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# Nutrition and Exercise Determinants of Postexercise Glycogen Synthesis

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During the initial hours of recovery from prolonged exhaustive lower body exercise, muscle glycogen synthesis occurs at rates approximating 1–2 mmol•kg$^{-1}$ wet wt•hr$^{-1}$ if no carbohydrate is consumed. When carbohydrate is consumed during the recovery, the maximal rate of glycogen synthesis approximates 7–10 mmol•kg$^{-1}$ wet wt•hr$^{-1}$. The rate of postexercise glycogen synthesis is lower if the magnitude of glycogen degradation is small, if less than 0.7 gm glucose•kg$^{-1}$ body wt•hr$^{-1}$ is ingested, when the recovery is active, and when the carbohydrate feeding is delayed. The rate of postexercise glycogen synthesis is not reduced during the initial hours (<4) after eccentric exercise. For studies evaluating muscle glycogen synthesis in excess of 12 hours of recovery, average rates of glycogen synthesis are below 4 mmol•kg$^{-1}$ wet wt•hr$^{-1}$. Glycogen synthesis is known to be impaired for time periods in excess of 24 hours following exercise causing eccentric muscle damage. Following intense exercise resulting in high concentrations of muscle lactate, muscle glycogen synthesis occurs at between 15–25 mmol•kg$^{-1}$ wet wt•hr$^{-1}$. These synthesis rates occur without ingested carbohydrate during the recovery period and are maintained when a low intensity active recovery is performed.

Blood-borne glucose can be utilized by muscle during exercise; however, the majority of the glucose-6-phosphate flux through glycolysis is derived from muscle glycogen (18, 22). During prolonged exhaustive exercise, muscle glycogen stores decline to low levels and an athlete's endurance capacity and exercise performance become impaired (1, 18, 22, 31). The ingestion of carbohydrate during exercise can delay symptoms of fatigue and enhance exercise performance (17, 22). However, for athletes who participate in intermittent exercise and/or exhaustive exercise over successive days, the rapid replenishment of muscle glycogen stores is also of added importance. The ability to maximize muscle glycogen stores during restricted recovery intervals would give an athlete an advantage during repeated bouts of exercise.

This review will present a detailed evaluation of the applied research of postexercise glycogen synthesis. The data presented will reveal how to maximize

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muscle glycogen stores during the postexercise recovery period, and identify conditions that may limit the ability of skeletal muscle to synthesize glycogen during recovery.

## Postexercise Glycogen Synthesis

Research on skeletal muscle postexercise glycogen synthesis in humans has been performed since the 1960s. To aid the review process, studies have been grouped into three divisions according to exercise intensity and the recovery duration from which glycogen synthesis rates were calculated. These categories are, (a) submaximal exercise and short-term recovery (<12 hrs), (b) submaximal exercise and longer term recovery (>12 hrs), and (c) high intensity exercise (Tables 1, 2, and 3, respectively). These divisions are required because of the decreased rate of glycogen synthesis across time (9, 12, 40, 54, 65, 80), and the different systemic and intramuscular glycogenic precursor conditions accompanying prolonged low intensity versus short-term high intensity exercise (11, 33, 60, 78, 79).

### *Submaximal Exercise and Short-Term Recovery*

***The Pattern of Postexercise Glycogen Synthesis.*** Danforth (24) indicated that the glycogen synthetase activity ratio decreased exponentially with an increasing glycogen concentration. Regardless of the inadequacy of the activity ratio in detecting prolonged in vivo glycogen synthetase activity (46, 74), these findings provided evidence for a potential nonlinear relationship between the rate of glycogen synthesis and the time of recovery. Although no single investigation with human muscle has demonstrated such a relationship, several studies have documented reduced rates of glycogen synthesis with increased recovery times (10, 12, 41, 54, 65, 80). The reduced rates of glycogen synthesis during recovery have been jointly explained by declines in the insulin-like effect of muscular contraction (increased insulin sensitivity) (39, 81), increased glycogen concentrations and the phosphorylation of synthetase (25, 69), and decreased muscle blood flow (35).

When the results from studies that used liquid glucose (>0.35 $gm \bullet kg^{-1}$ body $weight \bullet hr^{-1}$) or high carbohydrate solid food diets (>60% Kcals from CHO) during the recovery period are combined and graphed, an exponential relationship exists between the rate of glycogen synthesis and the recovery time (Tables 1 and 2; Figure 1). Large variability exists in the rates of glycogen synthesis between 0 and 12 hrs postexercise. As will be discussed in this review, the variability may be due to (a) methodological differences in the type, intensity, and duration of exercise, (b) the magnitude of glycogen degradation, (c) the type of exercise, and (d) the amount, type, and times of carbohydrate feedings.

Comment must also be made of the calculation of glycogen synthesis rates. As indicated by Figure 1, the rate of glycogen synthesis is a time average that will be affected by the total time period and the time since the start of recovery. It is also unclear whether the rate of glycogen synthesis during the first 12 hrs of recovery is linear or exponential. Nevertheless, research has frequently reported increases in muscle glycogen during the first 2 to 4 hrs of recovery as a linear synthesis rate (7, 8, 10, 40, 41, 68). Unfortunately, there is no data of glycogen synthesis after prolonged steady-state exercise during a recovery interval of less than an hour.

## Table 1

**Review of Research Using Prolonged Low Intensity Exercise to Evaluate Immediate Postexercise Muscle Glycogen Synthesis in Human Subjects**

| Study | Exercise | Δ G | [G] | CHO/subjects | Timing | Period | Rate | (*n*) |
|---|---|---|---|---|---|---|---|---|
| Ahlborg (1) | cycling 60% $VO_2$max | 63.8 | 21.1 | 0.0 | | 0-1 | 1.7 | 6 |
| | -exhaustion | 49.4 | 18.3 | g 0.8 | infusion | 0-1 | 11.1 | 1 |
| | | 59.4 | 16.7 | g 0.6 | infusion | 0-1 | 22.8 | 1 |
| Bergstrom (6) | 12-hr fast | | 77.2 | g 1.0 | infusion | 0-4 | 4.9 | 9 |
| | | | 76.1 | f 1.0 | infusion | 0-4 | 4.6 | 10 |
| | b) prolonged exercise | | 7.8 | g 1.0 | infusion | 0-2 | 25.0 | 8 |
| | -single leg cycling | | 57.8 | " | " | 2-4 | 14.7 | 8 |
| | | | 5.0 | f 1.0 | infusion | 0-2 | 12.7 | 8 |
| | | | 20.6 | " | " | 2-4 | 9.4 | 8 |
| | -single leg rest | | 76.1 | g 1.0 | infusion | 0-2 | 2.8 | 8 |
| | | | 81.7 | " | " | 2-4 | 2.5 | 8 |
| | | | 73.3 | f 1.0 | infusion | 0-2 | 3.3 | 8 |
| | | | 80.0 | " | " | 2-4 | 2.2 | 8 |
| Hultman (36) | a) cycling 60% W170 | 59.4 | 21.0 | 0.0 | | 0-1 | 0.5 | 10 |
| | b) intense int. cycling | 80.0 | 1.7 | 0.0 | | 0-4 | 3.3 | 5 |
| | -exhaustion | | | | | | | |
| Roch-Norlund (71) | single leg cycling | | | g 2.0 | | | | |
| | -prolonged | | | rested normals | infusion | 0-2 | 2.7 | 8 |
| | | | | | | 2-4 | 2.6 | 8 |
| | | | | diabetic | | 0-2 | –0.8 | 6 |
| | | | | | + insulin | 2-4 | 7.8 | 6 |

cont.

