Nutritional Strategies to Promote Postexercise Recovery

Milou Beelen, Louise M. Burke, Martin J. Gibala, and Luc J.C. van Loon

During postexercise recovery, optimal nutritional intake is important to replenish endogenous substrate stores and to facilitate muscle-damage repair and reconditioning. After exhaustive endurance-type exercise, muscle glycogen repletion forms the most important factor determining the time needed to recover. Postexercise carbohydrate (CHO) ingestion has been well established as the most important determinant of muscle glycogen synthesis. Coingestion of protein and/or amino acids does not seem to further increase muscle glycogen-synthesis rates when CHO intake exceeds 1.2 g · kg⁻¹ · hr⁻¹. However, from a practical point of view it is not always feasible to ingest such large amounts of CHO. The combined ingestion of a small amount of protein (0.2–0.4 g · kg⁻¹ · hr⁻¹) with less CHO (0.8 g · kg⁻¹ · hr⁻¹) stimulates endogenous insulin release and results in similar muscle glycogen-repletion rates as the ingestion of 1.2 g · kg⁻¹ · hr⁻¹ CHO. Furthermore, postexercise protein and/or amino acid administration is warranted to stimulate muscle protein synthesis, inhibit protein breakdown, and allow net muscle protein accretion. The consumption of ~20 g intact protein, or an equivalent of ~9 g essential amino acids, has been reported to maximize muscle protein-synthesis rates during the first hours of postexercise recovery. Ingestion of such small amounts of dietary protein 5 or 6 times daily might support maximal muscle protein-synthesis rates throughout the day. Consuming CHO and protein during the early phases of recovery has been shown to positively affect subsequent exercise performance and could be of specific benefit for athletes involved in multiple training or competition sessions on the same or consecutive days.

Keywords: glycogen, carbohydrate, protein, amino acids, caffeine, performance

Carbohydrate (CHO) and fatty acids are the main fuels oxidized by skeletal-muscle tissue during exercise, and the relative contribution of these fuel sources varies with exercise intensity and duration (Romijn et al., 1993; van Loon, Greenhaff, Constantin-Teodosiu, Saris, & Wagenmakers, 2001) and training status (van Loon, Jeukendrup, Saris, & Wagenmakers, 1999). The contribution of CHO oxidation to total energy expenditure rises with increasing exercise intensity (Figure 1), and without the intake of exogenous CHO, exercise performance is mainly determined by the availability of endogenous CHO (van Loon et al., 1999). Endogenous CHO is stored as muscle and liver glycogen and quantitatively represents less than 5% of total energy storage (McArdle, Katch, & Katch, 2001). However, muscle glycogen represents an important fuel source during prolonged moderate- to high-intensity exercise (Bergstrom & Hultman, 1967; Bosch, Welten, Dennis, & Noakes, 1996; Tsintzas & Williams, 1998), contributing more than 50% of total energy requirements (Romijn et al., 1993; van Loon et al., 2001). The latter might be even greater during high-intensity interval (Tsintzas & Williams, 1998) or resistance-type exercise (Koopman et al., 2005; Robergs et al., 1991; Roy & Tarnopolsky, 1998; Tesch, Coliander, & Kaiser, 1986).

After cessation of exercise, muscle glycogen is typically restored to preexercise concentrations within 24 hr, provided that sufficient amounts of CHO are ingested (Burke et al., 1995; Costill et al., 1981). However, for athletes involved in multiple training sessions or competitions on the same day or successive days, muscle glycogen stores need to be replenished more rapidly (Burke et al., 1995; Burke, Kiens, & Ivy, 2004; Burke, Loucks, & Broad, 2006). Therefore, much research has focused on nutritional interventions to optimize early postexercise muscle glycogen repletion (Burke et al., 1995; Burke, Collier, & Hargreaves, 1993; Costill et al., 1981; Ivy, Katz, Cutler, Sherman, & Coyle, 1988; Ivy, Lee, Brozinick, & Reed, 1988; Jentjens & Jeukendrup, 2003; Pedersen et al., 2008; Reed, Brozinick, Lee, & Ivy, 1989; van Loon, Saris, Kruijshoop, & Wagenmakers, 2000; Zawadzki, Yaspelkis, & Ivy, 1992). Postexercise CHO ingestion represents the most important factor determining the rate of muscle glycogen synthesis (Burke et al., 1995; Burke et al., 1993; Burke et al., 2004; Burke et al., 2006; Costill et al., 1981; Ivy, Katz, et al., 1988; Jentjens & Jeukendrup, 2003; Reed et al., 1989; van Loon, Saris, Kruijshoop, & Wagenmakers, 2000). However, coinigestion of protein (Burke et al., 2004; van Loon Saris, Kruijshoop, & Wagenmakers, 2000; Zawadzki et al., 1992) or caffeine (Pedersen et al., 2008) may further accelerate postexercise muscle glycogen synthesis. Furthermore, postexercise protein or amino acid ingestion is warranted to stimulate muscle protein synthesis, which is likely instrumental to help repair muscle damage and allow skeletal-muscle reconditioning (Hawley, Tipton, & Millard-Stafford, 2006; Koopman, Saris, Wagenmakers, 2006).
Beelen et al. & van Loon, 2007; Rennie & Tipton, 2000; Tipton & Wolfe, 2004). It is now generally accepted that optimal sports-recovery nutrition should contain both CHO and protein. However, there is still considerable discussion on the exact amount, type, and timing of ingestion of these nutrients and the further benefits of specific pharmaco-nutrients (Hawley et al., 2006; Koopman, Saris, et al., 2007; Rennie & Tipton, 2000; Tipton & Wolfe, 2004).

In this review we first elaborate on the proposed impact of postexercise CHO, protein, and caffeine ingestion on muscle glycogen synthesis during early postexercise recovery. Second, we focus on the impact of protein and/or amino acid administration during postexercise recovery as a means to stimulate muscle protein synthesis. Thereafter, we discuss the potential effects of postexercise CHO and protein ingestion on subsequent exercise performance. Finally, we give an overview of current ideas, speculations, and plans for future research on the application of specific proteins, amino acids and/or CHO to stimulate postexercise recovery. We end this review with an overall conclusion and some practical guidelines for optimal postexercise recovery nutrition.

