Maximal-intensity isometric and dynamic exercise performance after eccentric muscle actions

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A well-documented observation after eccentric exercise is a reduction in maximal voluntary force. However, little is known about the ability to maintain maximal isometric force or generate and maintain dynamic peak power. These aspects of muscle function were studied in seven participants (5 males, 2 females). Knee extensor isometric strength and rate of fatigue were assessed by a sustained 60 s maximal voluntary contraction at 80° and 40° knee flexion, corresponding to an optimal and a shortened muscle length, respectively. Dynamic peak power and rate of fatigue were assessed during a 30 s Wingate cycle test. Plasma creatine kinase was measured from a fingertip blood sample. These variables were measured before, 1 h after and 1, 2, 3 and 7 days after 100 repetitions of the eccentric phase of the barbell squat exercise (10 sets × 10 reps at 80% concentric one-repetition maximum). Eccentric exercise resulted in elevations in creatine kinase activity above baseline (274 ± 109 U·l⁻¹; mean ± sₓ) after 1 h (506 ± 116 U·l⁻¹, P < 0.05) and 1 day (808 ± 117 U·l⁻¹, P < 0.05). Isometric strength was reduced (P < 0.05) for 7 days (35% at 1 h, 5% at day 7) and the rate of fatigue was lower (P < 0.05) for 3 days at 80° and for 1 day at 40°. Wingate peak power was reduced to a lesser extent (P < 0.05) than isometric strength at 1 h (13%) and, although the time course of recovery was equal, the two variables differed in their pattern of recovery. Eccentrically exercised muscle was characterized by an inability to generate high force and power, but an improved ability to maintain force and power. Such functional outcomes are consistent with the proposition that type II fibres are selectively recruited or damaged during eccentric exercise.

Keywords: eccentric exercise, fatigue, muscle damage, strength, Wingate test.

Introduction

Exercise-induced muscle damage often results from eccentric muscle actions and is synonymous with impaired muscle structure (Friden et al., 1983; Newham et al., 1983a; Jones et al., 1986), metabolism (O’Reilly et al., 1987; Asp et al., 1998) and function (Sargeant and Dolan, 1987; Clarkson et al., 1992). Perhaps the most important feature of eccentric exercise-induced muscle damage is the long-lasting impairment of muscle function. Strength is typically reduced immediately after eccentric exercise, followed by a linear recovery over a time course of hours (Davies and White, 1981; Newham et al., 1983b), days (Golden and Dudley, 1992; Hortobagyi et al., 1998; Byrne and Eston, 2002) or even in excess of 1 week (Clarkson et al., 1992; Cleak and Eston, 1992; Byrne et al., 2001) or 1 month (Howell et al., 1993; Sayers and Clarkson, 2001). Muscle function has primarily been assessed using measures of isometric maximal voluntary force (Davies and White, 1981; Newham et al., 1983b; Jones et al., 1989; Clarkson et al., 1992; Cleak and Eston, 1992; Howell et al., 1993; Sayers and Clarkson, 2001) and, more recently, dynamic isokinetic force (Golden and Dudley, 1992; Eston et al., 1996; Hortobagyi et al., 1998; Byrne et al., 2001; Byrne and Eston, 2002). Much less is known about the ability of eccentrically exercised muscle to sustain isometric force (Davies and White, 1981; Balnave and Thompson, 1993; Behm et al., 2001) or the ability to generate and maintain maximal power output (Sargeant and Dolan, 1987).

Fatigue has been defined as ‘any exercise-induced reduction in force generating capacity’ (Gandevia, 2001). The long-lasting reduction in force-generating capacity that results from eccentric exercise is more
suitably viewed as muscle weakness rather than muscle fatigue, since it is not an acute effect that is reversed by rest. Gandevia (2001) stated that the maximal force-generating capacity of muscles starts to decline once exercise commences, so that fatigue really begins almost at the onset of exercise and develops progressively before the muscles fail to perform the required task. This aspect of muscle function has been studied infrequently after eccentric exercise. Davies and White (1981) investigated the ability of the triceps surae muscle group to maintain isometric force after eccentric exercise and reported the musculature to be weaker but no more fatiguable, whereas Balnave and Thompson (1993) reported that the knee extensors were weaker but, surprisingly, less fatiguable after eccentric exercise. Both studies used an electrically elicited isometric fatigue test consisting of trains of stimuli at 20 Hz, lasting 300 ms and repeated every second for 2 min. Using voluntary muscle actions, Behm et al. (2001) reported a significant reduction in endurance time of the elbow flexors when isometric contractions were maintained at 50% of the pre-exercise maximal voluntary contraction (MVC) force. No previous studies have investigated the ability of eccentrically exercised muscle to maintain maximal voluntary force. The first part of this study assessed the ability of the knee extensors to maintain force during sustained isometric MVCs of 60 s duration after eccentric exercise. Evidence suggests that muscle fatigue during MVCs is length-dependent (Fitch and McComas, 1985; McKenzie and Gandevia, 1987), with muscle demonstrating a greater ability to maintain force and appear fatigue-resistant when tested at a shortened rather than an optimal muscle length for force production. To investigate this phenomenon further, MVCs were performed at an optimal muscle length and at a shortened length before and for up to 7 days after eccentric exercise.