**Table 1 (Cont.)**

| Study | Exercise | ΔG | [G] | CHO/subjects | Timing | Period | Rate | (*n*) |
|---|---|---|---|---|---|---|---|---|
| | | | | exerc. normals | | 0-2 | 25.1 | 8 |
| | | | | | | 2-4 | 14.5 | 8 |
| | | | | diabetic | | 0-2 | 11.3 | 6 |
| | | | | | (+ insulin) | 2-4 | 17.9 | 6 |
| Nilsson (59) | 12–14-hr fast | | | g 1.0 | infusion | | | |
| | | | 281.0 | -liver | | 0-4 | 19.6 | 6 |
| | | | 88.0 | -muscle | | 0-4 | 6.1 | 6 |
| | | | | f 1.0 | infusion | | | |
| | | | 261.0 | -liver | | 0-4 | 68.6 | 5 |
| | | | 93.3 | -muscle | | 0-4 | 5.75 | 5 |
| Piehl (65) | 2 hr - exhaustion | 103 | 22.0 | sd 60% CHO | | 0-5 | 8.2 | 4 |
| | | | 63.0 | " | | 5-10 | 4.4 | 4 |
| | | | 85.0 | " | | 10-46 | 1.0 | 4 |
| Piehl (66) | cycling 70–80% | | | | | | | |
| | a) trained | 76 | 43.0 | sd high CHO | | 0-10 | 2.4 | 6 |
| | | | | | | 0-46 | 1.6 | 6 |
| | b) untrained | 65 | 16.0 | " | | 0-10 | 2.4 | 6 |
| Maehlum (54) | cycling 70% $VO_2max$ | 61.6 | | sd high CHO | | 0-46 | 1.4 | 6 |
| | -to exhaustion | | 15.0 | normal | | 10-4 | 7.2 | 6 |
| | | | 43.8 | | | 4-12 | 2.4 | 6 |
| | | | 15.0 | diabetic | | 0-4 | 6.4 | 6 |
| | | | 40.6 | | | 4-12 | 2.0 | 6 |
| Maehlum (55) | 98 min cycling | 48 | | sd high CHO | | | | |
| | -75% $VO_2max$ | | 12.4 | -diabetics | + insulin | 0-4 | 7.5 | 5 |
| | | | 13.1 | no insulin | | 0-4 | 4.6 | 5 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Maehlum (51) | cycling-exhaustion | 59.8 | 21.6 | g 0.5 | infusion | | | |
| | -60–70% $VO_2$max | | | | normals | 0-1 | 13.6 | 7 |
| | | 34.8 | 33.9 | | diabetics | 0-2 | 9.6 | 4 |
| Maehlum (53) | cycling 70% $VO_2$max | 48.8 | 21.6 | 0.0 | | 0-4 | 1.8 | 5 |
| | exhaustion | | | | | | | |
| Blom (7) | cycling 70% $VO_2$max | | | lg 0.35 | 0,2,4,6 | 0-8 | 2.3 | 4 |
| | -exhaustion | | | lg 0.7 | " " | 0-8 | 4.6 | 4 |
| | | | | lg 1.0 | " " | 0-8 | 5.6 | 4 |
| Bonen (12) | 30 min 70–80% $VO_2$max | | | | | | | |
| | cycling | | | | | | | |
| | a) passive recovery | 23.9 | 58.9 | lg 0.75 | 0,2 | 0-2 | 5.0 | 3 |
| | | | 68.9 | " | " | 2-4 | 0 | 3 |
| | b) active recovery | | 47.2 | lg 0.75 | 0,2 | 0-2 | –9.4 | 4 |
| | | | 28.3 | " | " | 2-4 | –6.1 | 4 |
| Blom (10) | int. cycling 75% $VO_2$max | 65 | 32 | lg 0.175 | 0,2,4 | 0-6 | 2.1 | 5 |
| | -exhaustion | " | " | " | " | 0-2 | 7.5 | 5 |
| | 84 | | 15 | lg 0.35 | | 0-6 | 5.8 | 5 |
| | | " | " | " | " | 0-2 | 7.5 | 5 |
| | 100 | | 23 | lg 0.7 | | 0-6 | 5.7 | 5 |
| | | " | " | " | " | 0-2 | 9.0 | 5 |
| | 84 | | 8 | lu 0.35 | | 0-6 | 6.2 | 5 |
| | | " | " | " | " | 0-2 | 7.5 | 5 |
| | 83 | | 23 | lf 0.35 | | 0-6 | 3.2 | 5 |
| | | " | " | " | " | 0-2 | 4.0 | 5 |
| Ivy (40) | 70 min cycling | | 35.8 | lg 1.0 | 0 | 0-2 | 7.7 | 12 |
| | -68–88% $VO_2$max | | 51.2 | " | " | 2-4 | 4.3 | 12 |
| | | | 30.9 | 0 | -- | 0-2 | 2.5 | 12 |
| | | | 35.9 | lg 1.0 | 2 | 2-4 | 4.1 | 12 |
| Ivy (41) | 2 hr cycling | | 36.4 | 0.0 | -- | 0-2 | 1.4 | 8 |
| | -62–75% $VO_2$max | | 35.8 | lg 0.75 | 0,2 | 0-2 | 5.2 | 8 |
| | | | 32.0 | lg 1.5 | 0,2 | 0-2 | 5.8 | 8 |

cont.

**Table 1 (Cont.)**

| Study | Exercise | ΔG | [G] | CHO/subjects | Timing | Period | Rate | (*n*) |
|---|---|---|---|---|---|---|---|---|
| Reed (68) | 2 hr cycling | | 27.4 | lg 0.75 | 0 | 0-2 | 6.2 | 8 |
| | | | 39.6 | " | 0,2 | 2-4 | 4.5 | 8 |
| | | | 24.5 | sg 0.75 | 0 | 0-2 | 6.3 | 8 |
| | | | 37.1 | " | 0,2 | 2-4 | 4.7 | 8 |
| | | | 30.6 | lg 0.75 | infusion | 0-2 | 7.0 | 8 |
| | | | 44.6 | " | " | 2-4 | 4.3 | 8 |
| Blom (8) | 3 x 20 min cycling | | 22.0 | lg 1.4,0.7,0.7 | 0,1,2 | 0-3 | 9.3 | 5 |
| | -75% $VO_2$max | | 14.0 | lg | infusion | 0-3 | 8.3 | 5 |
| Kiens (44) | exhaustive cycling | | | 70% CHO, high G.I. | | 0-6 | 9.8 | 6 |
| | -? % $VO_2$max | | | 70% CHO, low G.I. | | 0-6 | 5.9 | 6 |
| Zachweija (80) | 1 and 2 leg cycling | 94 | 57.0 | lg 0.70 | 20 min int. | 0-2 | 10.5 | 6 |
| | -70% $VO_2$max | | 78.0 | " | " | 2-6 | 7.5 | 6 |
| Doyle (25) | day 1-concentric | 49 | 80.7 | lg 0.70 | " | 0-2 | 4.7 | 6 |
| | -eccentric | | 90.1 | " | " | 2-6 | 2.2 | 6 |
| | day 2-concentric | | | lg 1.6 gm | 15 min int. | 0-4 | 9.9 | 10 |
| | eccentric | | | " | " | 0-4 | 9.1 | 10 |
| | 10 | | | " | " | 0-4 | 11.0 | 10 |
| | | | | | " | " | 0.4 | 8.1 |

Glycogen degradation (DG, mmol•kg$^{-1}$ wet wt); postexercise glycogen concentration ([G], mmol•kg$^{-1}$ wet wt); postexercise carbohydrate ingestion (CHO, gm•kg$^{-1}$•hr$^{-1}$); postexercise glycogen synthesis rate (Rate, mmol•kg$^{-1}$•hr$^{-1}$ wet wt); time of CHO ingestion (time, hrs); period of glycogen synthesis rate calculation (period, hrs); type of carbohydrate = glucose (g), sucrose (u), fructose (f), mixed diet (d), and liquid (l), or solid (s). Data reported as mmol•kg$^{-1}$ dry wt in the literature were divided by 4.1 to account for water weight. Research is listed in chronological order.

**Table 2**

**Review of Research Evaluating Long-Term (>12 hr) Postexercise Muscle Glycogen Synthesis in Human Subjects**

| Study | Exercise | ΔG | [G] | CHO/subjects | Timing | Period | Rate | (*n*) |
|---|---|---|---|---|---|---|---|---|
| Bergstrom (5) | a) single-leg int. cycling |  | 10.6 | s high CHO |  | 0-12 | 9.9 | 2 |
|  | -exhaustion |  | 61.6 | " |  | 12-60 | 1.8 | 2 |
|  | b) rest |  | 10.5 | " |  | 0-12 | 1.6 | 2 |
|  |  |  | 32.8 | " |  | 12-60 | 0.1 | 2 |
| Hultman (36) | c) single leg cycling |  |  |  |  |  |  |  |
|  | -nonexercise leg |  | 44.2 | sd high CHO |  | 0-96 | 0.5 | 2 |
|  | -exercised leg |  | 7.5 | sd high CHO |  | 0-96 | 1.9 | 2 |
| Costill (18) | 3 x 16.1 km run @ 80% $VO_2max$ |  |  |  |  |  |  |  |
|  | a) days 1–2 | 38.9 | 77.8 | sd |  | 0-24 | 0.5 | 5 |
|  | b) days 2–3 | 22.8 | 65.5 | sd |  | 0-24 | –0.2 | 5 |
|  | c) days 3–5 (rest) | 26.1 | 35.0 | sd |  | 0-48 | 1.4 | 5 |
| Piehl (65) | 2 hrs - exhaustion | 103.0 | 22.0 | sd 60% CHO |  | 0-5 | 8.2 | 4 |
|  |  |  | 63.0 | " |  | 5-10 | 4.4 | 4 |
|  |  |  | 85.0 | " |  | 10-46 | 1.0 | 4 |
| Piehl (66) | int. cycling 70–80% $VO_2max$-exhaustion |  |  |  |  |  |  |  |
|  | a) trained | 76.0 | 43.0 | sd high CHO |  | 0-10 | 2.4 | 6 |
|  |  |  |  |  |  | 0-46 | 1.6 | 6 |
|  | b) untrained | 65.0 | 16.0 | " |  | 0-10 | 2.4 | 6 |

cont.

Table 2 (Cont.)

| Study | Exercise | ΔG | [G] | CHO/subjects | Timing | Period | Rate | (*n*) |
|---|---|---|---|---|---|---|---|---|
| | | | | | | 0-46 | 1.4 | 6 |
| Maehlum (52) | cycling - exhaustion | 49.0 | 20.0 | 0.0 | | 0-14 | 1.7 | 6 |
| | -70% $VO_2$max | " | 44.0 | lg 1.4 | 14 | 14-16.25 | 5.3 | 6 |
| | | 45.0 | 16.0 | lg 1.4 | 0.25 | 0.25-2.25 | 7.1 | 5 |
| Kochan (46) | 60 min cycling | 107.8 | 12.0 | sd high CHO | | 0.5-24 | 5.0 | 6 |
| | -70% $VO_2$max | | 130.0 | " " | | 24-48 | 3.0 | 6 |
| | | | 201.9 | " " | | 48-96 | 0.5 | 6 |
| Costill (21) | 16.1 km run | | 56.0 | sd si CHO | | 0-24 | 3.5 | 4 |
| | -80% $VO_2$max | | | | | 24-48 | 0.9 | 4 |
| | | | 53.0 | sd com CHO | | 0-24 | 3.2 | 4 |
| | | | | | | 24-48 | 0.3 | 4 |
| | | | 71.3 | sd 25% CHO | | 0-24 | 0.2 | 4 |
| | | | 49.3 | sd 50% CHO | | 0-24 | 1.0 | 4 |
| | | | 55.3 | sd 70% CHO | | 0-24 | 2.2 | 4 |
| Sherman (75) | -depletion-taper sequence | | | | | | | |
| | -73% $VO_2$max running | | | | | | | |
| | day 4 | | 86.3 | sd low CHO | | 0-72 | 1.7 | 6 |
| | day 4 | | 133.9 | sd high CHO | | 0-72 | 1.0 | 6 |
| | day 4 | | 133.3 | sd mod CHO | | 0-72 | 0.4 | 6 |
| Sherman (74) | 42.2 km run | 171.0 | 25.0 | | | | | |
| | a) active recovery | | | sd high CHO | | 0-24 | 2.0 | 5 |
| | | | | | | 24-120 | 0.6 | 5 |
| | b) rest | | | sd high CHO | | 0-24 | 2.5 | 5 |
| | | | | | | 24-120 | 0.5 | 5 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| Kuipers (48) | 30 min conc. cycling | 24.0 | 71.6 | 0.0 | 0-24 | 1.0 | 6 |
| | 30 min ecc. cycling | 0.0 | 97.6 | 0.0 | 0-24 | –0.5 | 6 |
| | 30 min ecc. cycling | 0.0 | 101.5 | 0.0 | 0-24 | –1.3 | 6 |
| O'Reilly (62) | 45 min eccentric cycling | 33.0 | 52.0 | sd CHO 54% Kcal | 0-240 | –0.06 | 5 |
| Blom (9) | exhaustive running@ 70% $VO_2$max | | | | | | |
| | -trained | 123.0 | 57.0 | sd 68% CHO | 0-22 | 3.18 | 6 |
| | | | | | 22-70 | 0.17 | 6 |
| | -untrained | 76.0 | 26.0 | sd 68% CHO | 0-22 | 2.77 | 6 |
| | | | | | 22-70 | –0.46 | 6 |
| Kirwan (45) | 5 days of 20km day @ 80% $VO_2$max | | | | | | |
| | sd 8.0 gm CHO/kg | | | sd 3.8 gm CHO/kg | 216-288 | 0.31 | 10 |
| | sd 4.0 gm CHO/kg | | | " " | 216-288 | 0.78 | 10 |
| Costill (19) | Single leg eccentric & 1 hr of 2-legged cycling | | | | | | |
| | -eccentric leg | 41.0 | | sd 8.5 gm CHO/kg | 0-24 | 1.5 | 4 |
| | | | | | 24-72 | 1.0 | 4 |
| | | | | sd 4.25 gm CHO/kg | 0-24 | 1.2 | 4 |
| | | | | | 24-72 | 0.2 | 4 |
| | -concentric leg | 22.0 | | sd 8.5 gm CHO/kg | 0-24 | 3.5 | 4 |
| | | | | | 24-72 | 1.3 | 4 |
| | | | | sd 4.25 gm CHO/kg | 0-24 | 2.5 | 4 |
| | | | | | 24-72 | 0.8 | 4 |