**Postexercise Muscle Glycogen Synthesis**

From a quantitative point of view, muscle glycogen represents the most important fuel source during prolonged moderate- to high-intensity exercise (Figure 1; Romijn et al., 1993; van Loon et al., 2001). Resting muscle glycogen stores range between 500 and 600 mmol/kg dry weight (dw; Bosch et al., 1996; Costill et al., 1981) but can decrease considerably during prolonged endurance-type exercise or high-intensity exercise of relatively short duration. For example, in laboratory situations, muscle glycogen stores have been reported to decline by 50–75% after 3 hr of cycling at 70% VO$_{2\text{max}}$ (Bosch, Dennis, & Noakes, 1994; Bosch et al., 1996) and by 30–40% during an ~45-min resistance-type exercise session (Koopman et al., 2005; Robergs et al., 1991; Roy & Tarnopolsky, 1998; Tesch et al., 1986). Muscle glycogen is likely to be significantly decreased by many of the key training sessions commonly undertaken by swimmers, rowers, runners, team-sport players, and other athletes. However, studies of glycogen-depletion patterns in real-life sporting activities are surprisingly rare. Nevertheless, because a direct relationship between fatigue and muscle glycogen depletion has been well described (Bergstrom, Herman- sen, Hultman, & Saltin, 1967; Bergstrom & Hultman, 1967), postexercise muscle glycogen-repletion rate represents one of the most important factors that determine the time needed to recover.

Postexercise muscle glycogen synthesis occurs in two different phases (Garetto, Richter, Goodman, & Ruderman, 1984; Maehlum, Hostmark, & Hermansen, 1977; Price et al., 1994; Richter, Garetto, Goodman, & Ruderman, 1984). Early postexercise muscle glycogen synthesis seems to be independent of circulating insulin levels and lasts 30–60 min (Jentjens & Jeukendrup, 2003; Maehlum et al., 1977; Price et al., 1994; Richter et al., 1984). Glycogen-synthesis rates during this phase are
high (30–45 mmol · kg dw–1 · hr–1) but rapidly decrease by 60–90% when no CHO is ingested (Maehlum et al., 1977; Price et al., 1994). Several studies indicate that this insulin-independent phase only occurs when postexercise muscle glycogen concentrations are reduced to less than 150–200 mmol/kg dw (Jentjens & Jeukendrup, 2003; Maehlum et al., 1977; Nielsen et al., 2001; Price et al., 1994). Price et al. showed that glycogen repletion becomes biphasic when glycogen is depleted to 25% of preexercise resting levels. Muscle glycogen restoration was characterized by rapid synthesis during the first 30–60 min of postexercise recovery, followed by significantly lower glycogen-synthesis rates. The final synthesis rates did not differ from rates observed when glycogen was depleted to 50% and 75% of preexercise, resting levels. In accordance, inhibition of insulin secretion by the infusion of somatostatin did not alter the initial rate of glycogen synthesis but prevented muscle glycogen repletion during the slower second recovery phase. Consequently, the second recovery phase was defined as the insulin-dependent phase of postexercise glycogen synthesis (Price et al., 1994; Young, Wallberg-Henriksson, Sleeper, & Holloszy, 1987). With sufficient, nonlimiting CHO ingestion, the rate of muscle glycogen synthesis in this phase ranges from 20 to 35 mmol · kg dw–1 · hr–1 (Ivy, Katz, et al., 1988; Ivy, Lee, et al., 1988; Keizer, Kuipers, van Kranenburg, & Geurten, 1987; Maehlum, Felig, & Wahren, 1978; Maehlum et al., 1977), which is ~10–30% lower than repletion rates observed during the initial, insulin-independent phase (Ivy, Katz, et al., 1988; Ivy, Lee, et al., 1988; Jentjens & Jeukendrup, 2003).

Both phases of muscle glycogen synthesis are mediated by an increase in glucose-transport rate (Ploug, Galbo, Vinten, Jorgensen, & Richter, 1987; Wallberg-Henriksson, Constable, Young, & Holloszy, 1988; Young et al., 1987) and glycogen synthase activity (Jentjens & Jeukendrup, 2003; Richter, Derave, & Wojtaszewski, 2001). The latter enzyme catalyzes the incorporation of the glycosyl residues from UDP-glucose into glycogen (Danforth, 1965; Jentjens & Jeukendrup, 2003) and is believed to form the rate-limiting step in the process of glycogen synthesis (Danforth, 1965). During the initial, rapid phase of glycogen synthesis, the high rate of glucose transport into the cell is mediated by the contraction-induced translocation of the glucose transporter-4 (GLUT-4) to the sarcolemma (Hayashi, Wojtaszewski, & Goodyear, 1997; Richter et al., 2001; Richter et al., 1984). Furthermore, a low glycogen level also stimulates glucose transport during this phase, because a large portion of GLUT-4-containing vesicles is believed to be bound to glycogen and might become available when glycogen levels are depleted (Derave et al., 1999; Jentjens & Jeukendrup, 2003). Besides its effect on glucose transport, a low muscle glycogen concentration also stimulates glycogen synthase activity (Danforth, 1965; Nielsen et al., 2001). Nielsen et al. demonstrated that glycogen content is a far more potent regulator of glycogen synthase activity than insulin and that the stimulatory effect of muscle contraction on glycogen synthase activity is solely a function of the contraction-induced decline in muscle glycogen content.

The second phase of muscle glycogen synthesis is characterized by an increase in the sensitivity of muscle glucose uptake and glycogen synthase to circulating insulin levels (Cartee et al., 1989; Danforth, 1965; Jentjens & Jeukendrup, 2003; Wallberg-Henriksson et al., 1988). This greater muscle insulin sensitivity after exercise can persist for up to 48 hr depending on the intake of CHO and the amount of muscle glycogen that is restored (Cartee et al., 1989; Jentjens & Jeukendrup, 2003). The increase in insulin-stimulated GLUT-4 translocation to the sarcolemma is mediated by the activation of the insulin-signaling pathways (Hayashi et al., 1997). A more detailed description of the mechanisms of muscle glycogen synthesis is beyond the scope of this review, but it is well described in a review by Jentjens and Jeukendrup. Because of the role of insulin in regulating muscle glycogen synthesis during the insulin-dependent phase, there is much interest in nutritional interventions that can stimulate postexercise insulin secretion (van Loon, Krujishoop, Verhagen, Saris, & Wagenmakers, 2000; van Loon, Saris, Verhagen, & Wagenmakers, 2000c). In the next few paragraphs we will elaborate on the effects of CHO, amino acids and/or protein, and caffeine ingestion on glycogen-synthesis rates during postexercise recovery.

