Muscle glycogenolysis during differing intensities of weight-resistance exercise

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ROBERGS, ROBERT A., DAVID R. PEARSON, DAVID L. COSTILL, WILLIAM J. FINK, DAVID D. PASCOE, MICHAEL A. BENEDICT, CHARLES P. LAMBERT, AND JEFFREY J. ZACHWEIJA. *Muscle glycogenolysis during differing intensities of weight-resistance exercise.* J. Appl. Physiol. 70(4): 1700–1706, 1991.—Skeletal muscle glycogen metabolism was investigated in eight male subjects during and after six sets of 70% one repetition maximum (1 RM, I-70) and 35% 1 RM (I-35) intensity weight-resistance leg extension exercise. Total force application to the machine lever arm was determined via a strain gauge and computer interfaced system and was equated between trials. Compared with the I-70 trial, the I-35 trial was characterized by almost double the repetitions (13 ± 1 vs. 6 ± 0) and half the peak concentric torque for each repetition (12.4 ± 0.5 vs. 24.2 ± 1.0 Nm). After the sixth set, muscle glycogen degradation was similar between I-70 and I-35 trials (47.0 ± 6.6 and 46.6 ± 6.0 mmol/kg wet wt, respectively), as was muscle lactate accumulation (13.8 ± 0.7 and 16.7 ± 4.2 mmol/kg wet wt, respectively). After 2 h of passive recovery without caloric intake, muscle glycogen increased by 22.2 ± 6.8 and 14.2 ± 2.5 mmol/kg wet wt in the I-70 and I-35 trials, respectively. Optical absorbance measurement of periodic acid-Schiff-stained muscle sections after the 2 h of recovery revealed larger absorbance increases in fast-twitch than in slow-twitch fibers (0.119 ± 0.024 and 0.055 ± 0.024, $P = 0.02$). Data indicated that when external work was constant, the absolute amount of muscle glycogenolysis was the same regardless of the intensity of resistance exercise. Nevertheless the rate of glycogenolysis during the I-70 trial was approximately double that of the I-35 trial.

glycogen; lactate; glycogenesis; glycolysis

PAST RESEARCH evaluating muscle glycogenolysis and energy metabolism has predominantly been restricted to running or cycling (5, 7, 13, 19, 21, 24, 37). Of these two modes, cycling has been the preferred exercise because of the ease in quantifying power output and the relative subject accessibility for additional invasive and noninvasive data collection. This research has shown that as the intensity of exercise increases, muscle glycogenolysis increases and provides a greater supply of glucose residues for glycolysis and ATP production (15–17, 36). During high-intensity exercise, both muscle glycogenolysis and an increased glycolytic flux occur almost immediately (7, 15, 18, 22, 24, 37, 41).

Compared with cycling and running, limited research has been performed to evaluate muscle energy metabolism during resistance-type exercises (11, 27, 29, 31, 41). Early research indicated that short-duration weight-resistance exercise predominantly taxed the muscle's stores of ATP and creatine phosphate, with negligible glycogenolysis and a minor glycolytic energy contribution (31). However, recent research by MacDougall et al. (29) has indicated that muscle glycogen content can decrease by 25% (23 mmol/kg wet wt) in the biceps brachii after three sets of 10 weight-resistance repetitions to muscular failure. Also, Tesch et al. (41) reported that heavy weight-resistance exercise of the legs reduced muscle glycogen content of the vastus lateralis by 26% (42 mmol/kg wet wt). Results from the Tesch investigation also revealed that muscle lactate and glycolytic intermediates increased, indicating the flux of glycogen-derived glucose residues into the glycolytic pathway.

Despite this research, no data exist that compare muscle glycogenolysis during differing intensities of weight-resistance exercise. Therefore, it is unclear whether the positive relationship between exercise intensity and the magnitude of glycogenolysis is retained throughout a wide range of weight-resistance exercise intensities. Consequently, this study was designed to quantify the relationship between skeletal muscle glycogenolysis and exercise intensity when an indirect measure of force application was equated between trials. Two regimens of resistance exercise were used as follows: *1)* 70% one repetition maximum (1 RM, I-70) intensity and low repetition vs. 2) 35% 1 RM (I-35) intensity and higher-repetition exercise. In addition, the influence of these exercise regimens on specific fiber type glycogen content and the rate and magnitude of glycogen synthesis without caloric supplementation during 2 h of postexercise recovery were evaluated.

METHODS

Eight males currently participating in weight training and accustomed to leg extension resistance exercise served as the subjects for this research. Before the study, the procedures of the research were explained to each subject and a written informed consent was read and signed by each participant. All procedures were performed after approval by the University Internal Review Board.