Glycogen degradation (DG, mmol•kg$^{-1}$ wet wt); postexercise glycogen concentration ([G], mmol•kg$^{-1}$ wet wt); postexercise carbohydrate ingestion (CHO, gm•kg$^{-1}$•hr$^{-1}$); postexercise glycogen synthesis rate (Rate, mmol•kg$^{-1}$•hr$^{-1}$ wet wt); time of CHO ingestion (time, hrs); period of glycogen synthesis rate calculation (period, hrs); type of carbohydrate = glucose (g), sucrose (u), fructose (f), mixed diet (d), and liquid (l), or solid (s). Data reported as mmol•kg$^{-1}$ dry wt in the literature were divided by 4.1 to account for water weight. Research is listed in chronological order.

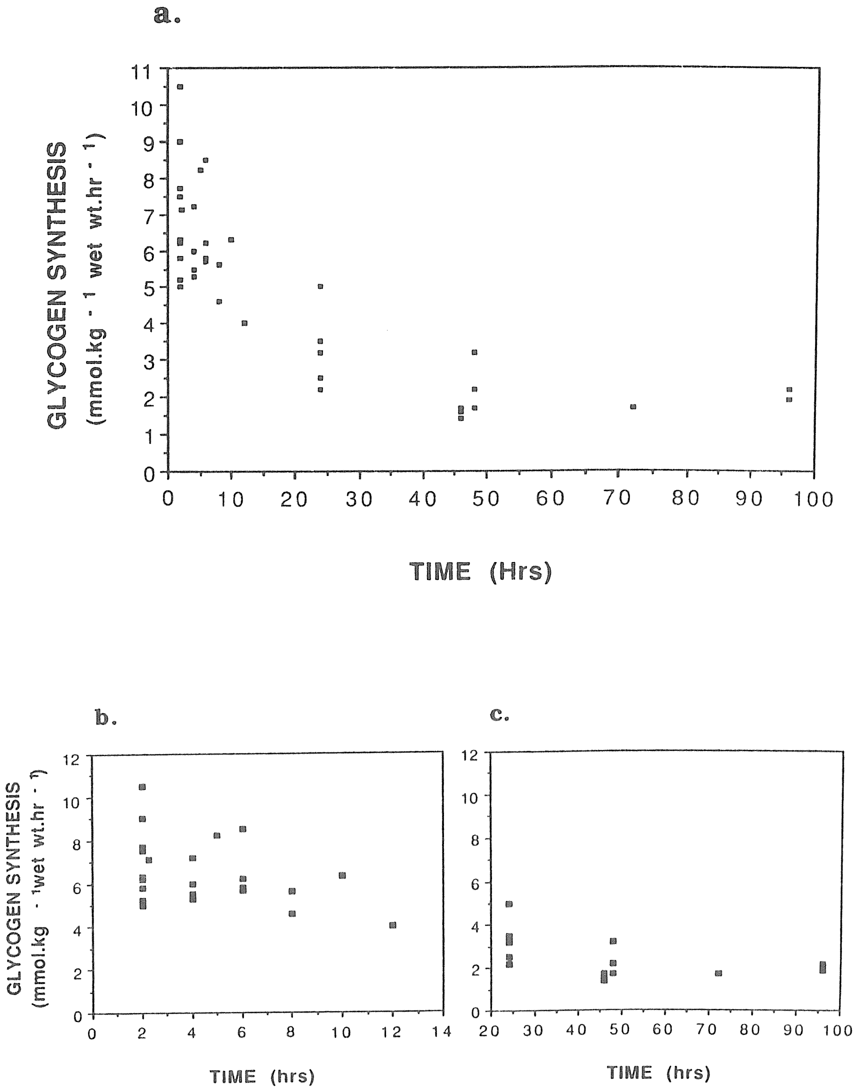


Figure 1 — (a) Decline in the rate of muscle glycogen synthesis during recovery from prolonged exhaustive submaximal exercise. Data were obtained from studies that provided either no carbohydrate or liquid and/or solid carbohydrate feedings after cycling or running exercise (Table 1 and 2). The relationship between glycogen synthesis and the duration of recovery included in the rate calculation (Time) appears to be exponential. (b) The variation in the rate of glycogen synthesis between 0 and 12 hrs recovery, and (c) between 20 and 60 hrs of recovery. Graphs (a) and (b) indirectly indicate the large decrease in glycogen synthesis after 20 hrs of recovery.

A possible explanation for this is the error of the muscle biopsy method (i.e., in randomly sampling exercised muscle of a heterogenous fiber type) and the muscle glycogen assay procedure. It is questionable whether whole muscle glycogen increases amounting to less than 8 mmol•$kg^{-1}$ wet wt could be reliably detected from muscle biopsy sampling and subsequent enzymatic analysis of muscle glucosyl unit concentrations (personal observations). Determination of human postexercise glycogen synthesis after intense exercise may be a better model for investigating this relationship, due to the larger rates of glycogen synthesis.

Another concern that should be considered when evaluating research of muscle glycogen metabolism is the reduced rate of glycogen synthesis in regions previously traumatized by the biopsy procedure (20). However, comparison of the data presented in this review does not definitively detect unusually low glycogen synthesis rates due to repeated biopsies from the same region of a muscle.

The distribution of postexercise glycogen synthesis among fast and slow twitch muscle fibers was reported by Vollestad et al. (79). Subjects completed cycle exercise at 75% $VO_2$max until exhaustion and were fed glucose solutions at 0, 60, and 180 min of recovery (1.4, 0.7, and 0.7 g•$kg^{-1}$ body wt, respectively). Glycogen concentration changes in muscle fiber types were estimated by PAS stain absorbance regression to biochemically determined whole muscle glycogen concentrations. Results indicated that glycogen resynthesis occurred at a 65% faster rate in type IIa and IIb fibers compared to type I fibers during 60 min of recovery. Similar results were reported by Kirwan et al. (45) during 3 days of rest following 5 days of intense running training. These results may have important implications to repeated submaximal exercise performed with 1- to 2-hr recovery intervals. Slow twitch muscle fibers are recruited first during submaximal exercise, and the delayed synthesis of muscle glycogen in these fibers may cause unexpected sensations of fatigue during subsequent bouts of exercise.

***The Magnitude of Glycogen Degradation.*** Biochemical research has revealed the independent role of the glycogen concentration in altering the phosphorylation state and activity of synthetase (24, 65, 70). Unfortunately, limited applied research has been performed to evaluate the relationship between the magnitude of glycogen degradation and postexercise glycogen synthesis. Bonen et al. (12) investigated postexercise glycogen synthesis in muscle having large (>80%, LG) and small (<35%, SM) glycogen degradation (Table 1). The rates of postexercise glycogen synthesis for the LG and SM trials amounted to (a) 0–2 hr: 15.3 and 5.0 mmol•$kg^{-1}$ wet wt•$hr^{-1}$, respectively, and (b) 2–4 hr: 1.9 and 0 mmol•$kg^{-1}$ wet wt•$hr^{-1}$, respectively. Glycogen synthesis during the first 2 hrs of recovery was higher when prior glycogen degradation was large. However, the fact that group sizes were unequal and that exercise during the LG trials finished with intermittent intense exercise biased this comparison.

Due to the methodological difficulties inherent in the Bonen et al. findings, a recent investigation was performed to directly ascertain whether different magnitudes of glycogen degradation result in differing rates of postexercise glycogen synthesis (80). Cycle ergometer exercise was performed by 6 subjects at 75% $VO_2$max (one leg and two leg $VO_2$max, respectively), with one leg exercising for 1 hr (L-60) and the other for 30 min (L-30) (Table 1). The L-60 leg also performed 10 × 1-min bouts of cycling at 120% $VO_2$max. The exercise resulted in a significantly larger decline in muscle glycogen in L-60. During a 6-hr passive

recovery, samples of a 24% CHO solution were ingested every 20 min, providing 0.7 gm CHO•$kg^{-1}$ body wt•$hr^{-1}$. Results indicated that both the rate of glycogen synthesis and glycogen synthetase activity in L-60 were significantly higher than in L-30. Consequently, the magnitude of glycogen degradation and/or exercise duration does influence the rate of postexercise glycogen synthesis.

