**Muscle Substrate Utilization and Lactate Production During Weightlifting**


**Abstract/Résumé**

Biopsies (biceps) were examined in 8 bodybuilders across a typical arm-curl training session (80% 1-RM). [PCr] and [glycogen] decreased 62 and 12% after 1 set (n = 4), and 50 and 24% after 3 sets (n = 4). [Lactate] was 91 and 118 mmol · kg⁻¹, respectively, after 1 and 3 sets. Fatigue was probably partially caused by decreased [PCr] and increased [H⁺] (first set) and by increased [H⁺] in subsequent sets.

On a examiné des biopsies (biceps) prises de 8 culturistes à travers une séance typique de formation en flexion des avant-bras (80% 1-RM). La [PCr] et la [glycogène] ont diminué de 62 et de 12% après 1 séance (n = 4), et de 50 et 24% après 3 séances (n = 4). Le [lactate] était de 91 mmol · kg⁻¹ après 1 séance et de 118 après 3 séances. La fatigue découlaient probablement en partie de l'amoindrissement en [PCr] et de l'augmentation en [H⁺] (première séance) et de l'augmentation en [H⁺] (séances suivantes).

**Introduction**

A typical exercise session for many athletes involved in a heavy resistance training program includes 3 or more sets of a given exercise to failure with a resistance equivalent to approximately 80% of their maximal strength (1-RM). At this intensity, an individual can complete approximately 12 repetitions on the first set, 9 or
10 on the second set, 7 or 8 on the third set, and so on (Sale and MacDougall, 1981). The mechanisms that result in muscular fatigue over a given set of such exercise are poorly understood and may involve decreased neural drive (Bigland-Ritchie et al., 1978), failure at the excitation-contraction coupling site (Bigland-Ritchie, 1984; Enoka and Stuart, 1992), or certain metabolic consequences such as a decrease in muscle [PCr] or an increase in muscle lactate and [H+] (Chasiotis et al., 1983; Hirvonen et al., 1987; Hultman et al., 1990; Spriet et al., 1987).

Since such exercise is dependent on maximal or near maximal ATP production rates from both PCr hydrolysis and anaerobic glycogenolysis, the present study was undertaken in order to assess the possible contribution of depleted muscle PCr and/or increased muscle lactate (H+) as the cause of fatigue during such exercise. Although glycogen depletion and lactate production have been examined following 3 and 6 sets of leg extension exercise (Robergs et al., 1991), following ~9 sets of concentric-only knee extension exercise (Pascoe et al., 1993), and following 20 sets of varied leg exercises (Tesch et al., 1986), to our knowledge there have been no investigations of substrate utilization and muscle lactate production across a single set of heavy resistance exercise.

Consequently, our purpose was to investigate PCr hydrolysis, glycogenolysis, and muscle lactate production across a single set of arm curl exercise at 80% of 1-RM and after 3 sets of such exercise by experienced bodybuilders.

Methods

Eight healthy men who had participated in resistance training (bodybuilding) for the previous 5 years (±1 yr) volunteered to participate in the study. Mean (±SD) age, height, and weight were, respectively, 24 ± 2 yrs, 178 ± 4 cm, and 79 ± 7 kg. After an explanation was given of the purpose of the study, along with procedures and possible risks, written consent was obtained. The study was approved by the Human Research Ethics Committee of McMaster University.

Each subject’s 1-RM for a single-arm seated elbow flexion (arm curl) was predetermined on a custom-made, padded curl bench that supported the upper arm. Free weight plates were loaded onto the device and the totals were recorded to the nearest 2 kg. Subjects were strapped into the seated position and seat height was kept consistent for each individual throughout the study. All exercise was performed by the dominant arm. After 30 min of recovery, subjects were asked to perform repetitions to failure with a resistance equivalent to 80% of this value in order to verify that the resistance selected was appropriate. At this intensity, it was found that all subjects were able to complete 11 to 13 reps. Partial repetitions were not recorded.

Four to 6 days later, subjects were asked to refrain from any training of their arms for 48 hrs before reporting to the Human Performance Laboratory at 30-min intervals. They were randomly assigned to Group A or Group B, with 4 in each group. Needle biopsies were taken under local anesthesia and with manual suction from biceps brachii of the control arm at rest, and an incision was made in the exercise arm and sealed with a steri-strip.

Subjects in Group A performed a single set of arm curl exercise to failure at 80% 1-RM as previously determined, with verbal encouragement being given throughout the set. At the point of failure, the steri-strip was immediately removed,
the biopsy sample was taken, and the needle was plunged directly into liquid nitrogen. The duration of the exercise bout and the elapsed time before final freezing of the sample were timed by stopwatch to the nearest second. Procedures were identical for the subjects in Group B, with the exception that they undertook 3 sets to failure with 3 min of recovery between each, and the biopsy sample was taken immediately after the third set. In both groups, a capillary blood sample was taken by finger-puncture before and 5 min after completion of exercise and analyzed for whole-blood lactate concentration (YSI lactate analyzer, Yellow Springs, OH).