### Carbohydrate Ingestion

Muscle glycogen-synthesis rates during postexercise recovery in the fasted state have been reported to be ~2 mmol · kg dw–1 · hr–1 (Ivy, Lee, et al., 1988). When sufficient CHO is ingested immediately after cessation of exercise, muscle glycogen-synthesis rates increase up to 20–45 mmol · kg dw–1 · hr–1 (Ivy, Katz, et al., 1988; Ivy, Lee, et al., 1988b; Jentjens, van Loon, Mann, Wagenmakers, & Jeukendrup, 2001; Keizer et al., 1987; Maehlum et al., 1978; Maehlum et al., 1977; van Hall, Shirreffs, & Calbet, 2000; van Loon, Saris, Kruijshoop, & Wagenmakers, 2000). Several studies have investigated the impact of consuming different amounts of CHO and the frequency with which CHO supplements are provided on postexercise muscle glycogen repletion (Table 1). Blom, Hostmaker, Vaage, and Maehlum (1987) initially suggested that a CHO intake of 0.35 g · kg–1 · hr–1, provided at 2-hr intervals, allows maximal muscle glycogen-synthesis rates. However, several other studies showed higher muscle glycogen-synthesis rates when 0.75–1.0 g CHO · kg–1 · hr–1 was provided (Casey, Short, Hultman, & Greenhaff, 1995; Ivy, Lee, et al., 1988; McCoy, Proietto, & Hargreaves, 1996; Tarnopolsky et al., 1997). Studies that applied a more frequent CHO supplementation protocol (i.e., every 15–30 min) reported even greater glycogen-synthesis rates (Jentjens et al., 2001; van Hall et al., 2000; van Loon, Saris, Kruijshoop, & Wagenmakers, 2000). Previously, van Loon, Saris, Kruijshoop, and Wagenmakers (2000) showed that the administration of 1.2 g · kg–1 · hr–1 CHO provided by CHO supplements every
30 min during postexercise recovery resulted in a muscle glycogen content that was 150% greater than with the ingestion of 0.8 g · kg\(^{-1} \cdot \text{hr}^{-1}\) (Figure 2). Because a further increase in the amount of CHO ingestion up to 1.6 g · kg\(^{-1} \cdot \text{hr}^{-1}\) does not seem to further augment postexercise muscle glycogen repletion (Howarth, Moreau, Phillips, & Gibula, 2009), 1.2 g CHO · kg\(^{-1} \cdot \text{hr}^{-1}\) can be considered the optimal amount of CHO intake to maximize postexercise muscle glycogen-storage rates.

Besides the amount of CHO, the timing and frequency of ingestion are also important factors that can modulate the rate of postexercise muscle glycogen repletion. More frequent provision of CHO supplements seems to further stimulate postexercise skeletal-muscle glucose uptake and glycogen repletion compared with ingestion at 2-hr intervals (Jentjens et al., 2001; van Hall et al., 2000; Tarnopolsky et al., 1997). Providing CHO at more frequent intervals seems to optimize muscle glycogen levels, particularly during the first few hours of postexercise recovery. Costill et al. (1981) and Burke et al. (1996) reported that frequent feedings of a high-CHO diet did not further enhance 24-hr muscle glycogen synthesis compared with an equal amount of CHO provided over several main meals.

In the absence of CHO intake, the contraction-induced increase in postexercise glucose transport reverses rapidly (Goodyear et al., 1990), with the number of glucose transporters at the plasma membrane returning to baseline values less than 2 hr after cessation of exercise. In accordance, Ivy, Katz, et al. (1988), reported 45% lower muscle glycogen-synthesis rates when postexercise CHO intake was delayed for 2 hr than with immediate