Interestingly, evidence of this fact was published by Sherman et al. (75) in their study of differing dietary regimens on glycogen supercompensation (Table 2). During the final 3 days of a high carbohydrate diet, greater glycogen synthesis occurred when the muscle glycogen concentration was lowest (see Figure 5 and the sections on Long-Term Recovery >12 hrs).

***The Type of Exercise.*** The influence of the type of exercise on postexercise glycogen synthesis during the first 12 hrs of recovery has not been widely researched. A recent study focused on the immediate 4 hrs of postexercise recovery between two trials of eccentric and concentric leg extension exercise (25) (Table 1). The results indicated no impairment of glycogen synthesis in the immediate 4-hr recovery period, but significantly different rates of synthesis during 4 hrs of recovery after concentric exercise performed 40 hrs later. A more detailed description of this study is given in the section on prolonged postexercise glycogen synthesis. The remaining research that has focused on glycogen synthesis during short-term recovery involves high intensity exercise, and this will be discussed later.

***CHO Feedings: Carbohydrate Infusion.*** The research evaluating short-term postexercise glycogen synthesis is summarized in Table 1. The earliest studies were published in 1967 and pertained to glycogen synthesis with CHO infusion and/or no CHO supplementation (1, 6, 36). Exhaustive submaximal cycling resulted in meager rates of glycogen synthesis during the first hour of recovery when no CHO was provided (0.5–1.7 mmol•$kg^{-1}$•$hr^{-1}$ wet wt) (1, 36). Conversely, postexercise infusion of 1.0 g glucose•$kg^{-1}$ body wt•$hr^{-1}$ yielded a glycogen synthesis rate of 25.0 mmol•$kg^{-1}$ wet wt•$hr^{-1}$ during the first 2 hrs of recovery (6). Glucose infusion resulted in blood glucose concentrations in excess of 20 mmol•$l^{-1}$, and blood insulin responses were not measured. When comparing these studies, the rate of glycogen synthesis during infusion was larger with longer infusion durations (1), prior exercise (6, 51), and when glucose rather than fructose was infused (6, 59).

Roch-Norlund et al. (71) also compared postexercise skeletal muscle glycogen synthesis when glucose infusion was combined with insulin infusion. Unfortunately, blood insulin concentrations were not measured and half the rate of glucose infusion was used in the insulin infusion experiments. These flaws complicated comparisons between glycogen synthesis rates. However, the results indicated that blood glucose concentrations were twice as high with the added carbohydrate and no infused insulin, and glycogen synthesis rates between 2 and 4 hrs of recovery were also higher (Table 1). These findings implied that either insulin was not directly important for postexercise glucose uptake or that normal physiological concentrations of insulin were adequate to maximize glucose uptake by muscle. This topic has been reviewed elsewhere (39).

The discrepancies in glycogen synthesis between fructose and glucose infusion were partially clarified when Nilsson et al. (59) compared the effects of fructose and glucose infusion on liver and muscle glycogen synthesis after a 12- to 14-hr fast. Liver glycogen synthesis was more than threefold higher during

fructose infusion compared to glucose, while muscle glycogen synthesis was similar for both sources of CHO. It is surprising that fructose and glucose infusion resulted in similar muscle glycogen synthesis rates. However, Nilsson used fasting rather than exercise to decrease glycogen stores. This regimen would cause greater preliminary glycogen loss in the liver, compared to muscle, and would not stimulate exercise induced increases in insulin sensitivity and glucose uptake by skeletal muscle. Consequently, it is difficult to provide a valid comparison of the results of Nilsson et al. (59) to those of Ahlborg et al. (1) and Bergstrom and Hultman (6).

More recently, Reed et al. (68) and Blom (8) compared rates of postexercise muscle glycogen synthesis between digested (liquid and/or solid) versus infused carbohydrate (Table 1). During the Reed study, similar amounts of carbohydrate (3 $g \bullet kg^{-1}$ body wt) were provided in each trial. Peak blood glucose during the infusion increased to almost double that of the oral carbohydrate trials (9.6 vs. 4.7–5.6 $mmol \bullet l^{-1}$), and insulin concentrations increased progressively during recovery but were not higher during the infusion trial. The rate of glycogen synthesis during the first 2 hrs of recovery during the infusion trial (7.0 $mmol \bullet kg^{-1}$ wet $wt \bullet hr^{-1}$) did not differ from the oral CHO trials. Nevertheless, the rate of glycogen synthesis during glucose infusion was low compared to previous research (11.0–25.0 $mmol \bullet kg^{-1}$ wet $wt \bullet hr^{-1}$) (1, 6).

Similar rates of postexercise glycogen synthesis between infused and oral glucose loads were also reported by Blom et al. (8) (8.3 and 9.3 $mmol \bullet kg^{-1}$ wet $wt \bullet hr^{-1}$). However, Blom infused glucose to mimic the blood glucose response during the oral feeding trial, and less than half the glucose was required during the infusion compared to oral feeding (96 $\pm$ 12 vs. 242 $\pm$ 12 gm, respectively). During the oral feeding trial, blood insulin concentrations were approximately double that of the infusion trial. These results indicated that glucose uptake by exercised muscle and subsequent synthesis to glycogen could occur with only minor increases in circulating insulin. Furthermore, the lower total infused glucose relative to ingestion indicated considerable glucose disposal in tissues other than skeletal muscle when glucose is ingested. It remains unclear why the moderate increases in blood glucose reported by Reed et al. (68) did not result in larger rates of glycogen synthesis, as initially reported by Ahlborg et al. (1), Bergstrom and Hultman (6), and Maehlum (51).

Research on postexercise glycogen synthesis has also investigated glycogen metabolism in diabetic patients (51, 52, 53, 54, 55, 71). A complete review of this literature and more recent investigations is beyond the scope of this review. Nevertheless, these early studies provided evidence of diabetic insulin resistance, as normal subjects had greater rates of postexercise glycogen synthesis than diabetic patients with combined insulin and glucose infusion (Table 1). Interestingly, insulin infusion did dramatically increase glycogen synthesis in the diabetic patients compared to glucose infusion alone.

***CHO Feedings: Carbohydrate Concentration.*** Since the late 1960s, carbohydrate restriction after exercise was known to result in low rates of postexercise glycogen synthesis (5, 36) (Table 1). In addition, the synthesis of muscle glycogen was shown to be influenced by a combination of the magnitude of glycogen degradation and the amount of dietary carbohydrate intake (5). Subsequent research during the 1970s revealed that high carbohydrate diets (>60% Kcals from CHO) consumed after prolonged exercise resulted in glycogen synthesis rates

approximating 7–8 mmol•$kg^{-1}$ wet wt (54, 65), thus confirming the earlier pioneering research of Bergstrom and Hultman.

Blom et al. (7) published the first results that compared rates of postexercise glycogen synthesis when consuming differing quantities of oral carbohydrate following cycling to exhaustion at 75% $VO_2max$. Subjects ingested liquid glucose feedings at a rate of 0.35, 0.7, and 1.0 gm•$kg^{-1}$ body wt•$hr^{-1}$. Glycogen synthesis rates during 8 hrs of recovery did not differ between the 0.7 and 1.0 gm trials but were both significantly different from the 0.35 gm trial. Similar findings were reported by Blom et al. (10) and Ivy et al. (40, 41) for a 2-hr recovery period (Table 1). Consequently, the rate of glycogen synthesis during the immediate recovery period (0–2 hrs) from cycling exercise was largest (7–9 mmol•$kg^{-1}$ wet wt) when a liquid glucose feeding approximating 0.7 gm•$kg^{-1}$ body wt•$hr^{-1}$ was ingested.

It is important to emphasize that the CHO feeding rates used by Blom et al. (7, 10) and Ivy et al. (40, 41) provided in excess of 400 g of CHO during a 4-hr period. Ivy et al. (41) revealed that based on a 10-kg muscle mass, between 25 and 35% of the ingested CHO was converted to muscle glycogen. The larger the quantity of ingested carbohydrate, the smaller the glycogen conversion percentage. Consequently, the majority of ingested glucose after exercise is not involved in skeletal muscle glycogen synthesis. It remains unclear whether this low efficiency is due to a peripheral utilization limitation or a limitation of supply (i.e., blood flow and muscle perfusion).

Caution should be used when applying these results to different exercise conditions. The dispersion of data concerning postexercise glycogen synthesis rates and the concentration of oral carbohydrate feedings from different studies is presented in Figure 2. Although specific research may have documented that the ingestion of 0.7 gm CHO•$kg^{-1}$ body wt•$hr^{-1}$ was adequate to elicit maximal rates of postexercise glycogen synthesis, this dosage may not be sufficient for all exercise conditions. For example, this review will show that postexercise glycogen synthesis is also influenced by the magnitude of glycogen degradation and the intensity of exercise. Furthermore, the previously identified carbohydrate dosage rate was determined when exercise was performed solely by the lower body. There is a lack of research for the carbohydrate concentrations needed for maximal glycogen synthesis following total body exercise. It is possible that the greater exercised muscle mass in total body exercise would lower the blood glucose response to oral carbohydrate ingestion, thereby necessitating a larger quantity of carbohydrate feeding for a given rate of postexercise muscle glycogen synthesis. Research is needed to clarify this thesis.

***CHO Feedings: Timing of Carbohydrate Ingestion.*** The first study to report different rates of glycogen synthesis when altering the time of postexercise carbohydrate ingestion was that of Maehlum et al. (52) (Table 2). These authors provided liquid solutions of 1.4 gm glucose•$kg^{-1}$ body wt•$hr^{-1}$ at either 15 min or 14 hrs postexercise. Although the focus of the study was on long-term glycogen synthesis, the results indicated that the rate of glycogen synthesis was higher when the CHO was ingested 15 min postexercise rather than 14 hrs postexercise.