Procedures. Each subject first reported to the laboratory for recording and measurement of standard physical and descriptive characteristics, familiarization with the leg extension machine and experimental protocol, and determination of the 1 RM of each leg (Fig. 1). To ensure

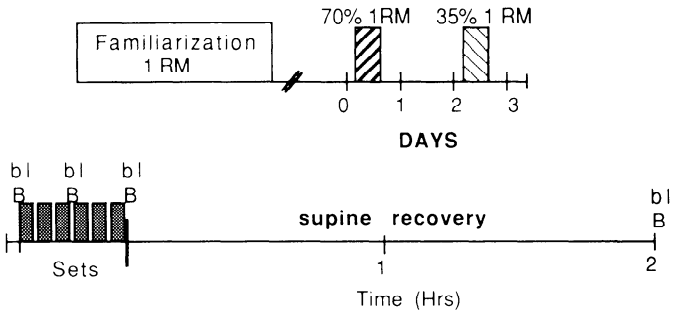

FIG. 1. Research protocol and timing of muscle biopsies (B) and blood sampling (bl).

stable preexercise glycogen values in an optimal range of 100–120 mmol/kg wet wt, subjects refrained from exercise for 2 days before testing and were advised of suitable varieties and quantities of food to provide approximately 4 g carbohydrate per kilogram body weight. The content of each subject's diet during the 2 days preceding the first trial was monitored by dietary recall and analyzed with a computer program (Nutricalc; 3.6 ± 0.4 g carbohydrate/kg body wt).

The leg extension exercise was performed on a model 4107 Eagle leg extension machine (Cybex). The machine was modified by incorporating a force transducer (strain gauge) in a shin pad attached to the leg extension lever arm. In addition, a potentiometer was attached to the rotational axis of the lever. Both the force transducer and potentiometer were electronically connected to an analog-to-digital converter and then interfaced with a computer (Apple II) and software. The computer software was programmed to calculate the angle of rotation, the total force accumulation, and the eccentric and concentric components of contraction duration and the accumulated force for each repetition. During leg extension exercise, the force applied to the strain gauge and lever arm was read and accumulated at approximately 39 readings/s and continually displayed on the computer screen. After each set of repetitions, values for each repetition and the mean kinetic data of the set were printed.

Force output from the strain gauge was calibrated using a regression equation generated by the placement of known weights on the strain gauge. The angle of rotation was calibrated with the use of a regression equation determined by lever movement through known angles via a goniometer. To provide a more comprehensible unit of work than force accumulation, external concentric work was calculated as the product of the weight of the stack and vertical distance lifted. The vertical distance was calculated from a regression between angular displacement and the vertical displacement of the stack. The correlation coefficients (r) from each regression calibration were linear (r = 1.0, respectively).

Each subject performed two trials (Fig. 1). The first trial (I-70) consisted of a high-intensity bout of one-legged leg extension exercise involving six sets of six repetitions at I-70 of the weakest leg (left). A 2-min rest interval separated each set. Based on preliminary testing, the 1 RM leg strength of each subject's dominant leg was consistently higher than the contralateral leg, yet these differences were not significant according to a paired t test analysis (52.1 ± 1.6 vs. 54.6 ± 1.5 kg; P = 0.23). Nevertheless, to ensure successful completion of the I-70 trial, each subject completed this trial with the dominant leg (right), with the use of the 1 RM for the left leg. Because the subjects were familiar with the exercise, the time for each repetition and the concentric and eccentric components were not regimented. However, subjects were instructed to lift as consistently as possible within and between trials.

The second trial (I-35) was performed on the following day with the contralateral leg and involved six sets of low-intensity (variable repetitions) leg extension exercise at I-35. The total number of repetitions per set could not be predetermined, because leg extension exercise continued in each set until the force accumulation matched that of the I-70 trial.

Muscle biopsies from the vastus lateralis were performed before each trial, after *sets 3* and *6* (final) of exercise, and after 2 h of recovery (2, 14). To prevent sampling of previously traumatized muscle, muscle biopsies were performed from two incisions located at least 3 cm apart, and repeated muscle biopsies from the same incision were angled proximally and distally (9). Muscle biopsies after the *sets 3* and *6* of exercise were performed with the subject remaining in the leg extension seat, whereas the biopsies at rest and after 2 h of recovery were performed with the subject in a supine position. No calories were ingested during the 2-h recovery, and subjects remained in a supine position throughout this period.

Blood samples from an antecubital vein were obtained before exercise, 2 min after *set 6*, and 2 h postexercise. Arterialized blood samples from a hyperemized ear lobe were obtained before exercise, after *sets 3* and *6*, and 2 h postexercise.

Analytical methods. Serum glucose concentrations were analyzed with the use of a YSI glucose analyzer (model 23A), and blood lactate concentrations were determined enzymatically from whole blood perchloric acid extracts (28). Changes in venous plasma volume were determined after measurement of blood hematocrit and hemoglobin concentrations with the use of the equations of Dill and Costill (10).

One large portion of each muscle specimen was frozen in liquid nitrogen within 30 s after collection and stored at −80°C. The remaining portion was mounted in tragacanth gum, frozen in isopentane cooled to the temperature of liquid nitrogen, and stored at −80°C for subsequent histological analyses. Muscle lactate was determined from wet weight perchloric acid extraction, assayed enzymatically, and expressed as millimoles per kilogram wet wt (28). Muscle glycogen content was determined by HCl hydrolysis and glucose assay and recorded as millimoles of glucosyl units per kilogram wet wt (28).

Muscle histochemistry was performed on serial sections of tissue (~10 μm) cut in a cryostat and consisted of myosin adenosinetriphosphatase (ATPase) staining (pH 4.3 and 4.6) and periodic acid-Schiff (PAS) staining. Skeletal muscle fiber types were determined from the myosin ATPase stain preincubated at pH 4.3, and each fiber was designated as fast or slow twitch by a dark or light stain intensity, as described by Peter et al. (35).