To complement the isolated knee extensor experiments and in an attempt to make the results of the study more applicable to sport (Jones, 1993), we examined the ability of eccentrically exercised muscle to generate and maintain maximal power output. It is somewhat surprising that performance in maximal intensity sprint exercise, being essential to performance in many sports, has received almost no attention after eccentric exercise. In highlighting the lack of research in this area, Clarkson and Newham (1995) stated that it is not known if the time course of recovery for power generation is the same as that for isometric strength. Previously, only Sargeant and Dolan (1987) have investigated the ability of eccentrically exercised muscle to generate maximal power output, reporting an immediate, significant and sustained reduction in short-term power output, measured concentrically on an isokinetic cycle ergometer. This reduction persisted for 4 days and was accompanied by a reduction in maximal isometric strength of a similar magnitude.

Several events that occur during and after eccentric exercise-induced muscle damage are likely to have a large impact on maximal dynamic performance. Evidence suggests that type II fibres are selectively damaged during eccentric exercise (Friden et al., 1983; Jones et al., 1986) and a prolonged decrease in muscle glycogen content is commonly observed (O’Reilly et al., 1987; Costill et al., 1990), which is especially pronounced in type II fibres (Asp et al., 1998). The rapid and marked rise and subsequent decline in force and power output during maximal intensity exercise, as observed during sustained MVCs or maximal cycling, is thought to be closely related to the activation and rapid fatigue of type II fibres (Nevill et al., 1996). As the generation of power output by muscle shortening is more energetically demanding and requires higher rates of adenosine triphosphate (ATP) regeneration than isometric force generation (Woledge et al., 1985), the possibility exists that more dramatic performance decrements will be observed for power-generating ability than those commonly observed for the generation of isometric force. Therefore, in the second part of this study, we examined the effect of eccentric exercise on the ability to generate maximal isometric force and peak power (i.e. first few seconds) and investigated the rate of fatigue during a 60 s isometric MVC and during a 30 s maximal-intensity Wingate cycle test.

**Methods**

**Participants and design**

Seven healthy participants, five males and two females (age 22.6 ± 4.4 years, height 1.78 ± 0.08 m, mass 75.7 ± 11.7 kg; mean ± s), were involved in the study, all of whom were moderately active, but had not participated in any resistance training of the lower limbs in the 6 months before the study, and none had any musculoskeletal defects. Each participant provided written informed consent to take part in the study, which had previously been approved by the School ethics committee. The muscle group studied was the knee extensors. Each participant was evaluated for each criterion measure before, 1 h after and 1, 2, 3 and 7 days after a bout of eccentrically biased resistance exercise, chosen to induce the symptoms of exercise-induced muscle damage.

**Isometric strength at 40° and 80° knee flexion**

Isometric strength and fatigue were measured for the knee extensors of the non-dominant limb using a Kin-Com (500H, Chattanooga, Chattanooga, TN, USA).
isokinetic dynamometer. Participants performed isometric MVCs of the knee extensors at 40° and 80° knee flexion, corresponding to a shortened and optimal muscle length for torque generation, respectively. Newham et al. (1991) have previously reported that 80° knee flexion corresponds to the optimal angle for torque generation in this muscle group. The 40° joint angle was chosen to represent short muscle length, as it is a similar relative position to the muscle length used by McKenzie and Gandevia (1987), who reported less fatigue in the elbow flexors at short versus optimal muscle length. The testing positions were obtained by entering full knee extension (0°) as a reference value into the Kin-Com visual display. The reproducibility of this method was checked on each test occasion by noting the Kin-Com angle display when the lever arm was at true 90° (determined by spirit level). The pre-test angle display at true 90° was used as the criterion. If any difference was noted, the process was repeated until the criterion was achieved. Three submaximal and one maximal practice repetitions acted as warm-up at each test position. Three MVCs of 3 s duration, presented in a mixed order, were performed at each joint angle with a 60 s rest between repetitions. The highest peak torque from the three muscle actions was used as the criterion score for short and optimal muscle length, respectively.