The same topic was refined by Ivy et al. (40), who focused on the rate of glycogen synthesis during the more immediate hours of recovery. These researchers provided subjects with liquid glucose feedings of 1.0 gm•$kg^{-1}$ body wt•$hr^{-1}$ immediately postexercise, or at 2 hrs postexercise. When carbohydrate

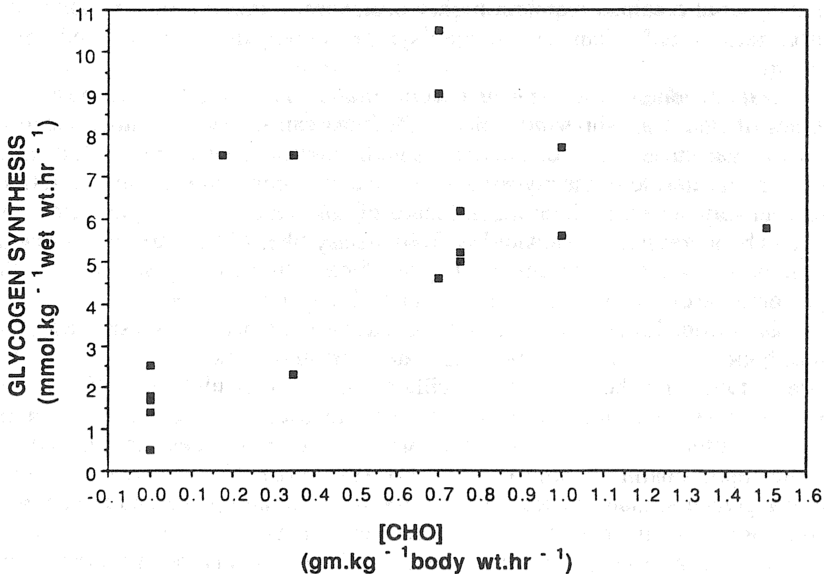


**Figure 2 — Relationship between postexercise glycogen synthesis and the rate of carbohydrate feedings. Glycogen synthesis appears to be maximal with a CHO ingestion approximately 0.7 gm•kg body wt$^{-1}$•hr$^{-1}$. Nevertheless, this rate of CHO ingestion may not be ideal for all types, durations, and/or intensities of prolonged submaximal exercise.**

feeding was delayed, the rate of glycogen synthesis was approximately half of that during the first 2 hrs of the immediate feeding trial (Table 1). The difference in glycogen synthesis resulted despite similar total postexercise muscle glycogen concentrations. There is a discrepancy between the results of Maehlum et al. (52) (Table 2) and Ivy et al. (40). The higher rate of glycogen synthesis after 14 hrs of CHO restriction reported by Maehlum may be a reflection of the lower muscle glycogen concentration compared to the postexercise conditions of Ivy. As previously explained, these results are an example of how the postexercise muscle glycogen concentration may be more influential in determining the rate of glycogen synthesis than the amount of ingested carbohydrate.

Potential differences between glycogen synthesis rates from bolus or serial feedings have been overlooked by research. The work of Ivy et al. (40, 41), Blom et al. (7, 10), and Reed et al. (68) involved bolus feedings of carbohydrate at 0, 2, and/or 4 hrs postexercise. Zachwieja et al. (80) provided serial feedings (every 20 min) of a CHO solution totaling 0.7 gm CHO•kg$^{-1}$ wet wt•hr$^{-1}$, and reported a glycogen synthesis rate approximating 10 mmol•kg$^{-1}$ wet wt•hr$^{-1}$. In addition Blom (8) provided serial feedings at 1-hr intervals and reported a glycogen synthesis rate of 9.3 gm CHO•kg$^{-1}$ wet wt•hr$^{-1}$. These rates are higher and occurred over longer durations than those previously reported for similar carbohydrate concentration feedings (10, 40, 41, 68) (Table 1). The question of

whether serial feedings maintain higher postexercise blood glucose and insulin concentrations and enhanced glycogen synthesis compared to bolus feedings is unclear.

***CHO Feedings: The Type of Carbohydrate.*** The digestible carbohydrate content of liquid and/or solid glucose feedings can consist of either glucose, fructose, galactose, lactose, sucrose, starch, and/or a synthetic multidextran polymer. Furthermore, the glycemic index of a meal containing these CHO structures can vary due to the delaying influence of fats and proteins on gastric emptying, and by increased gastric motility from dietary fiber (38). Therefore, obvious questions are whether the chemical type of carbohydrate ingested and/or the glycemic index of meals affect postexercise glycogen synthesis.

Data from Table 1 indicate that postexercise glycogen synthesis during the initial hours of recovery can occur at rates between 7 and 10 $mmol \bullet kg^{-1}$ wet $wt \bullet hr^{-1}$ following the ingestion of either a mixed diet high in CHO, a high glycemic index diet, or solutions of sucrose, glucose, and/or glucose polymers. However, after fructose ingestion, the rate of glycogen synthesis is depressed to a value approximating 4 $mmol \bullet kg^{-1}$ wet $wt \bullet hr^{-1}$ (10). This difference is due to the low glycemic index of fructose and the preferential uptake and metabolism of fructose by the liver relative to skeletal muscle (58).

Kiens et al. (44) investigated postexercise muscle glycogen synthesis following the ingestion of high carbohydrate diets that differed in glycemic index (GI) (Table 1). Subjects exercised until muscle glycogen stores were exhausted, and were then fed either a high (HGI) or low glycemic index (LGI) isocaloric diet, each of which provided 70% of Kcals from CHO. Results indicated that the high GI diet was accompanied by a 98% higher blood insulin concentration despite similar blood glucose concentrations. After 6 hrs of recovery, muscle glycogen synthesis during the HGI trial had occurred at almost double the rate of the LGI trial. However, after 20, 32, and 44 hrs postexercise, muscle glycogen concentrations were similar between trials. The authors concluded that glycogen synthesis occurred at a faster rate during the initial hours after exercise when a high GI diet was ingested, but that longer term glycogen storage was independent of the type of ingested CHO.

These data imply that mixed diets high in carbohydrate may be as adequate as liquid glucose feedings in maximizing postexercise glycogen synthesis. Nevertheless, studies that directly address this question have not been performed.

***Trained Versus Untrained Subjects.*** Muscle glycogen stores are larger in trained subjects, whether the training is from endurance, strength, or power related exercises (9, 49, 78). These findings provide indirect evidence of a greater capacity for glycogen synthesis in trained muscle. Additional results from both animal and human research also indicate an increased insulin sensitivity of endurance trained skeletal muscle during the initial hours after exercise (57). Nevertheless, studies that have directly compared postexercise glycogen synthesis between trained and untrained human muscle have been inadequate.

Piehl et al. (66) reported differences between resting muscle glycogen stores, exercise induced glycogen degradation, and postexercise glycogen synthesis between contralateral trained and untrained legs of 4 subjects (Table 1). Resting glycogen concentrations in the trained and untrained legs were 119 $\pm$ 11 and 81 $\pm$ 9 $mmol \bullet kg^{-1}$ wet wt, respectively. Submaximal and intermittent maximal exercise to exhaustion decreased muscle glycogen to low concentra-

tions, and muscle glycogen increased at a similar rate of 2.4 mmol•kg$^{-1}$ wet wt•hr$^{-1}$ for each leg during the first 10 hrs of recovery. The similar rate of glycogen synthesis after exercise is misleading, due to the lower concentration of postexercise muscle glycogen in the untrained leg (see Zachwieja et al., Table 1). Furthermore, inference from the data is limited due to the small number of subjects, and the resting glycogen concentration of the trained leg was not high relative to known values for well trained endurance athletes (5, 31).

***The Type of Recovery.*** Athletes are not always able to remain inactive during the postexercise recovery period. It is therefore beneficial to know whether even mild exercise/activity during the recovery period will decrease glycogen synthesis. Unfortunately, evaluation of this topic has been limited and the research has focused on the recovery from intense intermittent short-term exercise. Nevertheless, a study by Bonen et al. (12) included a comparison between postexercise glycogen synthesis with or without an active recovery following 30 min of cycling at 75% $VO_2$max. With an active recovery, muscle glycogen was further degraded, yet during the first 2 hrs of a passive recovery the glycogen synthesis occurred at a rate of 5 mmol•kg$^{-1}$ wet wt•hr$^{-1}$. Carbohydrate feedings provided glucose at a rate of 0.75 gm•kg$^{-1}$ body wt•hr$^{-1}$ in each trial (Table 1). These data should be interpreted with caution, as the exercise only decreased muscle glycogen by an estimated 24 mmol•kg$^{-1}$ wet wt. Greater glycogen degradation would be accompanied by increased synthetase activity, and it is unclear whether this would be sufficient to increase muscle glycogen stores during mild postexercise activity.

## Conclusions

The rate of postexercise glycogen synthesis will be predominantly determined by the magnitude of prior glycogen degradation. However, when assuming equal glycogen degradation, greater short-term postexercise glycogen synthesis occurs when approximately 0.7 gm CHO•kg$^{-1}$ body weight is ingested as early into the recovery period as possible. However, this optimal amount of carbohydrate ingestion has only been verified for lower body exercise. Whether additional amounts of carbohydrate need to be ingested following exhaustive total body exercise would require further investigation. The CHO source should be glucose, sucrose, and/or a multidextran mix, and can be liquid or solid.

Additional research is needed concerning the benefit of liquid versus solid carbohydrate feedings on glycogen synthesis during short-term recovery periods. There is indirect evidence that sequential feedings may increase the rate of glycogen synthesis compared to a bolus ingestion, and research has not clarified whether trained subjects have a greater capacity to synthesize glycogen during the recovery period. Glycogen synthesis is not impaired during the initial 4 hrs of recovery following eccentric exercise. Finally, an active recovery ($<$40% $VO_2$max) following prolonged steady-state exercise may further decrease muscle glycogen concentrations.

