Muscle glycogen accumulation after endurance exercise in trained and untrained individuals


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Hickner, R. C., J. S. Fisher, P. A. Hansen, S. B. Racette, C. M. Mier, M. J. Turner, and J. O. Holloszy. Muscle glycogen accumulation after endurance exercise in trained and untrained individuals. J. Appl. Physiol. 83(3): 897–903, 1997.—Muscle glycogen accumulation was determined in six trained cyclists (Trn) and six untrained subjects (UT) at 6 and either 48 or 72 h after 2 h of cycling exercise at ~75% peak O2 uptake (V\text{O}_2\text{peak}), which terminated with five 1-min sprints. Subjects ate 10 g carbohydrate·kg\text{body weight}^{-1}·day^{-1} for 48–72 h postexercise. Muscle glycogen accumulation averaged 71 ± 9 (SE) mmol/kg (Trn) and 31 ± 9 mmol/kg (UT) during the first 6 h postexercise (P < 0.01) and 79 ± 22 mmol/kg (Trn) and 60 ± 9 mmol/kg (UT) between 6 and 48 or 72 h postexercise (not significant). Muscle glycogen accumulation was 164 ± 21 mmol/kg (Trn) and 99 ± 16 mmol/kg (UT) 48–72 h postexercise (P < 0.05). Muscle GLUT-4 content immediately after exercise was threefold higher in Trn than in UT (P < 0.05) and correlated with glycogen accumulation rates (r = 0.66, P < 0.05). Glycogen synthase in the active I form was 2.5 ± 0.5, 3.3 ± 0.5, and 1.0 ± 0.3 μmol·g\text{muscle tissue}^{-1}·min^{-1} in Trn at 0, 6, and 48 or 72 h postexercise, respectively; corresponding values were 1.2 ± 0.3, 2.7 ± 0.5, and 1.6 ± 0.3 μmol·g\text{muscle tissue}^{-1}·min^{-1} in UT (P < 0.05 at 0 h). Plasma insulin and plasma C-peptide area under the curve were lower in Trn than in UT over the first 6 h postexercise (P < 0.05). Plasma creatine kinase concentrations were 125 ± 25 IU/l (Trn) and 91 ± 9 IU/l (UT) preexercise and 112 ± 14 IU/l (Trn) and 144 ± 22 IU/l (UT; P < 0.05 vs. preexercise) at 48–72 h postexercise (normal: 30–200 IU/l). We conclude that endurance exercise training results in an increased ability to accumulate muscle glycogen after exercise.

fiber type; GLUT-4; glycogen synthase; glycogen supercompensation

MUSCLE GLYCOGEN accumulation and supercompensation have been extensively studied since the pioneering investigation of Bergström and Hultman (3). However, there have been few studies of this process in untrained individuals, and, remarkably, only one investigation in which a direct comparison of trained and untrained individuals was made (5). This is surprising not only in light of the wealth of publications concerning glycogen accumulation in skeletal muscle but also because of possible insights regarding the glycogen accumulation process that such a comparison might provide.

Muscle glycogen accumulation has been shown to correlate with the relative proportion of glycogen synthase in the active (I) form (4, 17). However, the amount of the glucose transporter protein (GLUT-4) appears to be rate limiting for this process because GLUT-4 content has been shown to determine the rate of glucose transport into muscle (33). Untrained individuals have lower levels of GLUT-4 than trained individuals, and the level of GLUT-4 increases with endurance exercise training (11, 15, 31). Muscle GLUT-4 content has also been reported to be correlated with glycogen accumulation rates over the 6 h immediately after exercise when endurance-trained and physically active subjects are fed a high carbohydrate diet (25). However, there have been no investigations into whether a correlation exists between muscle GLUT-4 content and muscle glycogen accumulation rates in trained and untrained individuals.

Recently Nakatani et al. (28) have reported that exercise training that induces an increase in muscle GLUT-4 content results in a large increase in the rate of muscle glycogen accumulation after a glycogen-depleting bout of exercise in rats. In this context, the purpose of the present investigation was to determine whether a similar phenomenon is seen in humans. To this end, we compared endurance-trained cyclists with untrained individuals with respect to muscle glycogen accumulation rates after glycogen-depleting endurance exercise.

METHODS

Subjects. Six healthy endurance-trained cyclists and six healthy untrained (sedentary) men participated in this study after giving informed consent, according to the Human Studies Committee at Washington University School of Medicine. Physical characteristics are presented in Table 1.