Isometric fatigue at 40° and 80° knee flexion

Fatigue was assessed by having the participants perform a 60 s MVC at both 40° and 80° knee flexion. A target of 100% of the current MVC was entered into the Kin-Com visual display, which gave real-time feedback of the participants’ torque production and the target torque. The test started when the participants achieved the 100% MVC target torque. Tests at 40° and 80° were presented in a mixed order and were separated by 20 min of recovery. Torque was recorded each second during the 60 s MVC.

Fatigue was quantified as the slope of a linear regression line, fitted to the 60 data points. The slope of the line or regression coefficient (b) represents the rate of change in y with a unit change in x. In this study, for every unit change in x (time), y (torque) changed by b. Since torque declined over time during the fatigue test, b always took a negative value. As such, the less negative the value, the less fatiguable the knee extensors were.

The Wingate test

The Wingate test is a maximal-intensity exercise test used to assess power output of the legs. The sensitivity and reliability of the test have previously been examined (Bar-Or, 1987). Before testing, a fingertip blood sample was taken using a softclix lancet and capillary tube and was analysed for lactate using an Accusport analyser (Boehringer Mannheim, Lewes, UK). The participants performed a 5 min warm-up on a cycle ergometer at 100 W, which included a flat-out sprint at 3 min for 5 s, followed by 5 min of rest. The participants were then transferred to the test ergometer (Monark 814 E). Seat height was adjusted for comfort and was recorded, toe clips were secured, the resistive load was attached and a restraining harness was positioned to prevent the participants rising from the seat. The resistive load corresponded to 10% and 8% of body mass for males and females, respectively. The participants began pedalling at 50–60 rev·min⁻¹ with the external load supported. Upon the command ‘3, 2, 1, GO’, the load was applied abruptly, the participants pedalled flat out for 30 s, and power output data, corrected for the moment of inertia of the flywheel (Lakomy, 1986), was logged by computer for 30 s. The participants then performed a warm-down, which consisted of 2 min of cycling at 100 W. A post-exercise blood lactate sample was taken at 3 min. Peak power was quantified as the highest power output in the first 5 s of the test. Regression analysis was performed on the 30 data points and the regression coefficient (b) was used to quantify fatigue.

The rationale for using the regression coefficient to quantify fatigue in MVC and Wingate tests is that the calculation takes into account every data point rather than a simple percentage decline based on the highest and lowest data points. Balnave and Thompson (1993) also used this method, which allows for a direct comparison between the two studies. In addition, there appears to be no standard or widely accepted method of quantifying fatigue in the muscle function literature.

Creatine kinase measurement

Plasma creatine kinase activity was determined from a fingertip blood sample. A warm fingertip was cleaned with a sterile alcohol swab and allowed to dry. Capillary puncture was made with a softclix lancet and a sample of whole fresh blood (30 μl) was pipetted from a capillary tube onto the test strip and analysed for creatine kinase activity using a colorometric assay procedure (Reflotron, Boehringer Mannheim, Lewes, UK). This system uses a plasma separation principle, which is incorporated in the reagent carrier on the test strip.

Muscle-damaging exercise

Each participant performed 10 sets of 10 repetitions of the eccentric phase of the barbell squat exercise. The load on the barbell corresponded to 80% of the participant’s concentric one-repetition maximum. They started the movement with the barbell resting on their shoulders, body erect, legs fully extended (knee = 180°)
and the toes pointing forward. The movement consisted of an eccentric action of the knee extensors to lower the barbell to a knee angle of approximately 90°. The barbell was then raised to the starting position by two individuals acting as spotters. Two minutes of rest separated each set. Since all participants were unaccustomed to this form of exercise, the load was moderate to ensure correct technique and the movement was slow and careful to avoid injury. The volume of exercise was high and the amplitude of the stretch large, since these parameters have been shown to induce muscle damage (Newham et al., 1988). Furthermore, this form of exercise has previously been shown to produce long-lasting decrements in muscle function (Byrne and Eston, 2002).