## Submaximal Exercise and Long-Term Recovery (>12 hrs)

The evaluation of postexercise glycogen synthesis during the first 12 hrs of recovery revealed the importance of the type, timing, and concentration of ingested

carbohydrate. However, questions of additional importance are, (a) What is the time required for muscle glycogen concentrations to return to resting levels? (b) Do carbohydrate feedings immediately after exercise result in greater stores of muscle glycogen beyond 24 hrs of recovery compared to normal dietary carbohydrate intake regimens (i.e., 3 meals/day)? (c) What is the relationship between dietary carbohydrate content and prolonged glycogen synthesis? (d) Does the eccentric component of certain exercises influence muscle glycogen stores during long-term (>12 hrs) recovery periods? The studies of longer term postexercise glycogen synthesis after low to moderate intensity exercise to exhaustion are listed in Table 3. As will be discussed, only some of these questions have been answered.

*The Type of Exercise.* In 1971 Costill et al. (18) reported changes in muscle glycogen (gastrocnemius) during 3 successive days of running 16.1 km in excess of normal training volume (8 km•day$^{-1}$) (Table 2, Figure 3). Preexercise muscle glycogen concentrations decreased from 118.8 (Day 1) to 88.3 (Day 2) and 61.1 (Day 3) mmol•kg$^{-1}$ wet wt. The increases in muscle glycogen from Days 1 to 2 and Days 2 to 3 were approximately 11 and −4 mmol•kg$^{-1}$ wet wt, respectively. The authors concluded that more than 24 hrs were required to replete muscle glycogen to preexercise concentrations when exercise of this duration and frequency was performed and a moderate carbohydrate diet was consumed.

Interestingly, research from the same laboratory performed almost two decades later further refined this topic and again indicated an inability to restore muscle glycogen to preexercise resting concentrations after 5 days of increased run training when consuming either a high or a low CHO diet (~8 and 4 gm CHO•kg$^{-1}$ body wt) (Table 2) (45). Simonsen et al. (76) performed similar

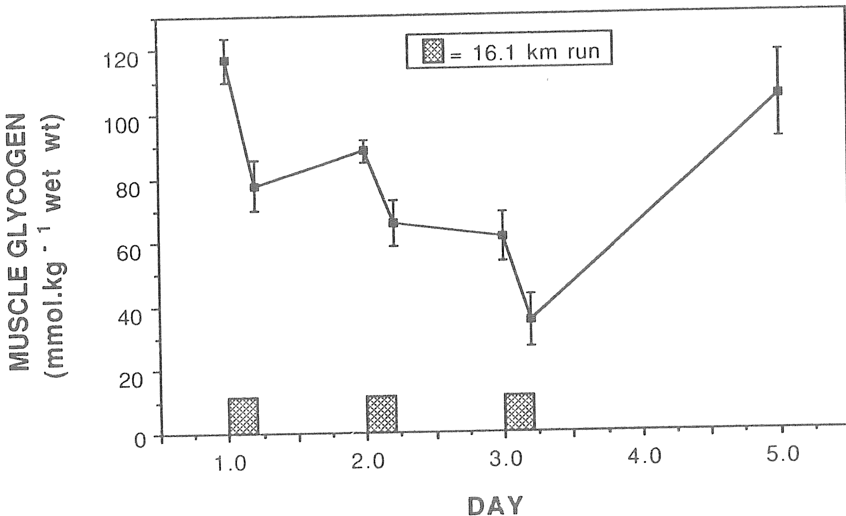


**Figure 3 — Results from the study by Costill et al. (18), indicating the incomplete recovery in muscle glycogen concentrations during 3 successive days of running 16.1 km.**

## Table 3
**Review of Research Evaluating Muscle Glycogen Synthesis Following High Intensity Exercise in Human Subjects**

| Study | Exercise | Δ G | [G] | CHO/subjects | Timing | Period | Rate | (*n*) |
|---|---|---|---|---|---|---|---|---|
| MacDougall (50) | int. cycling -140% $VO_2$max | 57.0 | 23.0 | 0 | | 0-2 | 4.5 | 6 |
| Hermansen (33) | int. max. cycling | 38.1 | 49.6 | 0 | | 0-0.5 | 33.6 | 9 |
| Bonen (12) | 60 min submax & sprint cycle | | | | | | | |
| | a) passive recovery | 70.6 | 21.1 | lg 0.75 | 0,2 | 0-2 | 15.3 | 7 |
| | | | 51.7 | " | " | 2-4 | 1.9 | 7 |
| | b) active recovery | 76.9 | 18.9 | lg 0.75 | 0,2 | 0-2 | 4.7 | 5 |
| | | | 28.3 | " | " | 2-4 | 3.3 | 5 |
| Hultman (37) | 5 min max. cycling | 41.0 | | 0 | | 0-1 | 16.2 | 7 |
| Keizer (43) | intermittent bouts | 60.7 | 17.3 | lg 71.4% CHO | 0.5,1.5,2.5,3.5 | 0-5 | 6.1 | 8 |
| | | | | | 6,9,19 | 0-22 | 1.5 | 8 |
| | | 52.7 | 17.6 | sg 77.5% CHO | 0.5,1.5,2.5,3.5 | 0-5 | 6.6 | 8 |
| | | | | | 6,9,19 | 0-22 | 1.5 | 8 |
| | | 59.3 | 17.6 | | ad.lib. | 0-5 | 4.0 | 4 |
| Peters Futre (64) | 5 x 90 s cycling at 120% $VO_2$max intensity | 106.0 | | | | | | |
| | a) passive recovery -both legs | | 25.6 | 0 | | 0-0.75 | 18.0 | 8 |
| | | | | | | 0.75-1.5 | 15.3 | 8 |
| | b) one-legged cycling at 30% $VO_2$max | | 42.5 | 0 | | 0-0.75 | 16.0 | 8 |
| | | | | | | 0.75-1.5 | 16.0 | 8 |
| | c) nonexercised recovery leg | | 42.5 | 0 | | 0-1.5 | 25.2 | 8 |
| Robergs (70) | leg extensions | | | | | | | |
| | 70% 1 RM intensity | 47.0 | 73.4 | 0 | | 0-2 | 10.1 | 8 |
| | 35% 1 RM intensity | 46.6 | 75.9 | 0 | | 0-2 | 7.1 | 8 |

Glycogen degradation (DG, mmol•$kg^{-1}$ wet wt); postexercise glycogen concentration ([G], mmol•$kg^{-1}$ wet wt); postexercise carbohydrate ingestion (CHO, gm•$kg^{-1}$•$hr^{-1}$); postexercise glycogen synthesis rate (Rate, mmol•$kg^{-1}$•$hr^{-1}$ wet wt); time of CHO ingestion (time, hrs); period of glycogen synthesis rate calculation (period, hrs); type of carbohydrate = glucose (g) and liquid (l), or solid (s). Data reported as mmol•$kg^{-1}$ dry wt in the literature were divided by 4.1 to account for water weight. Research is listed in chronological order.

research on athletes during 4 weeks of twice daily rowing training when fed either 5 or 10 gm CHO•$kg^{-1}$ body wt. Muscle glycogen stores (vastus lateralis) continued to increase during the 4 weeks of training on both diets, yet concentrations were significantly larger at the end of the training period on the higher CHO diet (155 $\pm$ 7 and 124 $\pm$ 10 mmol•$kg^{-1}$ wet wt, respectively).

It is interesting to compare the results of Simonsen to those of Costill et al. (18) and Kirwan et al. (45). An attractive hypothesis that was not addressed by either of Costill or Kirwan was that the increased eccentric exercise performed during consecutive days of running distances in excess of normal training volume may induce muscle damage and impair the process of postexercise glycogen synthesis compared to exercise without an eccentric component (e.g., rowing).

Pascoe et al. (63) employed a similar design to the Costill et al. (18) study by comparing 3 successive days of exercise (60 min at 75% $VO_2$max) between running and cycling. It was hypothesized that if the eccentric component of running caused muscle trauma, postexercise glycogen synthesis should be lower following running compared to cycling. Results were inconclusive, as cycling caused greater muscle glycogen degradation than did running, and running and cycling caused similar delays in glycogen storage during successive days of exercise. Results may have differed if running had involved a downhill component and if a larger subject sample had been used.

Recent research has associated excessive running (e.g., marathon) with reduced rates of postexercise glycogen synthesis (74), and muscle trauma characterized by necrosis and macrophage and leukocyte infiltration (33) (Table 2). O'Reilly et al. (62) evaluated glycogen synthesis (vastus lateralis) following 45 min of eccentric cycling exercise. The exercise bout decreased muscle glycogen from approximately 84 to 58 mmol•$kg^{-1}$ wet wt. Ten days after the exercise, muscle glycogen had further decreased to 37 mmol•$kg^{-1}$ wet wt. The loss of muscle glycogen was shown to be greatest in fast twitch muscle fibers. The authors presented microscopic evidence of muscle damage and hypothesized that the damage restricted glucose uptake into skeletal muscle, thus preventing glycogen synthesis. Due to the large time delay between the immediate postexercise and the 10-day biopsy sample, limited information on the relationships among postexercise muscle soreness (maximal between 24 and 48 hrs), muscle damage, and glycogen synthesis was provided.

Costill et al. (19) improved the design of the O'Reilly study by investigating muscle glycogen synthesis following single leg eccentric contractions of the leg extensor muscles, and 1 hr of two-legged cycling at 74% $VO_2$max (Table 2). During the recovery period, subjects consumed a diet of either 4.25 or 8.5 gm•$kg^{-1}$ body wt, and muscle glycogen content in the vastus lateralis was evaluated at 0, 24, and 72 hrs postexercise. Significantly less glycogen was stored in the eccentrically exercised leg (EL) compared to the cycling exercised leg (CL), yet subjects who consumed the high carbohydrate diet stored significantly more glycogen in the EL than in the CL. Histological preparation of muscle sections revealed leukocyte, lymphocyte, and macrophage infiltration into and between muscle fibers, and these cells are known to increase glucose utilization and lactate production (29, 34, 73). Consequently, it has been proposed that postexercise glycogen synthesis is impaired following eccentric exercise due to increased competition between the inflammatory and muscle cells for blood glucose.

Doyle and Sherman (25) adopted a research protocol similar to that of Costill et al. (19) but compared the initial hours of recovery following concentric or eccentric leg extension exercise to the same recovery from concentric exercise performed 48 hrs later (Table 1). The rate of postexercise glycogen synthesis was not different between concentric and eccentric exercise legs during the first 4 hrs of recovery, but the rate of glycogen synthesis was significantly lower in the eccentric leg 48 hrs later.