The frozen tissue samples were removed from the biopsy needles and stored in liquid nitrogen until analysis. Samples were dissected free of visible blood, fat, and connective tissue, weighed, freeze-dried, and weighed again. They were then added to test tubes containing chilled 1.5-M perchloric acid, left on ice for 30 min, and centrifuged at 4°C. The supernatant was neutralized to pH 7.0 with 2.3 M KCO₃ and again centrifuged. The neutralized extract was assayed for concentrations of adenosine triphosphate (ATP), phosphocreatine (PCr), lactate, and glycogen via enzymatic fluorometric techniques (Bergmeyer, 1965; Lowry and Passonneau, 1972). Muscle metabolite concentrations are expressed in mmol·kg⁻¹ dry weight.

Statistical analyses were conducted using a 2-group-by-2-condition (control and postexercise) ANOVA with repeated measures on the second factor. Differences between groups were detected via Tukey A post hoc analysis when significant F ratios (p < 0.05) were found. Differences between pre- and postexercise blood lactate concentrations were examined via Student t-test with an alpha level set at p = 0.05. Data are presented as means ± SD.

Results

Group A completed an average of 12.0 ± 4 reps in their single set before failure, over a total exercise time of 37 ± 3 sec. Group B completed a total of 11.7 ± 2 reps on their first set (36 sec), 9.2 ± 2 on their second set (33 sec), and 7.2 ± 1 on their third set (27 sec), for a total exercise time of 96 ± 17 sec. An average of 17 ± 5 sec elapsed between termination of exercise and final freezing of the tissue sample.

Individual muscle concentrations of ATP and PCr are presented in Figure 1 and individual concentrations of glycogen and lactate are presented in Figure 2 for both Group A (1 set) and Group B (3 sets). ATP concentration was slightly lower in both groups immediately after exercise, but this was not statistically significant. The PCr concentration was significantly lower in Group A by a mean value of 62%, and in Group B by 50%, compared to preexercise values. After 1 set, muscle glycogen concentration was reduced by approximately 12% in Group A, but this was not statistically significant. In contrast, after 3 sets, Group B had a significant (p < 0.05) reduction in muscle glycogen by a mean value of 24%. After 1 set, muscle lactate increased dramatically from 7.3 to a mean value of 91.4 mmol·kg⁻¹. After 3 sets, mean lactate concentration was 118 mmol·kg⁻¹ compared to 6.2 mmol·kg⁻¹ before exercise. With the exception of muscle glycogen concentration following exercise, there were no significant differences between groups.

Whole-blood lactate increased significantly from a mean of 1.7 ± 0.8 to 3.5 ± 0.7 mmol·L⁻¹ following 1 set of exercise, and from 1.7 ± 0.7 to 4.7 ± 0.8 mmol·L⁻¹ following 3 sets.
Figure 1. Individual muscle concentrations of phosphocreatine and adenosine triphosphate before and after 1 set (Group A, n = 4) and 3 sets (Group B, n = 4) of arm-curl exercise at 80% 1-RM. In both groups the change in phosphocreatine was statistically significant ($p < 0.05$), but the change in adenosine triphosphate was not.

Figure 2. Individual muscle concentrations of glycogen and lactate before and after 1 set (Group A, n = 4) and 3 sets (Group B, n = 4) of arm-curl exercise at 80% 1-RM. The decrease in muscle glycogen concentration was statistically significant following 3 sets of exercise, but not following 1 set. The increase in muscle lactate concentration was statistically significant ($p < 0.05$) in both groups.

Discussion

The performance data confirm that, when experienced resistance-trained athletes perform weightlifting exercise which has both a concentric (lifting) component and an eccentric (lowering) component at a resistance equivalent to 80% 1-RM, fatigue occurs after approximately 12 reps. In addition, fatigue is cumulative in that, as subsequent sets are attempted with the same resistance, there is a progressive
decrease in the number of repetitions that can be completed in each set, following 3 min of rest between sets.

Our finding that muscle ATP concentration was not significantly reduced at the point of fatigue in either group is consistent with a number of studies which have verified that skeletal muscle is, for the most part, able to preserve its ATP content even during intensive exercise to fatigue (Fitts, 1994; Fitts and Holloszy, 1976; Nevill et al., 1989). Furthermore, in the present study, fatigue (as indicated by the inability to complete the next repetition) can be considered as occurring when muscle-force-generating capacity has declined by 20%. Thus, at this point the muscle cannot be considered “exhausted” since it is still capable of generating a force up to 80% of its maximal capacity.