### Table 1: Studies Investigating the Effect of Carbohydrate (CHO) Intake on Postexercise Muscle Glycogen Synthesis

<table>
<thead>
<tr>
<th>Reference</th>
<th>Exercise protocol</th>
<th>Participants</th>
<th>CHO intake (g · kg(^{-1} \cdot \text{hr}^{-1}))</th>
<th>CHO intake</th>
<th>Timing (hr)</th>
<th>Glycogen postexercise (mmol/kg dw)</th>
<th>Recovery period (hr)</th>
<th>Glycogen-synthesis rate (mmol · kg (\text{dw}^{-1} \cdot \text{hr}^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blom et al., 1987</td>
<td>Cycling to exhaustion</td>
<td>5 male</td>
<td>0.18, 0.35, 0.70</td>
<td>Glucose (drink)</td>
<td>0, 2, 4</td>
<td>137, 64, 98</td>
<td>0–5</td>
<td>9.0, 24.8, 24.4</td>
</tr>
<tr>
<td>Casey et al., 1995</td>
<td>1-legged cycling to exhaustion</td>
<td>7 male</td>
<td>1.0</td>
<td>Glucose (drink)</td>
<td>0, 1, 2</td>
<td>25</td>
<td>0–3</td>
<td>40</td>
</tr>
<tr>
<td>Howarth et al., 2009</td>
<td>2 hr cycling</td>
<td>6 male</td>
<td>1.2, 1.6</td>
<td>Glucose (drink)</td>
<td>15-min intervals</td>
<td>90, 80</td>
<td>0–4</td>
<td>23, 25</td>
</tr>
<tr>
<td>Ivy, Katz, et al., 1988</td>
<td>70 min interval cycling</td>
<td>12 male</td>
<td>1.0, 0, 0, 1.0</td>
<td>Glucose (drink)</td>
<td>0, 2</td>
<td>153, 132</td>
<td>0–2, 2–4</td>
<td>33, 10.7, 18.4, 17.6</td>
</tr>
<tr>
<td>Ivy, Lee, et al., 1988</td>
<td>2 hr cycling</td>
<td>8 male</td>
<td>0, 0.75, 1.5</td>
<td>Glucose (drink)</td>
<td>0</td>
<td>156, 153, 137</td>
<td>0–4</td>
<td>3.0, 22, 22</td>
</tr>
<tr>
<td>Jentjens et al., 2001</td>
<td>Glycogen-depletion cycling</td>
<td>8 male</td>
<td>1.2</td>
<td>Glucose (drink)</td>
<td>30-min intervals</td>
<td>106</td>
<td>0–3</td>
<td>40</td>
</tr>
<tr>
<td>Keizer et al., 1987</td>
<td>Interval cycling to exhaustion</td>
<td>8 male</td>
<td>0.75</td>
<td>Solid, liquid</td>
<td>1-hr intervals</td>
<td>72, 69</td>
<td>0–5</td>
<td>24.8, 24.6</td>
</tr>
<tr>
<td>Maehlum et al., 1978</td>
<td>Cycling to exhaustion</td>
<td>6 male</td>
<td>0.55</td>
<td>Glucose (drink)</td>
<td>0.25</td>
<td>68</td>
<td>0–2.25</td>
<td>27.6</td>
</tr>
<tr>
<td>McCoy et al., 1996</td>
<td>2 hr cycling</td>
<td>11 male</td>
<td>1.0</td>
<td>Glucose (meals)</td>
<td>0, 2, 4</td>
<td>116</td>
<td>0–6</td>
<td>37.4</td>
</tr>
<tr>
<td>Tarnopolsky et al., 1997</td>
<td>90 min cycling</td>
<td>8 male, 8 female</td>
<td>0, ~1.0</td>
<td>Glucose (drink) + lunch(^{\text{a}})</td>
<td>0, 1 + lunch(^{\text{a}})</td>
<td>210, 163</td>
<td>0–4</td>
<td>7, 37</td>
</tr>
<tr>
<td>van Hall et al., 2000</td>
<td>Glycogen-depletion cycling</td>
<td>5 male</td>
<td>0, 1.2</td>
<td>Sucrose (drink)</td>
<td>15-min intervals</td>
<td>78, 90</td>
<td>0–4</td>
<td>11, 37</td>
</tr>
<tr>
<td>van Loon, Saris, Kruischoop, and Wagenmakers, 2000</td>
<td>Glycogen-depletion cycling(^{\text{a}})</td>
<td>8 male</td>
<td>0.8, 1.2</td>
<td>Glucose (drink)</td>
<td>30-min intervals</td>
<td>190, 138</td>
<td>0–5</td>
<td>16.6, 44.8</td>
</tr>
</tbody>
</table>

**Note.** dw = dry weight. All studies tested endurance-trained subjects with a VO\(_{\text{max}}\) range of 44–67 ml · kg\(^{-1} \cdot \text{min}^{-1}\).

\(^{\text{a}}\)Glycogen-depletion protocol adapted from Kuipers, Saris, Brouns, Keizer, and ten Bosch (1989). \(^{\text{b}}\)In this study by Tarnopolsky et al., subjects received 1 g/kg glucose or placebo immediately and 1 hr after exercise. However, during the 4-hr recovery period they also received a standardized lunch, which is not described in detail. Therefore, we cannot provide the exact glucose-ingestion rate during recovery, but it is estimated to be 0.75–1.0 g · kg\(^{-1} \cdot \text{hr}^{-1}\). 

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30 min during postexercise recovery resulted in a muscle glycogen content that was 150% greater than with the ingestion of 0.8 g · kg\(^{-1} \cdot \text{hr}^{-1}\) (Figure 2). Because a further increase in the amount of CHO ingestion up to 1.6 g · kg\(^{-1} \cdot \text{hr}^{-1}\) does not seem to further augment postexercise muscle glycogen repletion. More frequent provision of CHO supplements seems to further stimulate postexercise skeletal-muscle glucose uptake and glycogen repletion compared with ingestion at 2-hr intervals (Jentjens et al., 2001; van Hall et al., 2000; van Loon, Saris, Kruischoop, & Wagenmakers, 2000). Providing CHO at more frequent intervals seems to optimize muscle glycogen levels, particularly during the first few hours of postexercise recovery. Costill et al. (1981) and Burke et al. (1996) reported that frequent feedings of a high-CHO diet did not further enhance 24-hr muscle glycogen synthesis compared with an equal amount of CHO provided over several main meals.

In the absence of CHO intake, the contraction-induced increase in postexercise glucose transport reverses rapidly (Goodyear et al., 1990), with the number of glucose transporters at the plasma membrane returning to baseline values less than 2 hr after cessation of exercise. In accordance, Ivy, Katz, et al. (1988), reported 45% lower muscle glycogen-synthesis rates when postexercise CHO intake was delayed for 2 hr than with immediate
CHO ingestion. However, this seems only evident during short-term recovery (<8 hr), because Parkin, Carey, Martin, Stojanovska, and Febbraio (1997) observed no differences in muscle glycogen content after immediate and delayed CHO ingestion during recovery periods of 8 and 24 hr. More research is needed to establish the optimal timing and frequency of CHO supplementation to accelerate muscle glycogen repletion during the first stages of postexercise recovery.

Efforts to manipulate rates of muscle glycogen synthesis by changing the form of CHO administration (i.e., solid vs. liquid) have largely been unsuccessful (Keizer et al., 1987; Reed et al., 1989). In addition, the mode of glucose administration (i.e., oral vs. intravenous infusion) did not result in different postexercise muscle glycogen-synthesis rates (Reed et al., 1989). However, the glycemic index of the CHO sources might be of some importance. Burke et al. (1993) reported greater muscle glycogen concentrations after 24 hr of postexercise recovery when high-glycemic-index meals were ingested than with the ingestion of foods with a low glycemic index, despite the fact that the same amount of CHO was provided in both conditions (10 g/kg). However, the difference in 24-hr glycemic response between diets was neither significant nor of the same order of magnitude as the difference in glycogen storage. This was largely because the first meal consumed after exercise produced a large blood glucose response, regardless of the glycemic index of its composite foods. Therefore, the authors speculated that the difference in glycogen storage between the two diets might be better explained by the malabsorption of CHO in the low-glycemic-index foods, noting that diets should be constructed based on knowledge of available CHO in foods rather than total CHO per se. Further work, particularly among populations habituated to diets of different glycemic index, is warranted.