Research using animal models has clearly shown that eccentric exercise or forced muscle lengthening results in muscle damage and impaired glycogen synthesis (2). However, due to the extreme efforts used to invoke damage in these models, the results may have limited application to human athletes. Additional research is needed to evaluate the relationships between impaired glycogen synthesis, muscle trauma, and the development of muscle soreness following running for both trained and untrained individuals (16). Furthermore, as evidence of leukocyte infiltration and muscle soreness are not evident until between 12 to 24 hrs of recovery, it would be beneficial to investigate the effects of eccentric exercise on postexercise glycogen synthesis at repeated intervals between 0 and 24 hrs of recovery.

***The Amount of Carbohydrate.*** The observation of the need for carbohydrate for long-term glycogen synthesis was first reported by Ahlborg et al. (1) and Bergstrom and Hultman (5) (Tables 1, 2). More elaborate investigations of this topic were performed by Costill et al. (21) and Sherman et al. (75) (Table 2). Both studies reported that greatest postexercise glycogen synthesis occurred following diets high in CHO (75% Kcals), providing up to 650 gm CHO•day$^{-1}$, and that even modest decreases in dietary CHO content (to 50% Kcals) significantly reduced long-term muscle glycogen synthesis. These data are supported by more recent research of muscle glycogen concentrations during repeated days of rowing (76) and running training (45) when dietary carbohydrate has been controlled.

These studies indicated that muscle glycogen concentrations can return to preexercise values during the immediate 24 hrs after exercise with the consumption of approximately 10 gm CHO•kg body wt$^{-1}$•day$^{-1}$. Nevertheless, repeated days of running exercise may require larger amounts of carbohydrate consumption and/or longer recovery periods compared to rowing, as indicated by the eccentric exercise research. Unfortunately, no study has evaluated the influence of liquid glucose feedings immediately postexercise on long-term glycogen synthesis.

***Glycogen Supercompensation.*** In 1967 Bergstrom and Hultman (5) reported that muscle glycogen stores could be increased above normal concentrations if a specific diet/exercise regimen was followed during 7 days prior to competition. These findings were supported by additional research indicating that the most successful means for elevating skeletal muscle glycogen stores was to first perform exhaustive exercise to deplete muscle glycogen stores, followed by 3 days on a diet low in carbohydrate and then a diet high in carbohydrate for an additional 3 days with rest (3, 36, 72). However, Sherman et al. (75) questioned the practicality of a low carbohydrate diet and exhaustive exercise during the week prior to competition and also pointed out that the earlier research was predominantly based on cycling exercise with relatively untrained subjects. The Sherman study revealed that a high carbohydrate diet during the 3 days prior to

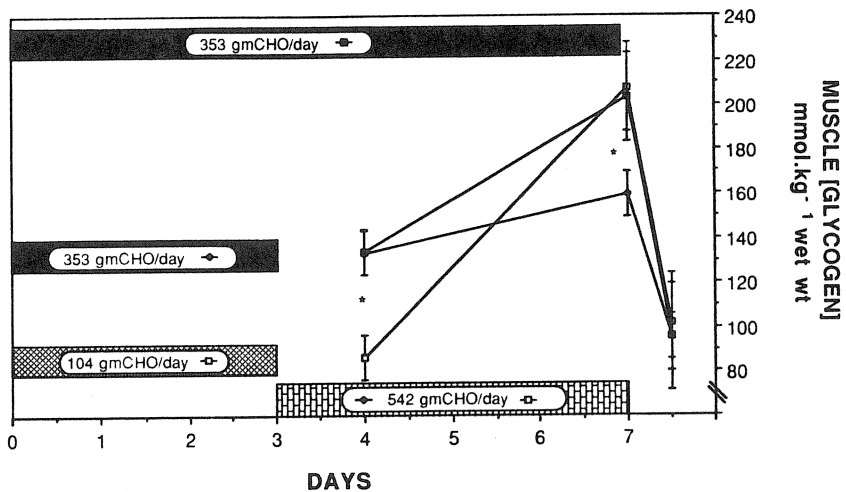


**Figure 4 — Results of Sherman et al. (75) comparing muscle glycogen supercompensation following differing regimens of diet and exercise. Interestingly, glycogen synthesis was greater when muscle glycogen was lowest at Day 4.**

competition was as adequate at increasing muscle glycogen in trained runners as the original diet/exercise regimen (Figure 4). In each of these studies, muscle glycogen increased to approximately 220 mmol•kg$^{-1}$ wet wt (Table 2).

Knowledge of the effects of exhaustive running during the preliminary glycogen depletion phase of the glycogen supercompensation sequence was provided by Blom et al. (9) (Table 2). When running at 75% $VO_2$max to exhaustion, postexercise glycogen synthesis when fed carbohydrate occurred at approximately 3 mmol•kg$^{-1}$ wet wt•hr$^{-1}$ during the first 22 hrs of recovery. However, negligible muscle glycogen synthesis occurred beyond 22 hrs for each of the trained and untrained subjects. Results were similar for trained and untrained subjects; however, this comparison was biased by differing amounts of exercise-induced glycogen degradation (Table 2). The authors concluded that ultrastructural muscle damage may have occurred during the exhaustive running, possibly due to the eccentric component of running. Consequently, the use of running exercise to exhaust muscle glycogen stores during the preliminary phase of glycogen supercompensation may be inappropriate.

## *Conclusions*

After 24 hours of recovery, muscle glycogen can be increased to preexercise concentrations when a diet high in carbohydrate (>70% Kcals) providing approximately 10 gm CHO•kg body wt$^{-1}$•day$^{-1}$ is ingested. Supercompensating levels of muscle glycogen storage can be attained following 3 days of rest with a high carbohydrate diet (>70% Kcals). However, the synthesis of muscle glycogen during the recovery may be compromised if unaccustomed exercise involving an eccentric component is performed. Eccentrically induced muscle damage lowers glycogen synthesis when measured beyond 24 hrs after exercise; however, the

time when the rate of glycogen synthesis becomes impaired following 4 hrs postexercise is not known. Evidence indicates that high carbohydrate diets during the immediate hours following eccentric exercise may enhance muscle glycogen storage. Research has been inadequate for comparing glycogen synthesis between trained and untrained individuals. Nevertheless, considerable evidence indicates that resting muscle glycogen concentrations are larger in both endurance and strength trained subjects compared to untrained individuals.

### *Intense Exercise*

The studies of postexercise glycogen synthesis after intense short-term exercise to exhaustion are listed in Table 3. It is apparent that postexercise glycogen synthesis rates following intense exercise are higher than for exhaustive exercise at lower intensities and can occur without carbohydrate ingestion during the recovery.

***Characteristics of Carbohydrate Feedings.*** Limited research has evaluated the influence of carbohydrate feedings on muscle glycogen synthesis during the recovery from intense exercise. As previously mentioned, Bonen et al. (12) exercised subjects with prolonged cycling exercise, finishing with 10 to 15 1- to 2-min intervals of sprint cycling (Table 3). Subjects consumed liquid glucose solutions at 0 and 2 hrs of recovery, resulting in high rates of glycogen synthesis (0–2 hrs). However, these rates were similar to those of Peters Futre (64) and Hultman (37) when carbohydrate feedings were not provided.

The influence of the type of carbohydrate feeding on muscle glycogen synthesis during the recovery following intense exercise was investigated by Keizer et al. (43) (Table 3). No differences existed between the rate of glycogen synthesis when fed either solid or liquid meals containing approximately 70% Kcals from carbohydrate during the first 5 hrs of recovery. However, the rates of glycogen synthesis were low compared to other studies, and muscle lactate concentrations were not measured. Of added interest was that subjects who consumed food ad libitum had a significantly lower rate of glycogen synthesis compared to the liquid and solid carbohydrate trials. Furthermore, despite almost complete restorage of muscle glycogen after 22 hrs of recovery in the liquid and solid carbohydrate trials, exercise performance was significantly lower after the recovery when compared to an identically administered performance ride prior to the initial glycogen depletion exercise. The authors concluded that liquid or solid carbohydrate diets after intense exercise resulted in similar amounts of glycogen storage, and that the fatigue accompanying incremental exercise performed over successive days is not totally dependent on muscle glycogen stores.

***The Type of Recovery.*** The influence of an active postexercise recovery after intense exercise on postexercise glycogen synthesis was evaluated using a one-leg repeated-measures design by Peters Futre et al. (64). Subjects performed intermittent high intensity exercise which depleted muscle glycogen by 70%. Subjects then completed one-legged cycling at 30% $VO_2max$ for 45 min, followed by 45 min of additional passive recovery. The contralateral leg remained inactive for the 90 min. No significant difference was found in postexercise glycogen synthesis between active and passively recovered legs at either 45 or 90 min (Table 3), despite significantly larger muscle lactate concentrations in the active compared to passively recovered leg at 45 min of recovery. The rates of glycogen synthesis were comparable to those from studies involving a passive

recovery following intense exercise. The authors concluded that a low intensity active recovery after high intensity exercise may not decrease postexercise glycogen synthesis, and that the data did not support the explanation that lactate removal was predominantly accounted for by intramuscular conversion to glycogen.

Recently Nordheim and Vollestad (60) investigated the effects of an active recovery on muscle glycogen concentrations in type I and II fibers following intense intermittent cycling exercise that increased muscle lactate concentrations in excess of 26 mmol•kg$^{-1}$ wet wt. The results indicated that during the recovery from intense exercise, low intensity exercise (40% $VO_2$max) during the recovery was not accompanied by increased glycogen in type I muscle fibers, yet an increase in type II fiber glycogen content occurred. Fiber type glycogen content was estimated from ATPase and PAS stained serial muscle sections. The authors concluded that lactate was used in preference to glycogen for ATP regeneration in type I muscle fibers during low intensity exercise, and that lactate could have been a substrate for glycogen synthesis in type II fibers under the experimental conditions.

