5	85%	77.5 (10)
Weighted sit-ups	3x15	15 kg	-

** Mean ± standard deviation rounded to nearest 2.5 kg increment*

Table 2

Comparison of 2,000 m performance, physiological measures, strength and power tests and markers of muscle damage across baseline and follow up trials for both groups

Measure	Intervention		Control	
	Baseline	Follow up	Baseline	Follow up
2,000 m (min:s)	6:46.3 (0:07.9)	6:46.3 (0:08.8)	6:45.3 (0:12.2)	6:43.7 (0:11.4)
$\dot{V}O_2$ (L·min^{-1})	4.99 (0.55)	5.14 (0.38)	5.04 (0.35)	4.92 (0.39)
[LA$^-$] (mmol·L^{-1})	16.8 (2.6)	15.7 (2.9)†	17.3 (3.5)	17.8 (4.1)
RPE	18 (1)	18 (1)	17 (1)	17 (1)
MVC (N)	569 (68)	533 (61)*	596 (82)	580 (81)
SSJ (cm)	32.6 (4.0)	29.8 (3.9)*	31.9 (5.7)	31.2 (5.6)
CMJ (cm)	35.6 (4.3)	32.7 (4.0)*	33.8 (5.9)	33.9 (6.5)
PS (W)	523 (51)	491 (49)*	511 (47)	509 (45)
Muscle soreness	2.2 (2.3)	4.2 (1.3)*	1.0 (1.0)	1.1 (1.4)
CK (U/L)	188 (125)	523 (348)*	195 (127)	190 (105)

** Significant difference from the baseline trial ($p < 0.05$).*

† = Trend for mean difference compared to the baseline trial (p = 0.051-0.10).