The large variability in postexercise muscle glycogen-synthesis rates between studies (for overview see Table 1) might be the result of differences in postexercise muscle glycogen content, the time interval of CHO supplementation, the training status of the subjects, the type of CHO consumed, or the duration of the recovery period during which muscle glycogen synthesis was assessed. It remains to be established whether gender affects muscle glycogen synthesis; only Tarnopolsky et al. (1997) addressed this issue and reported no differences in muscle glycogen-synthesis rates between men and women. However, when comparing the outcome of the various studies it seems reasonable to conclude that maximal postexercise muscle glycogen-synthesis rates occur at a CHO intake of ~1.2 g · kg⁻¹ · hr⁻¹ when provided frequently with 15- to 30-min intervals (Jentjens & Jeukendrup, 2003).

### Protein Coingestion

Coingestion of protein, protein hydrolysates, and/or free amino acids augments postprandial insulin secretion compared with CHO ingestion alone (Kaastra et al., 2006; Manders et al., 2006; Manders et al., 2005; Nuttall, Mooradian, Gannon, Billington, & Kreuzowski, 1984; Pallotta & Kennedy, 1968; Rabinowitz, Merimee, Maffezzoli, & Burgess, 1966; van Loon et al., 2003; van Loon, Kruisshoop, et al., 2000; van Loon, Saris, Verhagen,

![Figure 2](image-url)
In a recent study, Kaastra et al. showed that coingestion of a casein protein hydrolysate (0.4 g · kg⁻¹ · hr⁻¹) with CHO (0.8 g · kg⁻¹ · hr⁻¹) increased the postprandial insulin response more than twofold during postexercise recovery in young endurance-trained cyclists. In the same study they observed that addition of free leucine to the mixture (0.1 g · kg⁻¹ · hr⁻¹) could stimulate endogenous insulin release even further, resulting in a nearly threefold greater insulin response than the ingestion of CHO only. Because elevated insulin levels can stimulate glucose uptake and activate muscle glycogen synthase (Cartee et al., 1989; Jentjens & Jeukendrup, 2003; Wallberg-Henriksson et al., 1988), it has been speculated that coingestion of protein with CHO can further accelerate muscle glycogen synthesis during postexercise recovery. In accordance, Zawadzki et al. (1992) reported higher muscle glycogen-synthesis rates during 4 hr of postexercise recovery after coingestion of a whey-protein supplement (0.25 g · kg⁻¹ · hr⁻¹) than with ingestion of CHO only (0.75 g · kg⁻¹ · hr⁻¹). Many investigators criticized that study because it did not include an isoenergetic control trial. Consequently, it was suggested that the greater muscle glycogen-repletion rates after ingestion of the CHO and protein supplement might be attributed to the greater energy intake, increasing the availability of substrate for gluconeogenesis (Krebs et al., 2003). The latter seems unlikely because gluconeogenesis would contribute little under such postprandial conditions with high circulating plasma insulin levels (Barthel & Schmoll, 2003; Dentin et al., 2007), although a recent work in canines does not seem to support this hypothesis (Edgerton et al., 2009).

Several other studies (for an overview see Table 2) have compared the effect of CHO and protein coingestion during 4 hr of postexercise recovery after coingestion of a whey-protein supplement (0.25 g · kg⁻¹ · hr⁻¹) than with ingestion of CHO only (0.75 g · kg⁻¹ · hr⁻¹). Many investigators criticized that study because it did not include an isoenergetic control trial. Consequently, it was suggested that the greater muscle glycogen-repletion rates after ingestion of the CHO and protein supplement might be attributed to the greater energy intake, increasing the availability of substrate for gluconeogenesis (Krebs et al., 2003). The latter seems unlikely because gluconeogenesis would contribute little under such postprandial conditions with high circulating plasma insulin levels (Barthel & Schmoll, 2003; Dentin et al., 2007), although a recent work in canines does not seem to support this hypothesis (Edgerton et al., 2009).

### Table 2 Studies Investigating the Effect of Carbohydrate (CHO) and Protein (PRO) Coingestion on Postexercise Muscle Glycogen Synthesis