Statistical analysis

Isometric strength and fatigue data were analysed by two separate, two-factor (time and angle) repeated-measures analyses of variance. A comparison of isometric strength and Wingate peak power was analysed by a two-factor (time and mode) repeated-measures analysis of variance. Wingate fatigue, creatine kinase activity and post-Wingate blood lactate concentration were analysed using separate single-factor repeated-measures analyses of variance. The assumption of sphericity was tested by the Mauchly test of sphericity. Any violations of this assumption were corrected using the Greenhouse-Geisser adjustment to raise the critical value of $F$, as indicated by $(GG)$. To determine the regression coefficient ($b$) to quantify fatigue, individual regression analyses were conducted on performances during the isometric 60 s MVC tests and the Wingate 30 s test. Statistical significance was set at $P < 0.05$. Post-hoc tests were performed with paired-sample $t$-tests using the Bonferroni correction technique.

Results

Isometric strength at 40° and 80° knee flexion

Absolute values for isometric peak torque at 40° and 80° were 302 ± 19 N·m (mean ± sₓ) and 495 ± 33 N·m, respectively. There was a highly significant main effect for time ($F_{5,30} = 50.8$, $P < 0.001$) but no difference in strength loss between joint angles ($F_{1,6} = 2.5$, $P > 0.05$). Although the extent of strength loss was clearly length-dependent 1 h after exercise (see Fig. 1), the time by angle interaction was not significant ($F_{5,30} = 1.0$, $P > 0.05$). A disproportionate loss of relative strength at short versus optimal muscle length, either acute or long-lasting, has previously been observed (Saxton and Donnelly, 1996; Byrne et al., 2001) and may represent a change in the length–tension relationship of the muscles due to a non-uniform distribution of sarcomere length change during eccentric exercise (for a review, see Morgan and Allen, 1999). Strength was reduced 1 h after exercise and remained so for 7 days (Fig. 1). Strength was approximately 35% lower 1 h after exercise ($P < 0.05$), 28% lower after 1 day ($P < 0.05$), 20% lower after 2 days ($P < 0.05$), 15% lower after 3 days ($P < 0.05$) and 5% lower after 7 days ($P < 0.05$).

Isometric fatigue at 40° and 80° knee flexion

Significant main effects were revealed for time ($F_{5,30} = 23.2$, $P < 0.001$) and angle ($F_{1,6} = 17.0$, $P < 0.01$) on the rate of fatigue. The results also revealed a significant interaction of time and angle ($F_{5,30} = 6.6$, $P < 0.001$). Before damaging exercise, the knee extensors were significantly less fatiguable ($P < 0.05$) at 40° ($b = -2.39 ± 0.26$) than at 80° ($b = -5.50 ± 0.72$). After eccentric exercise, the knee extensors became less fatiguable; however, the magnitude and time course of this effect was dependent on joint angle (see Fig. 2). At 80°, the knee extensors were less fatiguable ($P < 0.05$) after 1 h, 1 day and 3 days. At 40°, the knee extensors were less fatiguable ($P < 0.05$) after 1 day only.

Figure 3 shows the response of one participant to the isometric fatigue tests before eccentric exercise and 1 day after. Before eccentric exercise, there was a greater initial isometric force generation and a greater rate of fatigue at the optimal versus shortened muscle length, as evidenced by the steeper slope of the regression line at 80° ($b = -7.68$) than at 40° ($b = -1.84$) knee flexion
One day after eccentric exercise, the initial isometric force was reduced at both 40° and 80° (Fig. 3b). The rate of fatigue was still greater at 80° ($b = -4.58$) than at 40° ($b = -1.40$); however, at both 40° and 80°, the knee extensors were less fatiguable than before eccentric exercise, which was most evident at 80°. Thus, after eccentric exercise, the knee extensors became weaker but less fatiguable – that is, there was an improved ability to maintain force. Task failure was a common observation during the last 15 s of the tests after eccentric exercise. This is clearly evident in Fig. 3b, where force intermittently dropped to zero as the participant could no longer continue the exercise. The participant’s ability to quickly overcome this fatigue and generate force suggests that central fatigue was responsible.

Isometric strength at 80° knee flexion versus Wingate peak power

A significant main effect was shown for time ($F_{5,30} = 20.4, P < 0.01$) and, although the main effect for mode was not significant ($F_{1,6} = 2.6, P > 0.05$), the interaction of time and mode was ($F_{5,30} = 4.8, P < 0.05$). Figure 4 illustrates the effect of eccentric exercise on the ability to generate isometric force and Wingate peak power. The interaction of time and mode was the result of initial differences (1 h after exercise) of the effect of eccentric exercise on isometric force (30% reduction) and Wingate peak power (13% reduction) and subsequent differences in the pattern of recovery. Isometric force followed a linear recovery pattern at day 1 (26%) and day 2 (19%), whereas peak power suffered further decrements at day 1 (18%) and day 2 (16%) before starting to recover.