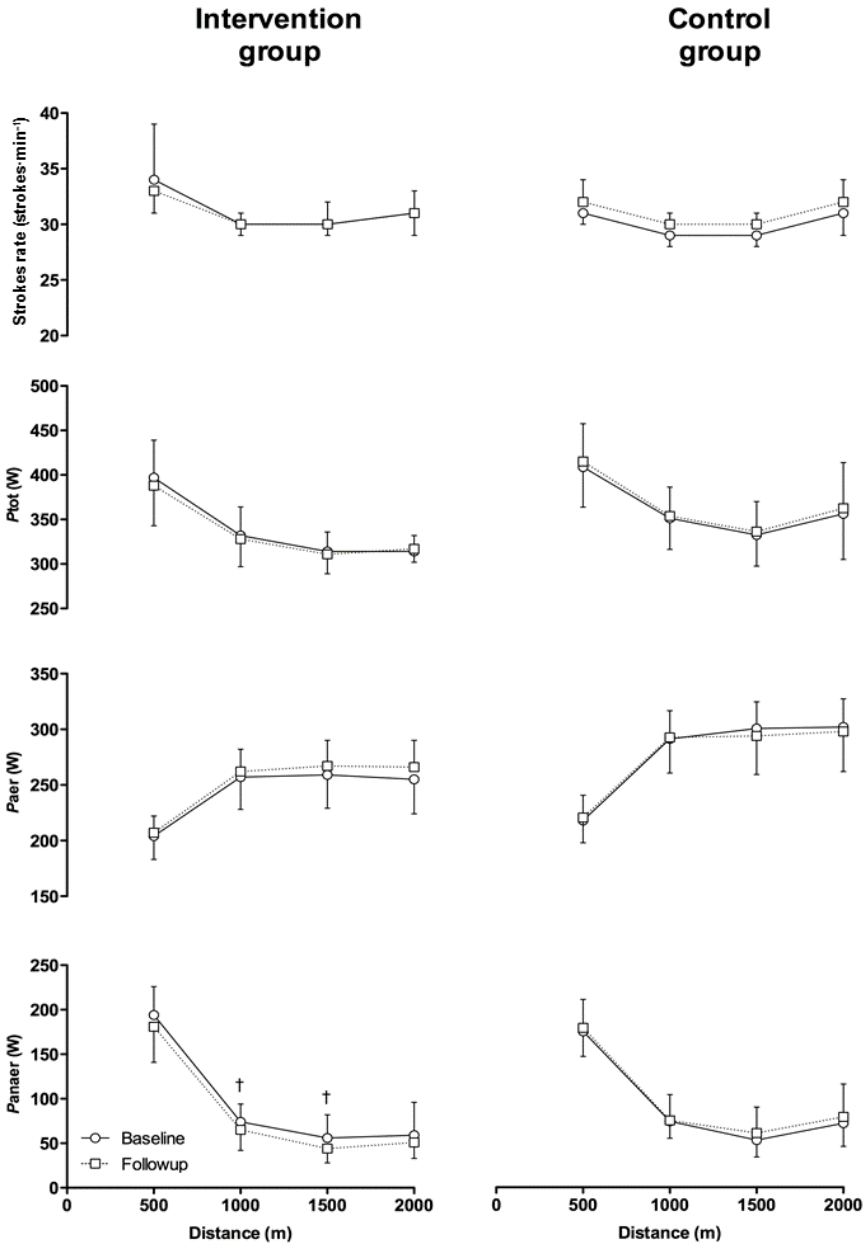

Figure 1

Anaerobic and aerobic contributions to total power (watts) and a stroke rate (strokes·min^{-1}) for baseline and follow trials during successive 500 m stages of the 2,000 m time trial for the intervention (n = 11; errors occurred during breath-by-breath measurement for 3 participants) and control groups (n = 14).
** Significant difference between baseline and follow-up trials p < 0.05.*
† Trend for a significant difference between intervention baseline and follow-up trials p < 0.10

There was a trend for a stage x group interaction for P_{anaer} ($F_{3,13}$ = 2.84, p = 0.085), which indicated decreases in the follow-up trial for the STG ($F_{1,10}$ = 3.23, p = 0.086), compared to the baseline trial, due to decreases in P_{anaer} during stages two (p = 0.089, ES = 0.42) and three (p = 0.099, ES = 0.56) of the follow-up trial. No changes occurred in P_{anaer} for CON ($F_{1,13}$ = 0.86, p = 0.363). There were no changes in P_{aer} ($F_{3,13}$ = 0.48, p = 0.553), mean $\dot{V}O_2$ ($F_{3,13}$ = 0.41, p = 0.632) or a stroke rate ($F_{3,13}$ = 0.412, p = 0.589) per 500 m stage from baseline to follow-up either within or between groups (Figure 1). Rectified EMG was unchanged for all seven sites between baseline and follow-up trials for the CON (p > 0.05). For the STG rectified EMG for the GM, VM and BF were unchanged between baseline and follow-up trials, however, changes were detected in the other four muscles. Rectified EMG for the RA ($F_{1,6}$ = 7.71, p = 0.013) significantly decreased during the follow-up trial in comparison to baseline, however, significant increases were shown in the follow-up trial for GA ($F_{1,9}$ = 5.09, p = 0.038), LD ($F_{1,10}$ = 14.15, p = 0.001) and ES ($F_{1,9}$ = 4.25, p = 0.053) demonstrating greater overall muscle activation in the STG post-ST 2,000 m trial.

Discussion

The most significant finding of this study was that trained rowers could maintain 2,000 m ergometer rowing performance following a protocol involving three high-load strength training sessions over a five day period, despite suffering symptoms of muscle damage and soreness as well as significant reductions in strength and power development. The pacing profile of the 2,000 m row was also unaffected by ST, although there was some indirect evidence of a redistribution of the anaerobic energy contribution during the middle portion of the trial. These data provide some evidence that three intensive strength training sessions over five days can be tolerated by trained rowers, and supports reported coaching practice with competitive rowers (Gee et al., 2011b).