<table>
<thead>
<tr>
<th>Reference</th>
<th>Exercise protocol</th>
<th>Participants</th>
<th>CHO intake (g · kg⁻¹ · hr⁻¹)</th>
<th>PRO intake (g · kg⁻¹ · hr⁻¹)</th>
<th>Timing (hr)</th>
<th>Glycogen postexercise (mmol/kg dw)</th>
<th>Recovery period (hr)</th>
<th>Glycogen-synthesis rate (mmol · kg dw⁻¹ · hr⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Berardi et al., 2006</td>
<td>1-hr time trial</td>
<td>6 male</td>
<td>0.6, 0.8</td>
<td>0.25, 0.05</td>
<td>0, 1, 2, 4</td>
<td>55, 56 mmol/L</td>
<td>0–6</td>
<td>4.8, 3.7 mmol · L⁻¹ · hr⁻¹</td>
</tr>
<tr>
<td>Carrithers et al., 2000</td>
<td>Glycogen-depletion cycling a</td>
<td>7 male</td>
<td>1.0, 0.7, 0.86</td>
<td>0, 0.20, 0.14</td>
<td>30-min intervals</td>
<td>107, 118, 87</td>
<td>0–4</td>
<td>31, 28, 30</td>
</tr>
<tr>
<td>Howarth et al., 2009</td>
<td>2 hr cycling</td>
<td>6 male</td>
<td>1.2, 1.2, 1.6</td>
<td>0, 0.4, 0</td>
<td>15-min intervals</td>
<td>90, 80, 70</td>
<td>0–4</td>
<td>23, 25, 25</td>
</tr>
<tr>
<td>Ivy et al., 2002</td>
<td>Glycogen-depletion cycling b</td>
<td>7 male</td>
<td>0.5, 0.5, 0.7</td>
<td>0, 0.2, 0</td>
<td>0, 2</td>
<td>41, 41, 42 mmol/L</td>
<td>0–4</td>
<td>8, 12, 7.5 mmol · L⁻¹ · hr⁻¹</td>
</tr>
<tr>
<td>Jentjens et al., 2001</td>
<td>Glycogen-depletion cycling c</td>
<td>8 male</td>
<td>1.2, 1.2</td>
<td>0, 0.4</td>
<td>30-min intervals</td>
<td>106, 176</td>
<td>0–3</td>
<td>40, 25</td>
</tr>
<tr>
<td>Roy and Tarnopolsky, 1998</td>
<td>Resistance exercise at 80% 1-RM</td>
<td>10 male</td>
<td>0, 0.5, 0.33</td>
<td>0, 0, 0.11</td>
<td>0, 1</td>
<td>248, 235, 220</td>
<td>0–4</td>
<td>2.0, 19.3, 23.0</td>
</tr>
<tr>
<td>Tarnopolsky et al., 1997</td>
<td>90 min cycling</td>
<td>8 male, 8 female</td>
<td>~0.6–0.75 a, ~0.75–1.0 b</td>
<td>~0.08–0.14, 0</td>
<td>0, 1</td>
<td>142, 163</td>
<td>0–4</td>
<td>25, 37</td>
</tr>
<tr>
<td>van Hall et al., 2000</td>
<td>Glycogen-depletion cycling c</td>
<td>5 male</td>
<td>1.2, 1.2</td>
<td>0, 0.35</td>
<td>15-min intervals</td>
<td>90, 69</td>
<td>0–4</td>
<td>37, 37</td>
</tr>
<tr>
<td>van Loon, Saris, Kruijshoop, and Wagenmakers, 2000</td>
<td>Glycogen-depletion cycling c</td>
<td>8 male</td>
<td>0.8, 1.2</td>
<td>0.4, 0</td>
<td>30-min intervals</td>
<td>174, 138</td>
<td>0–5</td>
<td>35.4, 44.8</td>
</tr>
<tr>
<td>Zawadzki et al., 1992</td>
<td>2 hr cycling</td>
<td>9 male</td>
<td>0.75, 0, 0.75</td>
<td>0, 0.25, 0.25</td>
<td>0, 2</td>
<td>233, 188, 217</td>
<td>0–4</td>
<td>25.6, 7.6, 35.5</td>
</tr>
</tbody>
</table>

Note. dw = dry weight. All studies tested endurance-trained subjects with a VO₂max range of 44–67 ml · kg⁻¹ · min⁻¹.

aGlycogen-depletion protocol consisted of 75 min cycling followed by six 1-min sprints. bGlycogen-depletion protocol consisted of 2 hr cycling followed by 1-min sprints to exhaustion. cGlycogen-depletion protocol adapted from Kuipers et al. (1989). dIn this study by Tarnopolsky et al., subjects received 1 g/kg glucose or 0.75 g/kg glucose plus 0.1 g/kg protein immediately and 1 hr after exercise. However, during the 4-hr recovery period they also received a standardized lunch, which is not described in detail. Therefore, we cannot provide the exact glucose- and protein-ingestion rates during recovery, but they are estimated to be 0.75–1.0 or 0.6–0.75 g · kg⁻¹ · hr⁻¹ glucose and 0.08–1.0 g · kg⁻¹ · hr⁻¹ protein.
on postexercise muscle glycogen synthesis with that of ingestion of an isoenergetic amount of CHO (Berardi, Price, Noreen, & Lemon, 2006; Carrithers et al., 2000; Howarth et al., 2009; Ivy et al., 2002; Jentjens et al., 2001; Roy & Tarnopolsky, 1998; Tarnopolsky et al., 1997; van Hall et al., 2000; van Loon, Saris, Kruisjhoop, & Wagenmakers, 2000). Even though some groups have reported accelerated postexercise muscle glycogen-synthesis rates after amino acid/protein coingestion (Berardi et al., 2006; Ivy et al., 2002), others have failed to confirm these findings (Carrithers et al., 2000; Howarth et al., 2009; Jentjens et al., 2001; Roy et Tarnopolsky, 1998; Tarnopolsky et al., 1997; van Hall et al., 2000; van Loon, Saris, Kruisjhoop, & Wagenmakers, 2000). In accordance with the data of Zawadzki et al. (1992), van Loon, Saris, Kruisjhoop, and Wagenmakers investigated the dietary effect of protein coingestion (0.4 g · kg\(^{-1}\) · hr\(^{-1}\)) with CHO (0.8 g · kg\(^{-1}\) · hr\(^{-1}\)) on postexercise muscle glycogen repletion and reported increased muscle glycogen-synthesis rates with the coingestion of protein (35.4 vs. 16.6 mmol · kg dw \(^{-1}\) · hr\(^{-1}\) after the ingestion of CHO and protein vs. CHO only, respectively). However, increasing CHO intake to 1.2 g · kg\(^{-1}\) · hr\(^{-1}\) also increased muscle glycogen-repletion rates, which did not significantly differ from the rates observed after ingesting CHO and protein (Figure 2). The latter clearly shows that CHO intake should be greater than 0.8 g · kg\(^{-1}\) · hr\(^{-1}\) to allow maximal postexercise glycogen-synthesis rates.

Because similar glycogen-synthesis rates were observed after ingestion of CHO (0.8 g · kg\(^{-1}\) · hr\(^{-1}\)) with the amino acid/protein mixture (0.4 g · kg\(^{-1}\) · hr\(^{-1}\)) versus ingestion of an isoenergetic amount of CHO (1.2 g · kg\(^{-1}\) · hr\(^{-1}\)), it was questioned whether amino acid/protein coingestion represents a feasible dietary strategy to further accelerate muscle glycogen repletion when more than 0.8 g · kg\(^{-1}\) · hr\(^{-1}\) CHO is ingested. Since then, 3 studies addressed this research question (Howarth et al., 2009; Jentjens et al., 2001; van Hall et al., 2000), and it showed that the addition of protein to 1.2 g · kg\(^{-1}\) · hr\(^{-1}\) CHO did not further increase postexercise muscle glycogen-synthesis rates compared with the ingestion of the same amount of CHO (Jentjens et al., 2001; van Hall et al., 2000) or an isoenergetic amount of CHO (Howarth et al., 2009), the latter despite higher circulating plasma insulin concentrations after protein coingestion in two of the studies (Jentjens et al., 2001; van Hall et al., 2000). It was suggested that a further increase in insulin release does not further accelerate blood glucose disposal or increase glycogen synthase activity when ample amounts of CHO (>1.0 g · kg\(^{-1}\) · hr\(^{-1}\)) are already provided.