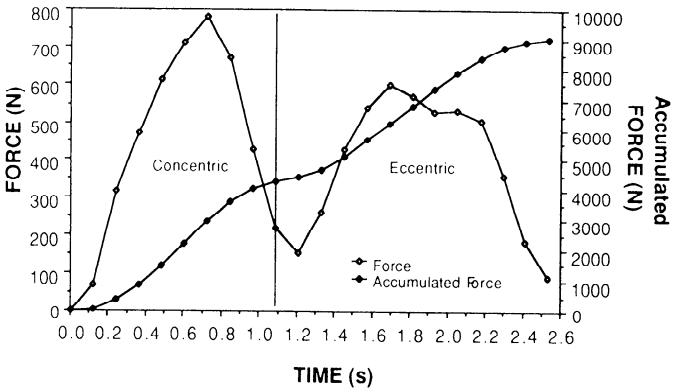

FIG. 2. Force application and force accumulation during concentric and eccentric phases of 1-leg extension repetition. Data are from *subject 4* during a simulated repetition from I-70 trial.

Fiber type glycogen content was determined photometrically from PAS-stained muscle sections (42, 43), without correction for mean fiber type areas. Because one subject's muscle sections did not react with the PAS stain, fiber type glycogen content was evaluated for seven subjects.

Statistical design. Muscle glycogen and lactate and blood lactate and glucose concentrations were analyzed by two-way analysis of variance (ANOVA) using trial and time as repeated factors (25). Comparisons between means of two different trials were analyzed by one-way ANOVA with the use of specific two-way ANOVA repeated factor error terms, and differences between the multiple means within a trial were assessed by the Tukey test (25). Fiber type differences in optical absorbance after data were combined from both trials were analyzed by two-way mixed-design ANOVA with fiber type as a between-group factor and time as a repeated factor. Muscle fiber type, additional fiber type PAS data, and simple pairwise comparisons were analyzed by paired *t* test. All data are expressed as means ± SE, and results of statistical tests were evaluated at the 0.05 confidence level.

RESULTS

The leg extension exercise involved concentric contractions at the knee against resistance, followed by controlled eccentric contractions of the knee extensors during the lowering of the lever arm. Although a known amount of weight was lifted, the resistance throughout each contraction was variable because of the presence of a nonuniformly arced cam connected to the cable and rotational axis of the lever arm. Figure 2 presents data from *subject 4* during a simulated repetition from the I-70 trial. The cam permitted the largest muscle force development midway (~50°) in the leg extension phase, after which a progressive decrease in concentric force occurred. Force application increased during the eccentric phase to a value approximating 70% of peak concentric force (at ~35°) and then decreased. This profile was similar for all subjects.

The physical characteristics of the subjects and descriptive characteristics of the I-70 and I-35 trials are presented in Table 1. The resistance load of the I-70 trial was almost double that of the I-35 trial, resulting in a similar relationship for peak concentric torque. The number of repetitions during the I-70 trial was approximately half that of the I-35 trial, whereas the duration of each repetition was similar between trials (2.9 ± 0.3 and 3.1 ± 0.4 s, respectively).

Because the cam was consistent between each trial and for each subject, external work was calculated as the product of the weight of the stack and the distance lifted. External work was calculated for the concentric phase only and was similar between I-70 and I-35 trials (Table 1). Although these values are biased by the mechanical advantage provided by the cam, they are presented to provide an alternative more comprehensible unit of work than force accumulation. Nevertheless, because of the likelihood of muscle glycogenolysis continuing during the eccentric phase of each contraction, the force accumulation data would more accurately reflect the biochemical work performed in each trial.

Muscle and blood biochemistry. The subjects' muscle glycogen and lactate concentrations at rest and during exercise are presented in Table 2. The subjects began the study with moderate muscle glycogen stores. However, despite the dietary and exercise constraints, two subjects' resting muscle glycogen concentrations were relatively high (161.0 and 169.6 mmol/kg wet wt).

Six sets of weight-resistance exercise of different intensities but similar force accumulation and external work resulted in similar amounts of muscle glycogen degradation (46.9 ± 6.6 vs. 46.6 ± 6.0 mmol/kg wet wt). Although not significant ($P = 0.07$), the decline in glycogen during the first three sets in the I-35 trial tended to be larger than during the I-70 trial. The rate of glycogenolysis during the I-70 trial (0.46 ± 0.05 mmol · kg wet $wt^{-1} \cdot s^{-1}$) was almost double that of the I-35 trial (0.21 ± 0.03 mmol · kg wet $wt^{-1} \cdot s^{-1}$ or 1.3 ± 0.03 vs. 0.61 ± 0.01 mmol · $kg^{-1} \cdot rep^{-1}$, respectively). The different glycogenolytic rates were to be expected because the I-35 trial required almost twice as many repetitions as the I-70 trial to obtain equal force accumulation (Tables 1 and 2).

TABLE 1. *Descriptive characteristics of the subjects and trials*

Age, yr	23.2±0.4
Weight, kg	78.9±3.0
Body fat, %	13.4±1.5
Lean body mass, kg	68.0±1.7
1 RM, kg	
Left	52.1±1.6
Right	54.6±1.5
Resistance load, kg	
I-70	38.2±1.1
I-35	9.5±1.1
Peak torque, Nm	
I-70	24.2+1.0
I-35	12.4±0.5
Work, Nm	
I-70	715.6±7.1
I-35	761.2±56.4
Repetitions per set	
I-70	6.0±0.0
I-35	12.7±1.1

Values are means + SE. Peak torque was for concentric phase only. Work was calculated as sum of weight of stack and vertical height lifted during concentric phase of exercise.