Exercise protocol. Subjects reported to the laboratory after an overnight fast. An intravenous catheter was inserted in an antecubital vein and kept patent with normal saline. Subjects rested for 5 min before the initial blood sample was drawn. Subjects then cycled on a cycle ergometer (Lode, Groningen, The Netherlands) at ~75% peak O2 uptake (V\text{O}_2\text{peak}) for 2 h, including a 4-min rest every 30 min. Subjects then performed five 1-min sprints at a workload that elicited 100% V\text{O}_2\text{peak} (as determined during V\text{O}_2\text{peak} testing). A 3-min rest was allowed between sprints.

The subjects did not exercise during the 2- to 3-day period of recovery after the glycogen-depletion exercise bouts. Diet. Subjects were given a controlled diet for 48 h before, and for 48 or 72 h after, exercise. Subjects ate at hours convenient to their schedule, except during the 6 h after termination of exercise. During this time, they ate at 0, 2, and 4 h postexercise. The preexercise diet consisted of 37 kcal·kg\text{body weight}^{-1}·day^{-1}, plus additional calories equal to estimated caloric expenditure during exercise-training sessions for the trained individuals. The preexercise diet consisted of 50% carbohydrate (CHO), 30% fat, and 20% protein. The postexercise diet, consisting of 80% CHO, 7% fat, and 13% protein, was designed to result in rapid accumulation of muscle glycogen stores and provided 10 g CHO·kg\text{body weight}^{-1}·day^{-1}. The diet during the first 6 h postexercise consisted of two-thirds of the carbohydrate for that day, supplying 1.4 g CHO·kg\text{body weight}^{-1}·h^{-1}. The meals at 0,
2, and 4 h postexercise consisted of a beverage containing 75–100 g glucose/mal, cereal and milk, bagels, raisins, jelly, fruit, and tuna.

Biopsy. Before initiation of exercise, an incision was made in the vastus lateralis of the quadriceps femoris muscle after administration of local anesthesia (2% lidocaine). The biopsy site was draped with sterile dressing and wrapped with an Ace bandage for the duration of the exercise session. A biopsy was taken from the vastus lateralis of the quadriceps femoris muscle immediately after the final sprint. The biopsy sample was immediately frozen in liquid nitrogen and stored at −80°C for subsequent analysis. A biopsy was also taken from the vastus lateralis of the quadriceps femoris muscle of the contralateral leg 6 h after the initiation of eating (which began within 20 min postexercise), as well as from the vastus lateralis of the same leg 48 h (6 subjects, 3 trained and 3 untrained) or 72 h (6 subjects, 3 trained and 3 untrained) after initiation of eating. The study was terminated at 48 h, instead of 72 h, to accommodate the subjects’ schedules because glycogen supercompensation has been shown to occur within 48 h (3). The 48/72-h biopsies were taken from a site at least 3 cm distal to the initial biopsy to avoid possible influences of the previous biopsy procedure on muscle glycogen (9). Muscle biopsy samples were analyzed for glycogen (29), GLUT-4 protein (12), glycogen synthase (30), and fiber type (6). GLUT-4 concentration was determined only on the biopsy sample obtained immediately after exercise.

Blood samples. Blood samples were drawn immediately before exercise, immediately before termination of the last 0.5 h ride, immediately after the last sprint, and every 0.5 h after initiation of eating for the subsequent 6 h. One blood sample was also drawn at 48/72 h postexercise. Blood samples were collected in tubes containing heparin for determination of plasma glucose by using the glucose oxidase method (Stratus II, Dade International, Miami, FL), trasylol for measurement of plasma glucose and insulin by using the glucose oxidase method, and either NEFA C kit (Waco Chemicals, Dallas, TX) or a modified hydrostatic weighing procedure by using partial expiration as employed by Kohrt et al. (22). Residual volume was measured outside the weighing tank by using the oxygen-dilution method described by Wilmore (36). Percent body fat was estimated by using the formula of Brozek et al. (7).

Statistics. Hormone and metabolite data, as well as glycogen concentration data, were analyzed by using two-way analysis of variance with repeated measures over time. When significance was attained, post hoc analysis was performed by using the Student-Newman-Keuls test. Student’s t-test was used to detect differences between groups with respect to glucose and insulin area under the curve, lactate concentration, glycogen accumulation rates, and fiber type. Multiple-regression analysis was performed, as well as analysis of residuals, by using BMDP software (Los Angeles, CA). All data are presented as means ± SE.