The 62% decline in PCr following 1 set of exercise (requiring ~37 sec) is similar to the 64% decrement observed by Cheetham et al. (1986) following a maximal 30-sec sprint on a nonmotorized treadmill. Since we did not occlude circulation to the arm following exercise, it is possible that significant ATP and PCr resynthesis could have occurred between the termination of contraction and extraction of the muscle sample (Harris et al., 1976; Sahlin et al., 1998). Thus it is probable that, at the point of fatigue, phosphagen concentration was even lower and may have been a cause for the reduction in force output. We interpret our finding—that PCr concentration following the third set did not differ from that of the subjects who performed only 1 set—as indicating that PCr concentration was probably restored during the 3-min recovery period between sets (Harris et al., 1976; Sahlin et al., 1998). Moreover, the accumulated fatigue as sets progressed was probably not due to PCr depletion since, if anything, [PCr] tended to be higher upon completion of the third set of exercise.

Although blood lactate concentration increased by three- to fourfold following exercise, such values are considerably less than the seven- to tenfold increases which are found following 30 sec of exhaustive cycling (Bogdanis et al., 1996; McCartney et al., 1986). We interpret this difference as being largely due to the relatively small muscle mass which is active with the single-arm curl exercise. Muscle lactate concentrations following a single set, although high (mean value = 91.4 mmol·kg⁻¹), were not as high as the 110–135 mmol·kg⁻¹ concentrations (Bogdanis et al., 1996; McCartney et al., 1986) that are typically reported following 30 sec of exhaustive cycling (Wingate test). We interpret this difference as being due to the fact that muscle fatigue was noted in the present study when voluntary force had decreased by 20%, whereas percent fatigue is typically 40% following a Wingate test (Odland et al., 1997). During exercise at 80% MVC, blood flow to biceps brachii can be considered completely occluded by the increase in intramuscular pressure (Bonde-Petersen et al., 1975), and thus little or no lactate would have escaped from the muscle during the set. With resumption of blood flow immediately after exercise, however, there was probably an efflux of lactate (and H⁺) before excision of the biopsy, suggesting even higher values at the point of fatigue.

The 24% depletion of muscle glycogen that we observed following 3 sets of exercise is similar to the 20% depletion reported by Robergs following 3 sets of knee extension exercise at 70% 1-RM (Robergs et al., 1991). In the present study, glycolysis rate over the first set was approximately 1.24 mmol·kg DW⁻¹·sec⁻¹ and 0.96 mmol·kg DW⁻¹·sec⁻¹ when averaged over 3 sets, suggesting a slowing of glycolysis as the sets continued. Such a finding has also been observed with repeated 30-sec exercise bouts on a cycle ergometer (McCartney et
al., 1986) and may be largely attributable to the increasing inhibitory effects of [H+] on glycogenolysis as exercise continues (Chasiotis et al., 1983).

Although the 3 minutes of recovery between sets was probably adequate for complete restoration of muscle PCR (Harris et al., 1976; Sahlin et al., 1998), it would not have been long enough for complete removal of muscle lactate (Bogdanis et al., 1996). As a result, each subsequent set would have begun with an elevated initial concentration of lactate and H+.

In order for the muscle to repeat the high force contractions in the present study, it must be able to maintain high ATP production rates from both PCR hydrolysis and anaerobic glycogenolysis. Thus fatigue would occur as a result of either a depletion of muscle PCR stores or a decrease in maximal glycogenolytic rate. It is also probable that fatigue results from the combined effects of several factors. In the present study we chose to evaluate only two possible contributors, phosphagen depletion and increased lactate (H+); we recognize that additional factors may have contributed to the decline in force-generating capacity which occurs with this form of exercise. It should also be noted that our data reflect muscle homogenate averages only. It is possible that the extent of phosphagen depletion and/or lactate production could have been exaggerated and the rate of PCR resynthesis slower (Söderlund and Hultman, 1991) in the high-force-producing type II units. When considered along with the possibility that these units are more susceptible to fatigue for a given level of phosphagen depletion or increase in [H+] (Stephenson et al., 1998), one might speculate that some of these units may have been the first to fatigue and thus have a major effect on force-generating capacity.

We interpret our findings as indicating that fatigue on the first set of weightlifting exercise at 80% 1-RM was probably at least partially due to both a decrease in [PCR] and the inhibitory effects of an increase in muscle lactate (H+). In subsequent sets, fatigue may be primarily due to the increase in muscle [H+]. In addition, it is apparent that glycogenolysis is the major energy delivery pathway for heavy resistance exercise and that significant glycogen depletion can occur with as few as 3 sets of exercise at 80% 1-RM.

References


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