TABLE 2. *Muscle glycogen and lactate concentrations and total accumulated force*

	$[Gly]_m$, mmol/kg wet wt	$[La]_m$, mmol/kg wet wt	F, N
Rest			
H	120.3±10.8	1.0±1.3	0.0
L	122.4±9.7	1.1±0.1	0.0
Set 3			
H	95.6±9.3	11.7±1.6	9,169±1,059
L	91.0±6.9	13.0±1.6	9,204±1,028
Set 6			
H	73.4±8.1	13.8±0.7	17,995±2,004
L	75.9±9.2	16.7±4.2	17,623±2,090
2 h			
H	95.6±11.1	1.3±0.1	
L	90.2±9.6	1.2±0.2	

Values are means ± SE. $[Gly]_m$ and $[La]_m$, muscle glycogen and lactate concn; F, total accumulated force; H and L, high- and low-intensity leg extension exercise. No significant differences ($P < 0.05$) existed between trials for either variable.

Muscle lactate concentrations increased to 13.8 ± 0.7 and 16.7 ± 4.2 mmol/kg wet wt after *set 6* of the I-70 and I-35 trials, respectively. Two minutes after *set 6*, muscle lactate concentrations remained approximately twice that of blood (Table 3).

Blood glucose concentrations were similar between trials before exercise, after *set 6*, and 2 h postexercise (Table 3). No differences were detected after correction for plasma volume shifts. During the 2-h postexercise recovery period, muscle glycogen synthesis after the I-70 and I-35 trials occurred at 11.1 ± 3.4 and 7.2 ± 1.3 mmol · kg wet $wt^{-1} \cdot h^{-1}$, respectively. These increases in muscle glycogen during recovery approached significance ($P = 0.08$; Table 2). No significant relationships existed between muscle glycogen synthesis and muscle or blood lactate concentrations after *set 6* of exercise.

Muscle histology. Muscle fiber type data varied considerably between biopsy specimens for any one subject (range 37–65%). Consequently, each subject's mean fiber type proportions were determined from the combined total of the eight sections, from which a mean of 2,993 fibers per subject were counted.

The linear relationship ($r = 0.89$) existed between absolute muscle glycogen concentrations and PAS stain optical absorbance values. On the basis of this relationship, changes in the content of glycogen between fiber types and between trials were evaluated. As expected from the muscle glycogen concentrations (Table 2), no differences in total absorbance existed between I-70 and I-35 trials at any time. In addition, individual fiber type absorbance values were similar between trials (Table 4).

To evaluate further glycogen degradation and storage between fiber types, data from both trials were combined to increase the sample size ($n = 14$). Fiber type comparisons revealed that absorbance values in fast-twitch fibers were significantly larger than those in slow-twitch fibers at rest (Fig. 3). Different rates of glycogenolysis between fiber types were implied by a significant interaction between fiber type absorbance and time ($P = 0.038$). These differences were also demonstrated by the significantly larger decreases in absorbance of fast-twitch compared with slow-twitch fibers after the third set (0.218 ± 0.045 vs. 0.140 ± 0.014, $P = 0.041$) and between *sets 3* and *6* (0.147 ± 0.027 vs. 0.066 ± 0.021, $P = 0.014$). A larger increase in absorbance also occurred in fast-twitch fibers during the 2-h recovery (0.119 ± 0.024 vs. 0.055 ± 0.024, $P = 0.02$).

DISCUSSION

The methodology used in this research was unique in that muscle force development during weight-resistance exercise was quantified and equated between two trials of differing intensity. Prior research of weight-resistance exercise has been hampered by the inability to control for variability in muscle force production or total work. This method was shown to be reliable because comparisons between trials for peak torque, force accumulation, and repetition times provided an internal calibration consistent with the proposed differences in exercise intensity and external work.

Glycogenolysis, muscle lactate, and blood lactate. As indicated in Tables 1 and 2, a greater rate of muscle glycogenolysis occurred in the I-70 trial; yet the longer duration of the I-35 trial resulted in similar glycogen degradation. These findings imply that the total amount of muscle glycogenolysis was dependent on the magnitude of muscle force development and that the rate of glycogenolysis was dependent on exercise intensity. These relationships are not surprising because several investigators have demonstrated an exponential increase in muscle glycogenolysis with increasing exercise intensity (percent maximal O_2 uptake) during cycling or running (36). The interesting fact is that muscle glycogenolysis appears to be dependent on exercise intensity during intense weight-resistance exercise.

The finding of a near linear decrease in muscle glycogen during six sets of leg extension exercise differs from the reported decrease in glycogenolysis during maximal intermittent cycle ergometry (32, 37). These studies revealed that skeletal muscle glycogenolysis was high during the initial bouts but then declined after the third bout of intense exercise. The results were influenced by a decreasing power output during the successive bouts of exercise as well as an increased oxidative contribution and the reversal of phosphorylase *b*-to-*a* activation (5, 6, 8, 37).