RESULTS

Muscle glycogen concentration. Muscle glycogen concentrations in trained and untrained subjects immediately after the exercise, 6 h after cessation of exercise, and either 48 or 72 h after cessation of exercise are presented in Fig. 1. Rates of muscle glycogen accumulation over the 6 h immediately after exercise were twofold greater in trained than in untrained individuals (70.8 ± 8.7 vs. 30.6 ± 9.3 mmol·kg wet wt⁻¹·6 h⁻¹, respectively; n = 6; P < 0.01). Muscle glycogen concentration at 48/72 h postexercise was greater in trained than in untrained individuals (164 ± 21 vs. 99 ± 16 mmol/kg wet wt; P < 0.05, n = 6), although the 30% higher rate of glycogen accumulation between 6 and 48/72 h in trained than in untrained individuals was

Body composition. A modified hydrostatic weighing procedure by using partial expiration was employed as described by Kohrt et al. (22). Residual volume was measured outside the weighing tank by using the oxygen-dilution method described by Wilmore (36). Percent body fat was estimated by using the formula of Brozek et al. (7).

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not statistically different (78.7 ± 21.8 vs. 60.2 ± 9.0 mmol/kg wet wt). The muscle glycogen concentration in
the trained subjects was 146 ± 38.5 mmol/kg wet weight (n = 3) at 48 h and 182.1 ± 20.8 mmol/kg wet weight (n = 3) at 72 h [P = 0.46; not significant (NS)]. The glycogen values for the untrained subjects were 63.4 ± 7.2 mmol/kg wet weight (n = 3) at 48 h and 134.4 ± 8.6 mmol/kg wet weight (n = 3) at 72 h (P < 0.01). Data at the specific time points of 48 and 72 h should be interpreted with caution because of the small number of subjects in each group (n = 3); nevertheless, it is clear that higher levels of glycogen were attained in the trained group.

Muscle GLUT-4 content. Muscle GLUT-4 content in trained and untrained subjects is presented in Fig. 2. Muscle GLUT-4 content was threefold higher in trained than in untrained subjects (P = 0.05) and correlated with glycogen accumulation rates over the initial 6 h after exercise (Fig. 3; r = 0.66; P = 0.05; n = 12), the glycogen level attained at 48/72 h (r = 0.57, P = 0.05; n = 12), and the percentage of type I fibers (Fig. 4; r = 0.64, P < 0.05; n = 10). Data are presented for only 10 subjects in Fig. 4 because it was not possible to perform muscle fiber typing on two of the subjects due to insufficient muscle sample size.

Muscle glycogen synthase activity. Total muscle glycogen synthase activity was not different at 0, 6, and 48/72 h postexercise; therefore, these values were pooled. Total muscle glycogen synthase activity was higher postexercise in the trained than in the untrained subjects (6.0 ± 0.4 vs. 4.4 ± 0.3 mmol·kg⁻¹·min⁻¹; P < 0.01). The percentage of glycogen synthase in the I form at 0, 6, and 48 h postexercise was calculated by using the values for total glycogen synthase at the corresponding individual time points and were 40.7 ± 6.1, 55.8 ± 8.8, and 17.2 ± 3.1% in trained subjects and 26.0 ± 5.1, 59.8 ± 9.6, and 35.8 ± 6.9% in untrained subjects, respectively. Muscle glycogen synthase I activity was higher in trained than in untrained subjects immediately after exercise (2.5 ± 0.5 vs. 1.2 ± 0.3 mmol·kg⁻¹·min⁻¹; P < 0.05; Fig. 5). Glycogen synthase I activity was not correlated with glycogen accumulation rates over the first 6 h after exercise (r = 0.661; P = 0.019; n = 12).

Fig. 2. Muscle GLUT-4 content immediately postexercise in trained and untrained subjects. Values are means ± SE; n = 6 subjects. OD, arbitrary optical density units. *Different from untrained subjects, P < 0.05.

Fig. 3. Relationship between GLUT-4 content immediately postexercise and muscle glycogen accumulation over initial 6 h postexercise. Twelve subjects cycled for 2 h at −75% VO₂peak, followed by 5 1-min sprints. Subjects then consumed a diet supplying 1.4 g CHO·kg body wt⁻¹·h⁻¹ over initial 6 h postexercise. n, No. of subjects.