Although this could be a valid explanation, it should be noted that the latter studies assessed postexercise muscle glycogen synthesis over relatively short recovery periods of only 3 (Jentjens et al., 2001) and 4 hr (Howarth et al., 2009; van Hall et al., 2000). It could be speculated that this time interval is too short to fully establish the impact of the greater postprandial insulin response after protein coingestion on subsequent postexercise muscle glycogen-synthesis rates, because glucose uptake and subsequent muscle glycogen synthesis will likely become more insulin dependent throughout the recovery period. Furthermore, it might be speculated that similar postexercise glycogen-synthesis rates acquired with a lower exogenous CHO intake would be preferred by athletes. The greater postprandial insulin levels after protein coingestion might stimulate the storage of the ingested CHO in the more insulin-sensitive tissues such as liver and previously exercised skeletal muscle, resulting in more efficient storage of the ingested CHO. Selective CHO uptake in previously exercised muscles will most likely improve performance in athletes who have to compete several times daily, because they experience a much shorter time frame for postexercise recovery.

In summary, coingestion of protein or amino acids with CHO does not further increase postexercise muscle glycogen synthesis when an ample amount of CHO (1.2 g · kg\(^{-1}\) · hr\(^{-1}\)) is ingested at frequent intervals (every 15–30 min). Coingestion of an insulinotropic amino acid/protein mixture might accelerate postexercise muscle glycogen-synthesis rates when less CHO is provided (<1.0 g · kg\(^{-1}\) · hr\(^{-1}\)).

### Caffeine Coingestion

Recently, Pedersen et al. (2008) reported a positive effect of caffeine coingestion with CHO on postexercise muscle glycogen synthesis. They showed a 66% greater increase in muscle glycogen-synthesis rate (58 vs. 38 mmol · kg dw \(^{-1}\) · hr\(^{-1}\); p < .05) over 4 hr of postexercise recovery when 2 mg · kg\(^{-1}\) · hr\(^{-1}\) caffeine was coingested with 1.0 g · kg\(^{-1}\) · hr\(^{-1}\) CHO. This is the first study to show a stimulating effect of caffeine coingestion on postexercise muscle glycogen repletion. The effect of caffeine supplementation on muscle glycogen synthesis was studied previously by Battram, Shearer, Robinson, and Graham (2004), who failed to observe any benefit of caffeine coingestion. However, subjects in the latter study received the caffeine supplementation (6 mg/kg) before and during exercise, whereas the CHO beverages (1.0 g · kg\(^{-1}\) · hr\(^{-1}\)) were provided during postexercise recovery. As such, it might be suggested that caffeine only exerts its effect on muscle glycogen synthesis when coingested with CHO. The mechanism by which caffeine coingestion might accelerate postexercise muscle glycogen synthesis remains debatable. In resting conditions, caffeine ingestion has been shown to result in a reduction in insulin-mediated glucose disposal (Battram, Arthur, Weekes, & Graham, 2006; Battram, Graham, & Dela, 2007; Greer, Hudson, Ross, & Graham, 2001; Moissey, Kacker, Bickerton, Robinson, & Graham, 2008; Pizzoli et al., 1998; Thong et al., 2002; Thong & Graham, 2002), which might be mediated by both β-adrenergic stimulation and adenosine-receptor antagonism (Battram et al., 2007; Thong & Graham, 2002). However, exercise has been shown to reduce the detrimental effects of caffeine on insulin action in muscle (Thong et al., 2002). Furthermore, caffeine coingestion during exercise seems to augment the intestinal absorption of...
CHO (Van Nieuwenhoven, Brummer, & Brouns, 2000; Yeo, Jentjens, Wallis, & Jeukendrup, 2005). It remains to be determined whether caffeine coingestion also stimulates intestinal glucose absorption during postexercise recovery and whether this might lead to greater muscle glycogen-synthesis rates. Furthermore, because caffeine coingestion may accelerate muscle glycogen synthesis by a mechanism other than protein coingestion, future studies should also focus on the additive effects of caffeine plus protein coingestion with CHO.

More studies are warranted to assess the proposed benefits of caffeine coingestion as a means to promote postexercise muscle glycogen repletion. In particular, studies should define the lowest caffeine intake at which any benefits on glycogen storage can be seen. After all, athletes will need to consider the possible disadvantages of caffeine intake on other aspects of recovery such as interference with sleep, because the interval between training sessions or competition may span periods in which an athlete would normally rest.

**Postexercise Muscle Protein Synthesis**

Besides the repletion of muscle glycogen stores, muscle-damage repair and skeletal-muscle reconditioning are important determinants of postexercise recovery. A positive muscle protein balance is needed to facilitate the repair of exercise-induced muscle damage and to allow the skeletal muscle’s adaptive response to exercise training to occur (Hawley et al., 2006). In the resting, fasted state muscle protein-breakdown rates exceed muscle protein-synthesis rates, and as a consequence net muscle protein balance is negative (Biolo, Maggi, Williams, Tipton, & Wolfe, 1995; Phillips, Tipton, Aarsland, Wolf, & Wolfe, 1997; Pitkanen et al., 2003; Tipton et al., 1996). Exercise stimulates both muscle protein synthesis and muscle protein breakdown during acute postexercise recovery (Biolo et al., 1995b; Biolo et al., 1999; Phillips et al., 1997; Pitkanen et al., 2003). Because exercise stimulates muscle protein synthesis more than muscle protein breakdown, net muscle protein balance becomes less negative. However, in the absence of subsequent food intake net protein balance after exercise does not become positive (Biolo, Maggi, et al., 1995; Phillips et al., 1997; Pitkanen et al., 2003). Postexercise nutrition is required to obtain a positive muscle protein balance and, as such, to facilitate muscle-damage repair and skeletal-muscle reconditioning (Figure 3).