Muscle contraction during leg extension weight-resistance exercise comprises both eccentric and concentric

TABLE 3. *Changes of blood parameters*

	ΔPV, %	$[La^-]_a$, mM	$[Glc]_v$, mM
Rest			
H		1.2±0.1	93.4±3.0
L		1.4±0.1	87.5±2.9
Set 6			
H	−7.3±1.6	6.1±0.4†	100.6±2.7†
L	−10.5±1.2*	7.0±0.6†	94.7±3.8†
2 h			
H	1.5±1.1‡	1.0±0.1‡	91.7±2.5‡
L	2.1±1.6‡	1.2±0.0‡	88.0±2.1‡

Values are means ± SE. PV, plasma volume; $[La^-]_a$, arterialized lactate concn; $[Glc]_v$, venous glucose concn. * Significantly different from I-70 trial, $P < 0.05$. † Significantly different from rest, $P < 0.05$. ‡ Significantly different from *set 6*, $P < 0.05$.

TABLE 4. *Fiber type optical absorbance data*

	I-70			I-35		
	Total	FT	ST	Total	FT	ST
Pre	0.72±0.09	0.77±0.09	0.67±0.10	0.69±0.10	0.75±0.10	0.64±0.10
Set 3	0.55±0.08	0.55±0.08	0.55±0.08	0.51±0.05	0.53±0.05	0.49±0.06
Set 6	0.42±0.05	0.37±0.04	0.46±0.07	0.42±0.06	0.42±0.05	0.41±0.07
2 h	0.51±0.06	0.52±0.06	0.50±0.06	0.48±0.07	0.50±0.06	0.44±0.08

Values are means ± SE and represent total absorbance adjusted for fiber type proportions (Total) and absorbance readings from fast- (FT) and slow-twitch (ST) fibers. No significant differences existed between trials or fiber types.

muscle actions, and an elevated intramuscular pressure remained throughout the duration of each repetition. Muscle blood flow has been shown to be occluded during isometric contractions in excess of 20% maximal voluntary contraction (12), whereas muscle blood flow increases linearly with exercise intensity during running and cycling (26). These contrasts in blood flow and muscle contraction patterns may result in energy metabolism differences between weight-resistance exercise and intermittent intense cycling or running.

The average decrease in muscle glycogen during the I-70 and I-35 trials (47 mmol/kg wet wt) was larger than the 23.0 mmol/kg wet wt reported by MacDougall et al. (29). The subjects of the MacDougall study performed three sets of single arm bicep curls to muscle failure, with 3-min recovery between sets. Although the total number of repetitions and mean resistance were not reported by MacDougall, the number of repetitions and total exercise time were likely to be less than the I-70 trial of this study.

The glycogenolysis of this study was similar to the 42.0 mmol/kg wet wt reported by Tesch et al. (41). Subjects from the Tesch investigation performed a total of 20 sets of five different leg exercises to contractile failure (6–12 repetitions), exercising for approximately 10 min with a total duration of 30 min. The subjects from this study exercised for approximately 2 and 4 min for the I-70 and I-35 trials, respectively, with total durations equaling 12 and 14 min. Consequently, the glycogen degradation of this study occurred over a shorter time period with more stringent control over exercise and rest interval durations and muscle force development.

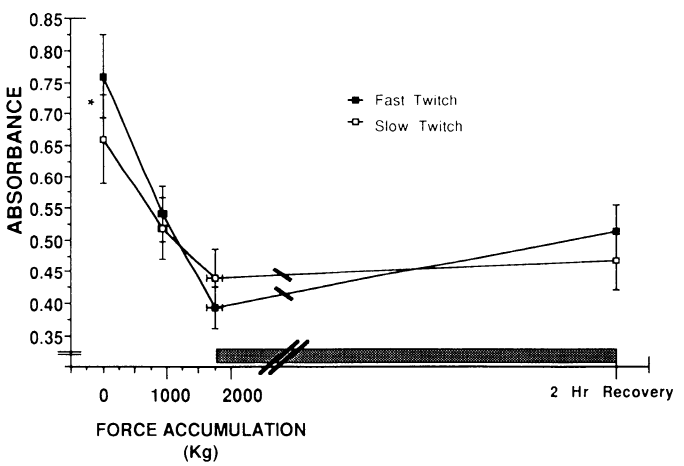

FIG. 3. Fiber type optical absorbance from periodic acid-Schiff-stained muscle sections. Absorbance data are combined from I-70 and I-35 trials (n = 14) and are plotted relative to force accumulation from leg extension machine. Data for 2-h recovery are also included. Before exercise, fast-twitch fibers had a significantly higher absorbance (*P < 0.05). When an ordinal time scale incorporating the 4 data points of each fiber type was used, a significant interaction existed between absorbance and time (P = 0.04).

Assuming no change in muscle glycogen content during the 2-min rest periods between sets, the glycogenolytic rate in the I-70 and I-35 trials of this study were 0.46 and 0.21 mmol · kg wet $wt^{-1} \cdot s^{-1}$, respectively. Spriet et al. (39) measured the rate of glycogenolysis in the human vastus lateralis during electrical stimulation. During the first 16 contractions, muscle glycogenolysis occurred at a rate of 0.41 mmol · kg wet $wt^{-1} \cdot s^{-1}$ and decreased to 0.17 mmol · kg wet $wt^{-1} \cdot s^{-1}$ during the remaining 32 contractions. In other studies, the calculated glycogenolytic rate after 30 s of sprint running approximated 0.56 mmol · kg wet $wt^{-1} \cdot s^{-1}$ (7), and 30 s of maximal intermittent isokinetic cycling has yielded glycogenolytic rates between 0.4 and 0.65 mmol · kg wet $wt^{-1} \cdot s^{-1}$ (32, 37). These latter values are slightly higher than the rate of our I-70 trial and indicate a similarity in the rate of skeletal muscle glycogenolysis between intense cycle ergometry and weight-resistance exercise.