Fig. 4. Relationship between GLUT-4 content and percentage of type I muscle fibers. n, No. of subjects.

Fig. 5. Muscle glycogen synthase I in trained and untrained subjects immediately after exercise, 6 h postexercise, and either 48 or 72 h postexercise. Values are means ± SE; n = 6 subjects. Subjects cycled for 2 h at −75% VO₂peak, followed by 5 1-min sprints. Subjects consumed a diet supplying 1.4 g CHO·kg body wt⁻¹·h⁻¹ over initial 6 h postexercise. Diet over 48–72 h postexercise, consisting of 80% CHO, 7% fat, and 13% protein, provided 10 g CHO·kg body wt⁻¹·day⁻¹. *Different from untrained subjects, P < 0.05.
Fiber type. The percentage of type I fibers was greater in trained (70.3 ± 3.7%) than in untrained (46.4 ± 3.0%; P < 0.01, n = 5) subjects, and the percentage of type IIB fibers was less in trained (1.6 ± 0.7%) than in untrained (22.2 ± 3.4%; P < 0.01, n = 5) subjects. There was no difference in the IIA fiber type percent between groups. The percentage of type I fibers was correlated with the rate of glycogen accumulation over the 6 h immediately after cessation of exercise (Fig 6A: r = 0.635, P = 0.048; n = 10). This correlation improved when type I and IIA fibers were combined (Fig 6B: r = 0.721, P = 0.018; n = 10).

Fig. 6. A: relationship between percentage of type I muscle fibers and glycogen accumulation rates over initial 6 h postexercise. Subjects cycled for 2 h at ~75% \( V\text{O}_2\text{peak} \), followed by 5 1-min sprints. Subjects then consumed a diet supplying 1.4 g CHO·kg body wt\(^{-1}\)·h\(^{-1}\) over initial 6 h postexercise. B: relationship between percentage of type I and type IIA muscle fibers and glycogen accumulation rates over initial 6 h postexercise. Subjects cycled for 2 h at ~75% \( V\text{O}_2\text{peak} \), followed by 5 1-min sprints. Subjects then consumed a diet supplying 1.4 g CHO·kg body wt\(^{-1}\)·h\(^{-1}\) over initial 6 h postexercise. n, No. of subjects.

Hormones and metabolites. Data for plasma insulin, C peptide, and glucose are presented in Figs. 7, A and B, respectively. Areas under the curves from 0–360 min postexercise were larger in the untrained than in the trained subjects for plasma insulin (31,819 ± 9,618 vs. 9,072 ± 1,468 µU·ml\(^{-1}\)·h\(^{-1}\); P < 0.05) and C peptide (4,355 ± 871 vs. 2,217 ± 144 ng·ml\(^{-1}\)·h\(^{-1}\); P < 0.05). There was a correlation of \( r = -0.55 \) (P = 0.06) between the area under the glucose curves and glycogen accumulation rate over the initial 6 h after exercise. Plasma glucose values were significantly lower in trained than in untrained subjects during the first 6 h of recovery when compared by using a two-way repeated-measures analysis of variance (Fig. 8). Plasma lactate concentrations immediately after exercise were 5.7 ± 1.3 and 6.9 ± 0.6 mM in trained and untrained subjects, respectively (P = NS). There were no significant differences in either norepinephrine or epinephrine concentrations between trained and untrained subjects (data not shown). No differences were observed in serum free fatty acid, plasma \( \beta \)-hydroxybutyrate, or plasma lactate concentrations between trained and untrained subjects immediately before (~141 min) and at end of (~21 min) endurance cycling exercise, as well as immediately after final sprint (0 min) and over initial 6 h postexercise (0–360 min). Values are means ± SE; n = 6 subjects. Subjects cycled for 2 h at ~75% \( V\text{O}_2\text{peak} \), followed by 5 1-min sprints. Subjects then consumed a diet supplying 1.4 g CHO·kg body wt\(^{-1}\)·h\(^{-1}\) over initial 6 h postexercise.