Fatigue during the Wingate test was reduced at all instants in time, but only significantly so 1 day after exercise ($F_{5,30} = 5.3, P < 0.05$). Values for Wingate fatigue were $-12.75 \pm 2.26$ before eccentric exercise, $-10.0 \pm 2.03$ at 1 h after, $-8.33 \pm 2.01$ at 1 day after, $-8.24 \pm 1.88$ at 2 days after, $-8.97 \pm 1.72$ at 3 days after and $-10.60 \pm 1.95$ at 7 days after exercise. Figure 5
displays the response of one participant to the Wingate test before and 1 day after eccentric exercise. Similar to the isometric responses in Fig. 3, the most striking difference between pre- and post-exercise responses is the reduction in force-generating capacity at the start of exercise. Power output could be well maintained, albeit at lower than pre-exercise values. Post-Wingate blood lactate concentration did not change significantly ($F_{5,30} = 1.2, P > 0.05$) after eccentric exercise. The post-Wingate blood lactate concentrations were $10.6 \pm 0.6$ mmol·l$^{-1}$ before eccentric exercise and $11.1 \pm 1.2, 9.6 \pm 1.0, 9.7 \pm 1.0, 9.1 \pm 0.7$ and $10.6 \pm 1.2$ mmol·l$^{-1}$ 1 h, 1 day, 2 days, 3 days and 7 days after exercise, respectively.

**Discussion**

**Evidence of muscle damage**

The eccentric exercise protocol produced changes in all muscle function variables. Isometric strength at 80° and Wingate peak power were reduced by 30% and 13% 1 h after exercise, respectively, and only returned to pre-exercise values 7 days later. During sustained maximal voluntary contractions and the Wingate test, the knee extensors were significantly less fatiguable for 3 days and 1 day after eccentric exercise, respectively. Creatine kinase activity was significantly elevated 1 h after exercise, reached a peak at day 1 and then gradually returned to baseline values. The immediate and sustained loss of muscle function and the increase in blood levels of creatine kinase are common indirect markers of exercise-induced muscle damage (Warren et al., 1999).

**Potential mechanisms of muscle function impairment**

The results of the isometric and dynamic parts of the study suggest that an inability to generate high force and power at the start of exercise is the common underlying mechanism responsible for the observed changes in strength, power and fatigue after eccentric exercise. The inability to generate an initial high force and power will depend on events that occur at or distal to the neuromuscular junction (peripheral fatigue) and on a failure to activate the muscles voluntarily (central fatigue).
A failure to activate intact force-generating structures because of excitation–contraction coupling impairment and disruption of force-generating or transmitting structures are considered to be the two main peripheral mechanisms responsible for the loss of maximal voluntary force after eccentric exercise (for reviews, see Morgan and Allen, 1999; Warren et al., 2001). Although full voluntary activation has been demonstrated during brief isometric MVCs after eccentric exercise (Rutherford et al., 1986; Saxton and Donnelly, 1996), it is doubtful whether this is true for sustained isometric and dynamic muscle actions. Bigland-Ritchie et al. (1978) reported that central fatigue consistently accounted for up to 30% of the force loss during sustained 60 s MVCs in fresh muscle and with well-motivated participants. Whether the central contribution to fatigue increased after eccentric exercise could not be determined; however, the increased incidence of task failure (see Fig. 3b) would suggest that this is the case. The relative contribution of central and peripheral mechanisms to the changes in muscle function observed in the present study could not be determined, although a discussion of potential mechanisms is warranted.