The majority of muscle lactate accumulation occurred during the first three sets in each trial. In fact, the muscle lactate accumulation during the six sets of the I-70 trial was attained after three sets in the I-35 trial. Consequently, the pattern of lactate accumulation differed between trials, despite similar glycogen degradation (Table 2). Although the data prevent a thorough evaluation of glycolysis, it appears that a greater glycolytic stimulus may have occurred during the final three sets of the I-35 trial. This is understandable, given the lower intensity and longer exercise duration. To account for the similar glycogenolysis yet different lactate accumulation between trials, a larger accumulation of glycolytic intermediates would have had to occur during the latter three sets of the I-70 trial. This interpretation requires further research yet is supported by the increase in glycolytic intermediates known to occur with fatigue during isometric muscle contractions, high-intensity running or cycling, and weight-resistance exercise (7, 18, 20–22, 32, 39).

Lesmes et al. (27) reported greater glycogen degradation in fast-twitch fibers after "isokinetic" resistance exercise. Our results appear to support this finding and imply that resistance exercise causes greater glycogenolysis in fast-twitch than in slow-twitch fibers. However, these results are not evidence of preferential fast-twitch fiber recruitment. Fast-twitch fibers are known to have a larger glycolytic capacity than slow-twitch fibers. In ad-

dition, the significantly larger resting glycogen stores within the fast-twitch fibers of this study (Fig. 3) would have favored greater fast-twitch fiber glycogen degradation.

Glycogenesis. The rates of glycogenesis during the I-70 and I-35 trials occurred at 11.1 ± 3.4 and 7.2 ± 1.3 mmol · kg wet $wt^{-1} \cdot h^{-1}$, respectively, and were similar to the 5–9 mmol · kg wet $wt^{-1} \cdot h^{-1}$ values reported for glycogen synthesis after submaximal exercise with carbohydrate feedings (23). The rate and total amount of postexercise glycogen synthesis during the I-70 trial are also comparable with the findings of Hultman (21) (16.2 mmol · kg wet $wt^{-1} \cdot h^{-1}$). These comparisons indicate that a large substrate supply for glycogenesis existed without carbohydrate ingestion and/or significantly elevated blood glucose concentrations (Table 3).

Previous explanations of high rates of glycogen synthesis after intense exercise have been based on the potential for lactate to be an endogenous muscle glycogenic precursor (1, 19, 21, 29, 30, 40). Direct evidence for this process has been provided by in vitro studies with the use of animal skeletal muscle. Enzyme activity of the gluconeogenic enzymes phosphoenolpyruvate carboxykinase and fructose bisphosphatase and minor activity of pyruvate carboxykinase have been shown in white (fast-twitch glycolytic) vertebrate muscle (1, 34). In addition, perfusion or incubation of fast-twitch glycolytic muscle fibers with solutions of high lactate concentrations (4–12 mmol/l) has resulted in high rates of muscle glycogen synthesis and evidence of the incorporation of ^{14}C from [^{14}C]lactate into glycogen (33).

Despite this evidence, doubt remains that lactate conversion to glycogen exists under in vivo physiological conditions (3, 4). The data of Hermansen and Vaage (19) and Hultman (21) indicated limited lactate removal from exercised muscle; however, the results from the Hermansen study were estimations from blood flow measurements of the lower leg rather than the thigh, and different subject groups and exercise modes were used for different aspects of data collection. It is unreasonable to assume that increases in blood lactate amounting to 20 mmol/l do not originate from the exercised muscle and/or that lactate efflux ceases during the recovery process. Data from Harris et al. (18) have shown that muscle lactate declines appreciably during the first 4 min of recovery from high-intensity exercise without detectable increases in muscle glycogen and that this lactate removal is blood flow dependent. Furthermore, short-term exercise to exhaustion results in a large increase in glycolytic intermediates above pyruvate. These increases often amount to concentrations equal to or in excess of glucosyl unit equivalents of lactate (18, 41). Although our data do not allow a more definitive appraisal of this topic, the contribution of muscle lactate or glycolytic intermediates to glycogen synthesis after intense exercise remains unclear.

On the basis of the positive relationship between exercise-induced glycogen degradation and subsequent glycogen synthesis (44), it is understandable that fast-twitch fibers also had a larger amount of postexercise glycogen storage. Nevertheless, it must be emphasized that considerable glycogen synthesis also occurred in slow-twitch muscle fibers (Table 4). This latter fact further decreases the likelihood of the conversion of intramuscular lactate to glycogen in this study, which previous research has shown should occur preferentially in fast-twitch glycolytic muscle (1, 33, 34).

Conclusions. The central finding of this investigation was that skeletal muscle glycogenolysis was similar in magnitude between 75 and 35% 1 RM intensity weight-resistance exercise when an indirect indicator of muscle force development was equated between trials. In addition, the results revealed that skeletal muscle glycogenolysis occurred at comparable rates with those reported during maximal isokinetic cycle ergometry (32, 37). Nevertheless, the contribution of glycogen-derived glucose residues to muscle energetics during weight-resistance exercise remains uncertain.