The data from Peters Futre et al. (64), Nordheim and Vollestad (60), and Vollestad et al. (79) combine to indicate that whole muscle glycogen concentrations should not be interpreted to indicate equal glycogen synthesis in all muscle fibers. The combination of an active recovery and the lower rate of postexercise glycogen synthesis in type I fibers may be very detrimental to subsequent exercise performance.

***Muscle Glyconeogenesis.*** Robergs et al. (70) reported that following glycogen degradation from leg extension/weight resistance exercise, muscle glycogen content increased by approximately 22 mmol•kg$^{-1}$ wet wt without carbohydrate intake during 2 hrs of passive recovery. These results are similar to those reported by MacDougall et al. (50) for intermittent high intensity cycle ergometry and they complement the remaining studies of short-term glycogen synthesis following intense exercise (33, 37, 64, 78). The relatively high postexercise glycogen synthesis rates after intense exercise have been explained by the potential for muscle lactate being an endogenous glycogenic precursor (33, 56) and/or by the accumulation of the hexose and triose phosphate glycolytic intermediates which can be readily incorporated into glycogen (32).

The explanation of muscle glycogen synthesis from lactate is controversial. If lactate is a physiological substrate for glycogen synthesis, either the pyruvate kinase reaction is reversible under physiological conditions or there must be alternate enzymatic pathways. Dyson et al. (26) have presented evidence of pyruvate kinase reversibility with product removal; however, more evidence is required to support the thermodynamics of this reaction under in vivo cellular conditions.

Lactate could also be incorporated into the glycolytic pathway by conversion to pyruvate and then to cytosolic oxaloacetate (Figure 5). The enzyme known to spontaneously catalyze the latter reaction is pyruvate carboxylase (PyC; E.C. 6.4.1.1). PyC activity is known to exist in lower vertebrate fast-twitch skeletal muscle, but its activity in human muscle is extremely low (61).

Consequently, the conversion of lactate to pyruvate and then the flux of pyruvate into the citric acid cycle for the eventual conversion to malate is a more realistic route. Malate could then be shuttled out of the mitochondria and converted to cytosolic oxaloacetate by malate dehydrogenase (malic enzyme

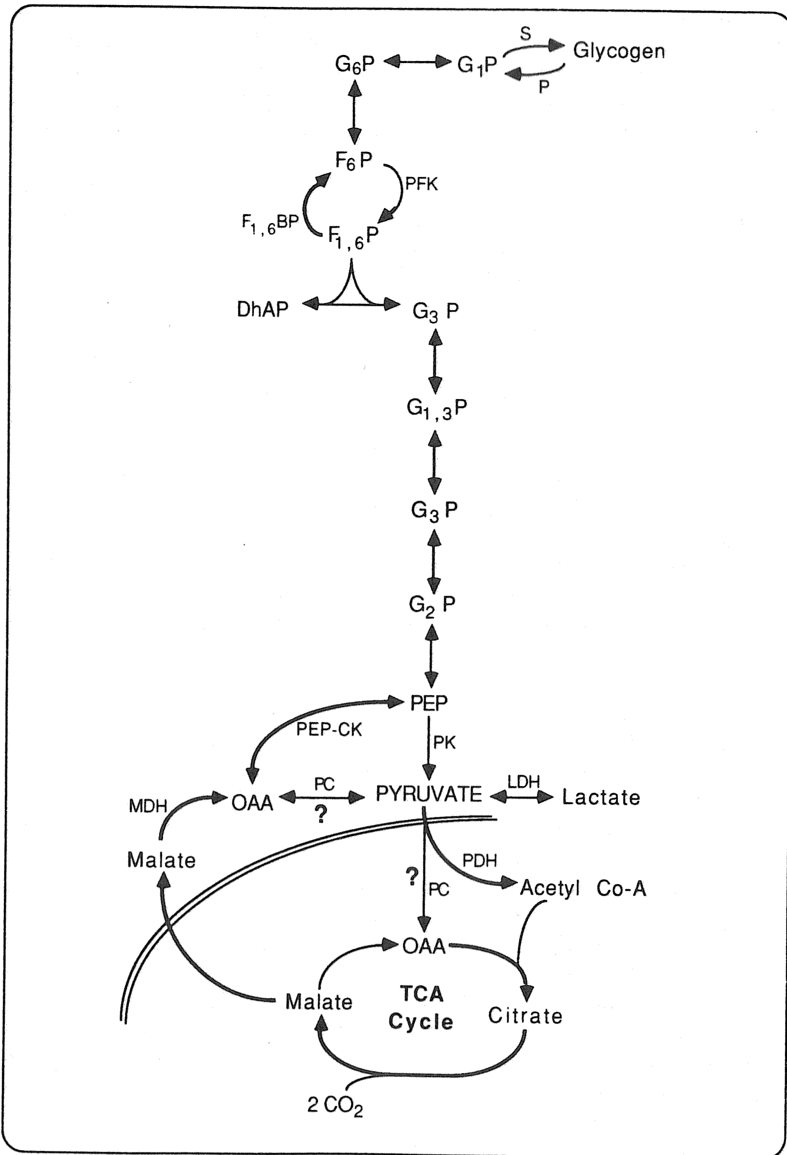


**Figure 5 — The glycolytic and possible lactate-glyconeogenic pathways in skeletal muscle. Additional substrates, products, and coenzymes for these reactions have not been included in the figure. (See text for explanation of abbreviations.)**

complex: L-malate-NADP oxidoreductase; E.C. 1.1.1.40 and L-malate-NAD oxidoreductase; E.C. 1.1.1.37). Oxaloacetate could then be converted to phosphoenolpyruvate (PEP) by phosphoenolpyruvate carboxykinase (PEPCK; E.C. 4.1.1.32). PEP could then be sequentially converted to fructose-1,6-bisphosphate

where fructose bisphosphatase (FbPase; E.C. 3.1.3.11) catalyzes the dephosphorylation of fructose-1,6-bisphosphate to fructose-6-phosphate. Interestingly, the malate dehydrogenase pathway also involves lactate oxidation with the liberation of carbon dioxide molecules from the TCA cycle. Lactate oxidation and glyconeogenesis should not be considered independent pathways that account for the fate of lactate after exercise, as many researchers assume.

Several in vitro studies using specimens of frog, rabbit, and pigeon muscles have revealed that lactate uptake and glycogen synthesis by skeletal muscle increases with increasing concentrations of lactate in the perfusate or incubation medium (4, 53, 60). Furthermore, glycogen synthesis was greater in FT than in ST muscle (56, 76). Examination of muscle enzyme activities has also established that FbPase activity is higher in FT than in ST muscle and that this activity is accompanied with higher PEPCK activity (23, 61, 76).

The results of Hermansen and Vaage (33) have often been interpreted as in vivo evidence for muscle lactate incorporation into glycogen. The rate of glycogen synthesis in this study was calculated from a 30-min biopsy sample; however, it remains high compared to the study by Hultman, which essentially improved the design used by Hermansen and Vaage. The glycogen synthesis rate of Hultman (37) was more realistic (Table 3), and several faults of the Hermansen and Vaage investigation have been documented (14, 64). Essentially, in the Hermansen and Vaage study, blood flow and muscle metabolism data were obtained from differing muscles and subjects, and the findings of limited lactate removal and glucose uptake by the vastus lateralis were estimations and thus were subject to considerable error.

Evidence against the incorporation of lactate into muscle glycogen is based on the argument that the preceding muscle enzyme research was not completed under human in vivo physiological conditions. Despite the indirect in vivo findings from Hermansen and Vaage (33) and Hultman (37), in vivo human conditions could provide alternative fates for accumulated muscle lactate, these being circulatory removal and oxidation.

Brooks and Gaesser (14) reported that following prolonged exercise and a sprint, there was little glycogen synthesis in rat muscle and that the fate of lactate was circulatory removal and oxidation. This conclusion was based on the post-exercise distribution of $^{14}C$ from $^{14}C$-labeled lactate, which showed that $^{14}C$ atoms were incorporated into amino acid pools, protein, and glycogen as well as bicarbonate and $CO_2$. The circulatory removal of lactate from muscle is expected due to the gradient that would exist between muscle and blood [$La^-$] after high intensity exercise (32). In addition, the translocation of lactate molecules from glycolytic and oxidative fibers is known to occur (13, 27, 30) and may be at least partially responsible for the high lactate oxidation (approximately 50% of lactate production) reported to occur in humans during and after exercise (13, 14, 15). Nevertheless, as previously explained, findings of high lactate oxidation do not automatically preclude lactate carbons being used as a substrate for muscle glycogen synthesis. In addition, evidence also indicates that blood lactate is a preferred substrate for liver gluconeogenesis and glyconeogenesis (42).

When all these studies are considered, it is apparent that skeletal muscle does have the potential to metabolize lactate into glycogen. However, it is uncertain whether this process is physiologically significant. There is limited direct evidence from in vivo human research indicating that muscle lactate is incorporated into muscle glycogen at physiologically significant rates after intense exer-

cise. One must conclude that the large glycogen synthesis rates observed after intense exercise may be due to the reversed flux of hexose-phosphate and/or triose-phosphate glycolytic intermediates into glycogen. Additional research designed to clarify the origin of glucose carbons used for postexercise glycogen synthesis in human skeletal muscle after intense exercise is warranted.

### Conclusions

Rates of postexercise muscle glycogen synthesis following intense exercise are more than twice as great as those following prolonged submaximal intensity exercise when fed carbohydrate. Furthermore, muscle glycogen synthesis during the initial hours after high intensity exercise occurs without the need for carbohydrate ingestion and is not increased if carbohydrate is ingested during the recovery. Controversy exists concerning the mechanisms that may account for glycogen synthesis after high intensity exercise. Muscle glyconeogenesis from lactate has been repeatedly proven with in vitro animal models; however, there is no direct human in vivo evidence to prove that an appreciable amount of muscle lactate is converted to muscle glycogen during recovery. An active low intensity recovery following intense exercise did not prevent postexercise muscle glycogen synthesis from occurring at rates similar to those reported in other passive recovery studies. Interestingly, estimations of muscle fiber type specific glycogen synthesis indicate that greater glycogen storage occurs in fast-twitch fibers after intense exercise during both an active and passive recovery.

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