It could be suggested that the imposed intensity and volume of the ST could act as a template for tolerable strength training prescription amongst rowers. However, the prescribed exercise repetitions during this session (most commonly five at 85% 1 RM) were generally at the limit of what the participants could perform at the prescribed loading. Previously Izquierdo-Gabarren et al. (2010) found that performing strength training with loading of 75-92% 1 RM and a sub-maximal number of repetitions per set (two to five) was more effective at improving rowing ergometer performance, after eight weeks of training, than training to volitional failure (four to ten repetitions) with the same loading. The authors suggested that the repetition to failure programme may have surpassed a threshold of a training load whereby sub-optimal adaptations in strength and endurance would result due to the development of residual fatigue in the neuromuscular system. Our findings demonstrated that high-load strength training, three times per week, could affect strength and power development and produce symptoms of muscle fatigue, damage and soreness.

The presence of such 'residual fatigue' has been suggested to manifest from alterations within the endocrine system and adrenal glands. Decreases in concentrations of anabolic hormones associated with adaptability such as testosterone and IGF-1 have been found to occur in response to both short-term two-week strength training overreaching interventions and a prolonged 16 week intervention utilising a repetition to failure approach (Fry et al., 2006; Izquierdo et al., 2006). In addition, epinephrine, the primary catecholamine contributing to physiological regulation, has been shown to increase following two weeks of strength training overreaching intervention, indicating the onset of sympathetic overtraining syndrome (Fry et al., 2006). The imposed ST in the current study featured higher volume (seven exercises, 3-4 sets of 5-8 repetitions (except sit-ups) at 75-85% 1 RM) than performed by the sub-maximal repetition group featured in Izquierdo-Gabarren et al. (2010) (four exercises, 3-4 sets of 2-5 repetitions at 75-92% 1 RM). Furthermore, significant decreases in maximal strength and power and likely muscle damage indicate the presence of acute 'residual fatigue' (Izquierdo-Gabarren et al., 2010). Consequently, strength training performed at this intensity over time in conjunction with the performance of regular rowing training could surpass the threshold of a tolerable training load and consequently decrease the potential for

longitudinal training adaptations (Izquierdo et al., 2006; Izquierdo-Gabarren et al., 2010). These findings taken together suggest that, to elicit optimal training adaptations from strength training, it is recommended that rowers should perform a sub-maximal number of repetitions at prescribed loading. In the context of the present study this would equate to two to three repetitions per set with loading of 85% 1 RM.

It is plausible that ST did not impact 2,000 m rowing performance or pacing strategy in the present study because of the sub-divisions of muscle fibres being primarily activated during the trial. The strength training prescribed reflects elite coaching practice (Gee et al., 2011b) and is aimed at developing rapid boat speed at the start of the race, hence force and impulse loads are high (generally 85% 1 RM). The featured Olympic lifting exercises involve the performance of explosive eccentric muscular contractions (Chiu and Schilling, 2005) and when coupled with a high load on most of the featured exercises it translates to a significant magnitude of eccentric muscle loading during the ST. Seemingly this eccentric loading was sufficient to cause muscle damage as reflected by the significant increases in associated markers of muscle soreness and CK activity (Byrne et al., 2004). Since muscle damage is preferentially precipitated in type II fibres following eccentric muscle actions, due to their limited resistance to fatigue and fragile structure (Friden and Lieber, 2001), it is likely that the ST caused damage and loss of function within these fibres.

Beltman et al. (2004) observed that type I and type IIA muscle fibres showed a significant reduction in phosphocreatine (PCr) (indicating their recruitment) at 39%, 72% and 87% of MVC during an isometric contraction, however, type IIAX fibres only demonstrated a reduction in PCr at 87% MVC. The strength and power tests in the present study required maximal instantaneous effort, likely recruiting the higher threshold type II fibres (IIAX, IIaX, IIX) (Sargeant, 2007). Subsequently the performance decreases within the strength and power tests can likely be attributed to a loss of function within these fibres. The power output generated during each 500 m segment of the 2,000 m time trial was 76%, 63%, 60% and 60% of the mean power generated during the power strokes test. Therefore, it seems probable that the power output produced during the 2,000 m time trial would have emanated largely from type I and IIA fibres, rather than the higher threshold fibres. This argument is supported by Fry et al. (1994) who found no changes in repetitions achieved on a squat machine at 70% 1 RM despite significant decreases in 1 RM on the same device following two weeks of strength training overreaching intervention. The authors suggested that the 70% effort was unaffected since the highest threshold motor units would not be activated throughout the activity. Therefore practically, higher intensity rowing training sessions such as sprints / power strokes and on-water efforts above race-pace requiring fast-starts (106-112% race pace) should be avoided in the 24 h following strength training (Gee et al., 2011b; Guellich et al., 2009). However, performance of endurance training at an intensity below the anaerobic threshold (< ~2 mmol·L-1), which accounts for the largest proportion of training time for rowers' and elite endurance athletes in general (~70% of the training load), is unlikely to be affected and therefore should be preferentially scheduled at this time (Secher, 1993). The findings of the present study would also indicate that high-quality, high-intensity rowing ergometer training is also still possible 24 h after high-load strength training.