The postexercise synthesis of glycogen occurred at rates higher than reported after exhaustive submaximal exercise when carbohydrate feedings were ingested during the first 6 h of recovery. The predominance of glycogenolysis and glycogenesis in fast-twitch fibers was interpreted as a reflection of the greater glycogenolytic and glycolytic capacities of this fiber type and the bias of the significantly larger resting glycogen content of the fast-twitch fibers.

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REFERENCES

1. BENDALL, J. R., AND A. A. TAYLOR. The Meyerhof quotient and the synthesis of glycogen from lactate in frog and rabbit muscle. *Biochem. J.* 118: 887–893, 1970.
2. BERGSTROM, J. Muscle electrolytes in man. *Scan. J. Clin. Lab. Invest. Suppl.* 68: 1–110, 1962.
3. BROOKS, G. A., K. E. BRAUNER, AND R. G. CASSENS. Glycogen synthesis and metabolism of lactic acid after exercise. *Am. J. Physiol.* 224: 1162–1166, 1973.
4. BROOKS, G. A., AND G. A. GAESSER. End points of lactate and glucose metabolism after exhausting exercise. *J. Appl. Physiol.* 49: 1057–1069, 1980.
5. CHASIOTIS, D. The regulation of glycogen phosphorylase and glycogen breakdown in human skeletal muscle. *Acta Physiol. Scand. Suppl.* 518: 1–68, 1983.
6. CHASIOTIS, D., E. HULTMAN, AND K. SAHLIN. Acidotic depression of cyclic AMP accumulation and phosphorylase *b* to a transformation in skeletal muscle of man. *J. Physiol. Lond.* 335: 197–204, 1982.
7. CHEETHAM, M. E., L. H. BOOBIS, S. BROOKS, AND C. WILLIAMS. Human muscle metabolism during sprint running. *J. Appl. Physiol.* 61: 54–60, 1986.
8. CONLEE, R. K., J. A. MCLANE, M. J. RENNIE, W. W. WINDER, AND J. O. HOLLOSZY. Reversal of phosphorylase activation in muscle despite continued contractile activity. *Am. J. Physiol.* 237 (*Regulatory Integrative Comp. Physiol.* 6): R291–R296, 1979.
9. COSTILL, D. L., D. R. PEARSON, AND W. J. FINK. Impaired muscle glycogen storage after muscle biopsy. *J. Appl. Physiol.* 64: 2245–2248, 1988.
10. DILL, B. D., AND D. L. COSTILL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J. Appl. Physiol.* 37: 247–249, 1974.
11. DUDLEY, G. A. Metabolic consequences of resistive-type exercise. *Med. Sci. Sports Exercise* 20: S158–S161, 1988.