Fig. 7. A: plasma insulin concentration in trained and untrained subjects immediately before (~141 min) and at end of (~21 min) endurance cycling exercise, as well as immediately after final sprint (0 min) and over initial 6 h postexercise (0–360 min). Values are means ± SE; n = 6 subjects. Subjects cycled for 2 h at ~75% \( V\text{O}_2\text{peak} \), followed by 5 1-min sprints. Subjects then consumed a diet supplying 1.4 g CHO·kg body wt\(^{-1}\)·h\(^{-1}\) over initial 6 h postexercise.
V̇O\textsubscript{2peak} and glycogen accumulation rate; however, it did study, there also appears to be a relationship between ration over the first 6 h after exercise. In the present a correlation between V̇O\textsubscript{2peak} and glycogen accumula-

The only previous accumulation in rats after exercise. The only previous subjects immediately before (–141 min) and at end of (–21 min) endurance cycling exercise, immediately after final sprint (0 min) and over initial 6 h postexercise (0–360 min). Values are means ± SE; n = 6 subjects. Subjects cycled for 2 h at –75% V̇O\textsubscript{2peak}, followed by 5 1-min sprints. Subjects then consumed a diet supplying 1.4 g CHO·kg body wt\textsuperscript{-1}·h\textsuperscript{-1} over initial 6 h postexercise.

CK concentration. Plasma CK concentrations were 125 ± 25 and 91 ± 9 IU/l before exercise and 112 ± 14 and 144 ± 22 IU/l at 48/72 h postexercise in trained and untrained subjects, respectively. Plasma CK values were increased in the untrained subjects 48–72 h postexercise compared with preexercise (P = 0.04); however, these values were not significantly different from those of the trained subjects and were in the normal range of 30–200 IU/l. There was no correlation between plasma CK concentration and the rate of glycogen accumulation at 6 or 48/72 hr after exercise.

Oxygen uptake. V̇O\textsubscript{2peak} data are presented in Table 1. The correlation between V̇O\textsubscript{2peak} and glycogen accumulation over the initial 6 h after exercise was r = 0.46 (P = 0.01). A similar correlation (r = 0.46; P = 0.01) was found between V̇O\textsubscript{2peak} and glycogen accumulation over the 48/72 h after exercise. McCoy et al. (25) have found a correlation between V̇O\textsubscript{2peak} and glycogen accumulation over the first 6 h after exercise. In the present study, there also appears to be a relationship between V̇O\textsubscript{2peak} and glycogen accumulation rate; however, it did not attain statistical significance.

There was no difference in the oxygen uptake relative to V̇O\textsubscript{2peak} between trained (72.2 ± 1.3% V̇O\textsubscript{2peak}) and untrained (73.3 ± 1.9% V̇O\textsubscript{2peak}) subjects during the endurance ride. The respiratory exchange ratio during the ride was 0.899 ± 0.014 for trained and 0.942 ± 0.247 for untrained subjects (NS). Oxygen consumption was 0.397 ± 0.26 l/min in trained and 0.295 ± 0.29 l/min in untrained subjects (NS) during the 6 h after exercise. The respiratory exchange ratio was similar in trained (0.894 ± 0.012) and untrained (0.896 ± 0.032) subjects during the 6 h after cessation of exercise.

**DISCUSSION**

Nakatani et al. (28) recently reported that endurance exercise training enhances the rate of muscle glycogen accumulation in rats after exercise. The only previous study of glycogen accumulation in trained and untrained individuals was that of Blom et al. (5), who compared well-trained runners and untrained subjects after a 90- to 120-min run to exhaustion. No difference was observed between trained and untrained subjects with respect to the rates of glycogen accumulation over the first 22 h after exercise. Muscle glycogen levels were lower immediately after exercise in untrained than in trained individuals in that study, which may have resulted in a higher rate of accumulation in the untrained group than would have occurred at a higher initial glycogen level immediately after exercise. This is probably not the cause of the discrepant results between the study of Blom et al. and the present study, however, because the glycogen levels immediately after exercise in the present study were also lower in the untrained than in the trained group.