Selective type II fibre damage

The knee extensors were significantly less fatiguable during MVCs and Wingate tests for 3 days and 1 day after eccentric exercise, respectively. This is in line with the findings of Balnave and Thompson (1993), who raised the question: How can a muscle be less fatiguable after a bout of prolonged eccentric exercise than it was before? Research into fatigue mechanisms during maximal exercise and selective fibre changes that occur in eccentrically exercised muscle help to answer this question. The loss of force during isometric muscle actions (Soderlund et al., 1992) and power output during maximal cycling (Nevill et al., 1996) has been attributed to declining rates of phosphocreatine and glycogen utilization in type II muscle fibres. Evidence suggests that type II fibres are selectively damaged during eccentric exercise (Friden et al., 1983; Jones et al., 1986) and this is possibly due to selective type II recruitment (Nardone et al., 1989; Enoka, 1996; Asp et al., 1998). If type II fibre function is preferentially affected by eccentric exercise, for example because of excitation–contraction coupling impairment, damage to the force-generating or transmitting structures, or selective glycogen depletion, then the contribution of this fibre type to the post-eccentric exercise fatigue responses will be diminished. As such, the fatigue responses will be without the marked rise and rapid decline in force and power that is associated with the activation and rapid fatigue of type II fibres and will, therefore, appear fatigue-resistant. Such a mechanism could explain the observed inability of eccentrically exercised muscle to generate an initial high force and power and, therefore, appear less fatiguable.

Central fatigue

The results revealed an immediate and long-lasting reduction in the ability to generate isometric force and dynamic peak power. One hour after eccentric exercise, the ability to generate isometric force was affected to a greater extent (69.8 ± 2.6% of pre-exercise values) than Wingate peak power (87.3 ± 1.7%). The two parameters then followed different temporal patterns of recovery; force recovered linearly, whereas peak power declined further at days 1 and 2 before starting to recover. Wingate peak power eventually recovered with a similar time course to that of isometric strength. This bimodal pattern of muscle function recovery is in line with the results of previous studies (Sargeant and Dolan, 1987; MacIntyre et al., 1996) and has been attributed to the inflammatory response that accompanies muscle damage (Sayers and Clarkson, 2001). In addition to selective damage of type II fibres, reductions in central drive resulting from voluntary conscious or subconscious reflex mechanisms could also explain the reductions in peak power and the temporal pattern of recovery. The reductions in peak power following the initial (1 h) decrement occurred during the typical arrival and peak (24–48 h) of delayed-onset muscle soreness (Clarkson et al., 1992), which also accompanies muscle damage. Voluntary effort may have been reduced at these times because of painful sensations or, alternatively, afferent signals from the active muscles and tendons may have produced reflex inhibition. An additional factor to consider is that measures of motor control, such as force and position sense, are impaired after eccentric exercise (Saxton et al., 1995; Brockett et al., 1997) and are more likely to affect a complex dynamic motor task such as cycling to a greater extent than a simple static task such as knee extension.

Metabolic dysfunction

The role of metabolic dysfunction after eccentric exercise is unclear, but defects are likely to affect force-generating capacity. The rate of energy release through anaerobic sources is of critical importance for the development and maintenance of high force and power output during maximal-intensity exercise (Nevill et al., 1996). Reductions in high-energy phosphate have been reported to occur 24 h after eccentric exercise in rats (van der Meulen et al., 1992) and delayed muscle glycogen replenishment after damaging exercise in humans is well documented (O’Reilly et al., 1987; Costill et al., 1990; Asp et al., 1998). It is possible that reduced
concentrations of ATP and phosphocreatine, as well as glycogen depletion, in the days after eccentric exercise compromised the ability to generate and maintain force and power. However, evidence from recent animal (Warren et al., 2000) and human (Rawson et al., 2001) studies suggests that increasing phosphocreatine through creatine supplementation has no effect on the magnitude of muscle function impairment or the time course of recovery after eccentric exercise. No changes in post-Wingate blood lactate concentrations were observed in the present study, suggesting that glycolysis was not impaired. However, the extrapolation of single post-exercise blood lactate measurements to underlying muscle metabolism is extremely tentative. Although glycogen depletion would be expected to reduce high-intensity exercise capacity, at present the role of metabolic dysfunction in determining muscle function after eccentric exercise is unclear.

Fatigue due to testing

Finally, it is possible that not all of the performance decrements observed in this study can be attributed to the eccentric exercise protocol. Fatigue may have occurred due to the volume of muscle function testing over consecutive days. The effect of fatigue due to testing could not be determined because of the absence of a control group.

Conclusions

Eccentric exercise resulted in a reduced ability of the knee extensors to generate rather than maintain an initial high force and power output during a sustained 60 s MVC and a 30 s Wingate cycle test. Isometric strength and Wingate peak power, although sharing a similar time course of recovery, differed in their pattern of recovery. Strength recovered linearly after the initial insult, whereas Wingate peak power declined further in the following days before starting to recover. The observed changes in muscle function are consistent with the proposition that eccentric exercise results in selective dysfunction of type II muscle fibres.

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References