In the present study, the observed increase in muscle soreness and elevated CK activity post-ST in the intervention group is indicative of moderate muscle damage (Byrne et al., 2004). As mentioned previously, muscle damage is preferentially precipitated in type II fibres following eccentric muscle actions and affects power producing ability (Friden and Lieber, 2001). Why this effect was not detrimental to 2,000 m rowing performance might be due to i) a different combination of muscle fibre recruitment being required for the activity and ii) that a loss of efficiency in some of the damaged / affected fibres, which would normally be recruited during the 2,000 m row, was compensated for by muscle rotation and additional muscle fibre activation as a result of increased efferent motor command (Proske et al., 2004). During the 2,000 m row, greater muscle activation at three muscle sites was detected in the post-ST intervention group trial, which taken cumulatively, might suggest that an increased

central motor drive was required to maintain performance during this trial. However, it must be acknowledged that caution should be taken when interpreting EMG data due to the inherent inter-participant variability in the measure (Fauth et al., 2010). An increased motor drive may have had a compensatory effect on power output, during the follow-up trial, which compensated for the effect of pre-existing damage to some of the type II fibres. In addition, a trend toward lower Panaer energy contribution and peak [LA-] might indicate less type II fibres were activated or functioning effectively in the follow-up trial and that force production was spread across type I fibres perhaps due to the process termed muscle wisdom (Enoka and Stuart, 1992). During this process the CNS provides economical activation of musculature by recruiting undamaged muscle fibres from the available pool, therefore compensating for any damaged fibres (Enoka and Stuart, 1992). Similarly, Scott et al. (2003) theorised that recruitment of undamaged muscle fibres from the available pool of fibres compensated for any damaged fibres 24-30 h following free weight strength training, which enabled endurance performance to be maintained. It has previously been shown that elite rowers display the ability to utilise a greater muscle recruitment alternation strategy during conditions of fatigue than novice rowers (So et al., 2007). The aforementioned authors suggested that this alternate muscle recruitment ability may be an important factor for maintenance of power output under fatigue. The increased muscle damage and associated decrease in muscle function within the intervention group may have provided impetus and warranted the observed additional central drive as well as modified muscle recruitment strategy in order to maintain 2,000 m performance.

Conclusions

This study demonstrated that trained rowers were able to maintain 2,000 m ergometer rowing performance even after an intensive period of strength training. However, three strength training sessions over a five day period caused disruption to muscle function leading to significant performance decrements in a range of strength and power assessments, coincident with residual muscle damage. This disruption likely reflects a loss of adaptability from the acute, repeated high-load strength training. It is likely that undertaking multiple high-load strength training sessions could adversely affect performance during subsequent high-intensity sprint based rowing training. This training modality would require significant recruitment of type II fibres which are likely to be negatively affected 24 h following a concentrated period of high-load strength training. Therefore, training modalities predominately involving the recruitment of type I fibres (< ~2 mmol·L-1) should be scheduled at this time. In this study, the muscle damage induced by strength training was likely more specific to the high threshold type II fibres, however, their apparent state of dysfunction did not adversely affect 2,000 m row performance suggesting sufficient power output could be produced by adjustments in muscle recruitment patterns. Significant increases in EMG at three anatomical sites during the follow-up 2,000 m trial may also suggest an increase in the central motor drive to compensate for peripheral damage in type II fibres.

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