Studies on rat muscles have shown that glucose transport rates are correlated with muscle GLUT-4 content (14, 18) and that an increase in muscle GLUT-4 is associated with an increase in the rate of insulin-stimulated glycogen synthesis (33). In the present study, the rate of glycogen accumulation over the 6 h immediately after exercise was correlated with GLUT-4 concentration, as has been demonstrated previously by McCoy et al. (25). The data from the present study furthermore demonstrate that the level of glycogen accumulation attained at 48–72 h after exercise is also correlated with GLUT-4 concentration. Because the rate of glycogen accumulation and GLUT-4 content were both correlated with percentage of type I fibers in the present study, it is possible that the difference between trained and untrained individuals with respect to glycogen accumulation is due to the different fiber type makeup of the two groups. This is probably not the case, however, because a recent swimming-training study in rats also demonstrated a higher rate of glycogen accumulation in trained than in untrained rats (28). In support of this interpretation, muscle glucose uptake was apparently higher in the trained than in the untrained individuals, as evidenced by the lower plasma glucose despite lower plasma insulin concentrations in the trained subjects over the 6 h immediately after exercise. There was also a strong tendency (r = –0.55, P = 0.06) for the rates of glycogen accumulation to be correlated with the area under the glucose curves over the initial 6 h after exercise. The higher rate of glycogen accumulation despite the lower prevailing plasma insulin concentrations in the trained individuals is impressive and clearly demonstrates the well-established finding of an enhanced insulin action on glucose uptake in trained individuals (13, 19, 20, 26), which is probably, at least in part, because of an increased amount of GLUT-4. The mechanisms responsible for the increased rate of glycogen accumulation do not involve differences in the prevailing serum free fatty acid concentration after exercise because free fatty acid levels were very similar in the two groups. Plasma catecholamine concentrations were also not different, ruling out the possibility of reduced counter-
regulatory action by these hormones in the trained subjects.

It is widely accepted that glycogen synthase, particularly the amount of this enzyme that is in the I form, is the major determinant of muscle glycogen accumulation after exercise (4). Glycogen synthase I activity in the present study was indeed twofold higher in trained than in untrained subjects immediately after exercise. However, the correlation between glycogen synthase I activity immediately after exercise and glycogen accumulation rates over the initial 6 h after exercise was not statistically significant (P = 0.08). In the present study, GLUT-4 content accounted for 44% of the variance in glycogen accumulation, whereas glycogen synthase I accounted for only 27% of the variance, indicating that muscle GLUT-4 content is more closely associated with glycogen accumulation rates than glycogen synthase I. Further support of the postulation that glycogen synthase is not the major determinant of glycogen accumulation rates after exercise comes from a recent study in rats by Ren et al. (33). Glycogen accumulation rates over the first 4 h after exercise were twofold higher in rats in which muscle GLUT-4 content had been elevated by means of 2 days of swimming training, despite similar glycogen synthase I activity in the swimming-trained and sedentary groups.

Numerous investigations have described the deleterious effects of muscle damage on glycogen accumulation, insulin sensitivity, and GLUT-4 in muscle (1, 8, 21, 35). Muscle damage probably did not result in reduced glycogen accumulation rates in skeletal muscle in the present study, as evidenced by the following findings: 1) exercise on a cycle ergometer has a low eccentric component; 2) the untrained subjects performed a 1-h bout of exercise on the cycle ergometer 3 wk before the glycogen depletion bout of exercise, which would have reduced muscle damage during the subsequent depletion ride (10); 3) although plasma CK values in untrained subjects were higher 48/72 h after exercise compared with preexercise values, these 48/72-h values were not significantly higher than in trained subjects, did not correlate with glycogen accumulation at these time points, and were not out of the normal range. Furthermore, Widrick et al. (35) found that muscle glycogen accumulation was not decreased over the initial 6 h, even after eccentric exercise.

There was no significant difference in accumulation rates between trained and untrained individuals between 6 and 48/72 h. The elevated muscle glycogen concentration at 48/72 h in the trained compared with untrained individuals was therefore mainly because of the difference in glycogen accumulation rates over the initial 6 h after exercise. It can thus be concluded that there was no significant difference in the slow phase of glycogen accumulation between the two groups. Whether this indicates that glycogen supercompensation is a phenomenon relative to “resting” glycogen levels and occurs in both trained and untrained individuals is not possible to determine from this study because preexercise biopsies were not obtained. The mean muscle glycogen concentration of 99 mmol/kg in untrained subjects at 48/72 h postexercise in the present study is not out of the previously reported range of values for resting muscle glycogen in untrained individuals (80–100 mmol/kg) (2, 16); however, the mean muscle glycogen concentration at 72 h postexercise for the three untrained subjects who consumed the diet for 3 days postexercise was ~130 mmol/kg.

We conclude that endurance training results in an increased ability to accumulate muscle glycogen after exercise and that this increase is associated with increased GLUT-4 content in trained muscle. This adaptation to training should be beneficial for performance of daily bouts of glycogen-depleting exercise.

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**REFERENCES**