12. EDWARDS, H. T., D. K. HILL, AND M. MCDONNELL. Myothermal and intramuscular pressure measurements during isometric contractions of the human quadraceps muscle. *J. Physiol. Lond.* 224: 58–59P, 1973.
13. ESSEN, B., AND L. KAIJSER. Regulation of glycolysis in intermittent exercise in man. *J. Physiol. Lond.* 281: 499–511, 1978.
14. EVANS, W. J., S. J. PHINNEY, AND V. R. YOUNG. Suction applied to a muscle biopsy maximizes sample size. *Med. Sci. Sports Exercise* 14: 101–102, 1982.
15. GOLDFARB, A. H., J. F. BRUNO, AND P. J. BUCKENMEYER. Intensity and duration of exercise effects on skeletal muscle cAMP, phosphorylase, and glycogen. *J. Appl. Physiol.* 66: 190–194, 1989.
16. GOLLNICK, P. D., R. B. ARMSTRONG, W. L. SEMBROWICH, R. E. SHEPHERD, AND B. SALTIN. Glycogen depletion pattern in human skeletal muscle fibers after heavy exercise. *J. Appl. Physiol.* 34: 615–618, 1973.
17. GOLLNICK, P. D., K. PIEHL, AND B. SALTIN. Selective glycogen depletion pattern in human muscle fibers after exercise of varying intensity and at varying pedal rates. *J. Physiol. Lond.* 241: 45–57, 1974.
18. HARRIS, R. C., E. HULTMAN, AND K. SAHLIN. Glycolytic intermediates in human muscle after isometric contraction. *Pfluegers Arch.* 389: 277–282, 1989.
19. HERMANSEN, L., AND O. VAAGE. Lactate disappearance and glycogen synthesis in human muscle after maximal exercise. *Am. J. Physiol.* 233 (*Endocrinol. Metab. Gastrointest. Physiol.* 2): E422–E429, 1977.
20. HIRVONEN, J., S. REHUNEN, H. RUSKO, AND M. HARKONEN. Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. *Eur. J. Appl. Physiol. Occup. Physiol.* 56: 253–259, 1987.
21. HULTMAN, E. H. Carbohydrate metabolism during hard exercise and in the recovery period after exercise. *Acta Physiol. Scand.* 128, *Suppl.* 556: 75–82, 1986.
22. HULTMAN, E., AND H. SJOHOLM. Energy metabolism and contraction force of human skeletal muscle in situ during electrical stimulation. *J. Physiol. Lond.* 345: 525–532, 1983.
23. IVY, J. L., M. C. LEE, J. T. BRONZINICK, JR., AND M. C. REED. Muscle glycogen storage following different amounts of carbohydrate ingestion. *J. Appl. Physiol.* 65: 2018–2023, 1988.
24. JACOBS, I., P. A. TESCH, O. BAR-OR, J. KARLSSON, AND R. DOTAN. Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. *J. Appl. Physiol.* 55: 365–367, 1983.
25. KEPPEL, G. *Design and Analysis: A Researchers Handbook.* Philadelphia, PA: Prentice-Hall, 1983, p. 155–157.
26. LAUGHLIN, M. H., AND R. B. ARMSTRONG. Muscle blood flow during locomotory exercise. *Exercise Sports Sci. Rev.* 13: 105–108, 1983.
27. LESMES, G. R., D. W. BENHAM, D. L. COSTILL, AND W. J. FINK. Glycogen utilization in fast and slow twitch muscle fibers during maximal isokinetic exercise. *Ann. Sports Med.* 1: 105–108, 1983.
28. LOWRY, O. H., AND J. V. PASSONNEAU. *A Flexible System of Enzyme Analysis.* New York: Academic, 1972, p. 179, 191, 195, 213.
29. MACDOUGALL, J. D., S. RAY, N. MCCARTENY, D. SALE, P. LEE, AND S. GADNER. Substrate utilization during weight lifting (Abstract). *Med. Sci. Sports Exercise* 20: S66, 1988.
30. MACDOUGALL, J. D., G. R. WARD, D. G. SALE, AND J. R. SUTTON. Muscle glycogen repletion after high intensity intermittent exercise. *J. Appl. Physiol.* 42: 129–132, 1977.
31. MACDOUGALL, J. D., G. R. WARD, D. G. SALE, AND J. R. SUTTON. Biochemical adaptation of human skeletal muscle to heavy resistance exercise training and immobilization. *J. Appl. Physiol.* 43: 700–703, 1977.
32. MCCARTNEY, N., L. L. SPRIET, G. J. F. HEIGENHAUSER, J. M. KOWALCHUCK, J. R. SUTTON, AND N. L. JONES. Muscle power and metabolism in maximal intermittent exercise. *J. Appl. Physiol.* 60: 1164–1169, 1986.
33. MCLANE, J. A., AND J. O. HOLLOSZY. Glycogen synthesis from lactate in the three types of skeletal muscles. *J. Biol. Chem.* 254: 6548–6553, 1979.
34. OPIE, L. H., AND E. A. NEWSHOLME. The activities of fructose 1,6-diphosphatase, phosphofructokinase and phosphoenolpyruvate carboxykinase in white muscle and red muscle. *Biochem. J.* 103: 391–399, 1967.
35. PETER, J. B., R. J. BARNARD, V. R. EDGERTON, C. A. GILLESPIE, AND K. E. STEMPEL. Metabolic profiles of three types of skeletal muscle in guinea pigs and rabbits. *Biochemistry* 11: 2627–2633, 1972.
36. SALTIN, B., AND J. KARLSSON. Muscle glycogen utilization during work of different intensities. In: *Muscle Metabolism During Exercise,* edited by B. Pernow and B. Saltin. New York: Plenum, 1971, vol. 11, p. 289–299.
37. SPRIET, L. L., M. L. LINDINGER, R. S. MCKELVIE, G. J. F. HEIGENHAUSER, AND N. L. JONES. Muscle glycogenolysis and H^+ concentration during maximal intermittent cycling. *J. Appl. Physiol.* 66: 8–13, 1989.
38. SPRIET, L. L., J. M. REN, AND E. HULTMAN. Epinephrine infusion enhances muscle glycogenolysis during prolonged electrical stimulation. *J. Appl. Physiol.* 64: 1439–1444, 1988.
39. SPRIET, L. L., K. SODERLUND, M. BERGSTROM, AND E. HULTMAN. Skeletal muscle glycogenolysis, glycolysis, and pH during electrical stimulation in men. *J. Appl. Physiol.* 62: 616–621, 1987.
40. TERJUNG, R. L., K. M. BALDWIN, W. W. WINDER, AND J. O. HOLLOSZY. Glycogen repletion in different types of skeletal muscles and liver after exhaustive exercise. *Am. J. Physiol.* 226: 1387–1391, 1974.
41. TESCH, P. A., B. COLLIANDER, AND P. KAISER. Muscle metabolism during intense, heavy resistance exercise. *Eur. J. Appl. Physiol. Occup. Physiol.* 55: 362–366, 1986.
42. VOLLESTAD, N. K., P. C. S. BLOM, AND O. GRONNEROD. Resynthesis of glycogen in different muscle fiber types after prolonged exhaustive exercise in man. *Acta Physiol. Scand.* 137: 15–21, 1989.
43. VOLLESTAD, N. K., O. VAAGE, AND L. HERMANSEN. Muscle glycogen depletion pattern in type I and subgroups of type II fibers during prolonged severe exercise. *Acta Physiol. Scand.* 122: 433–440, 1984.
44. ZACHWEIJA, J., D. L. COSTILL, D. D. PASCOE, R. A. ROBERGS, AND W. J. FINK. Influence of muscle glycogen depletion on the rate of resynthesis. *Med. Sci. Sports Exercise* 23: 44–48, 1991.