**Carbohydrate Ingestion**

Several studies have examined the effect of postexercise CHO intake on skeletal-muscle protein balance. CHO ingestion after exercise attenuates the exercise-induced increase in muscle protein breakdown but does not affect muscle protein synthesis (Borsheim, Cree, et al., 2004; Miller et al., 2003; Roy et al., 1997). Consequently, postexercise net muscle protein balance becomes less negative after CHO ingestion (Borsheim, Cree, et al., 2004). The inhibitory effect of CHO administration on muscle protein breakdown has been attributed to the concomitant rise in circulating plasma insulin concentrations (Biolo et al., 1999; Denne, Liechty, Liu, Brechtel, & Baron, 1991; Fryburg, Jahn, Hill, Oliveras, & Barrett, 1995; Gelfand & Barrett, 1987; Greenhaff et al., 2008). Although some studies reported that elevated plasma insulin levels can stimulate muscle protein synthesis (Biolo, Declan Fleming, & Wolfe, 1995; Fujita, Rasmussen, Cadenas, Grady, & Volpi, 2006; Hillier, Fryburg, Jahn, & Barrett, 1998), these properties are only evident under conditions of increased plasma amino acid availability (Fujita et al., 2006). In contrast, several other studies indicate that insulin is merely permissive and does not represent a major factor regulating muscle protein synthesis (Denne et al., 1991; Gelfand & Barrett, 1987; Greenhaff et al., 2008; Koopman, Beelen, et al., 2007). In a recent study, Greenhaff et al. (2008) examined how graded doses of insulin during continuous hyperaminoacidemia affect muscle protein synthesis, breakdown, and net balance. Rates of mixed muscle protein synthesis were effectively stimulated by hyperaminoacidemia but were identical between conditions in which plasma insulin concentrations varied between 5 and 167 mU/L. A modest increase in insulin levels up to 30 mU/L reduced leg protein-breakdown rates by more than 50% and increased net protein leg balance, with no further increase at higher insulin concentrations. Thus, the positive effect of CHO administration on net protein balance seems to be mediated by the insulin-induced decrease in protein-breakdown rates, without any major impact on protein synthesis.

**Protein Ingestion**

The administration of amino acids and/or protein effectively stimulates muscle protein-synthesis rates at rest (Bohe, Low, Wolfe, & Rennie, 2003; Bohe, Low, Wolfe, & Rennie, 2001) and after exercise (Biolo, Tipton, Klein, & Wolfe, 1997; Wittert et al., 2009). Numerous studies have shown that amino acid and/or protein administration increases muscle protein-synthesis rates after resistance exercise (Borsheim, Aarsland, & Wolfe, 2004; Borsheim, Tipton, Wolf, & Wolfe, 2002; Gibala, 2000; Koopman, Pennings, Zorenc, & van Loon, 2007; Koopman et al., 2005; Levenhagen et al., 2002; Miller et al., 2003; Rasmussen et al., 2000; Tipton et al., 2007; Tipton et al., 1999; Tipton et al., 2001). Furthermore, amino acid and/or protein administration has also been shown to increase muscle protein-synthesis rates after endurance-type exercise (Gibala, 2007; Howarth et al., 2009; Levenhagen et al., 2002). However, there is still considerable debate on the exact amount and type of protein and the desired timing of protein ingestion to maximize postexercise muscle protein synthesis. Tipton et al. (1999) showed that postexercise ingestion of 40 g of either mixed amino acids (MAA) or essential amino acids only (EAA) effectively
stimulated muscle protein synthesis. Because the ingestion of 40 g MAA and 40 g EAA resulted in a similar net protein balance, it was suggested that it might not be necessary to ingest nonessential amino acids during immediate postexercise recovery. Follow-up studies assessed the impact of only 6 g EAA with and without CHO and showed that this amount was also effective in stimulating postexercise muscle protein synthesis (Borsheim et al., 2002; Miller et al., 2003; Rasmussen et al., 2000). However, ingestion of such a small amount of EAA after exercise resulted in a positive net protein balance for a period of up to 2 hr only, after which net protein balance became negative again (Borsheim et al., 2002). This could suggest that ingestion of this amount of amino acids is not sufficient to maintain an anabolic state. Recently, a study by Moore et al. (2009) revealed a dose-response relationship between protein ingestion and postexercise muscle protein-synthesis rates. The fractional synthetic rate of mixed muscle protein increased with the ingestion of greater amounts of protein, reaching a maximal rate after the ingestion of 20 g intact egg protein containing approximately 8.6 g EAs. The authors speculated that athletes should ingest this amount of protein five or six times daily to maximize muscle protein-synthesis rates throughout the day.

It seems obvious to question which protein or amino acid source would be most effective to promote postexercise muscle protein anabolism. It appears that milk

![Figure 3 — Muscle protein synthesis (S), breakdown (B), and net balance (N) at rest in the fasted state, after exercise in the fasted state, after exercise with carbohydrate supplementation, and after exercise with protein supplementation. Data adapted from Biolo, Williams, Fleming, and Wolfe (1999); Borsheim, Aarsland, and Wolfe (2004); Borsheim, Cree, et al. (2004); Borsheim, Tipton, Wolf, and Wolfe (2002); Howarth et al. (2009); Koopman, Wagenmakers, et al. (2005); Miller, Tipton, Chinkes, Wolf, and Wolfe (2003); Phillips et al. (1997), Pitkanen et al. (2003), Rasmussen, Tipton, Miller, Wolf, and Wolfe (2000); Roy, Tarnopolsky, MacDougall, Fowles, and Yarasheski (1997); Tipton, Ferrando, Phillips, Doyle, and Wolfe (1999); and Tipton, Ferrando, Williams, and Wolfe (1996).]
proteins and their isolated forms, whey and casein, offer an anabolic advantage over soy protein in promoting muscle hypertrophy (Fouillet, Mariotti, Gaudichon, Bos, & Tome, 2002; Tang & Phillips, 2009; Wilkinson et al., 2007). Casein and whey protein seem to have distinct anabolic properties, which is attributed to differences in digestion and absorption kinetics (Boirie et al., 1997; Dangin et al., 2001; Dangin et al., 2003; Tipton et al., 2004a). Whereas whey protein is a soluble protein that leads to fast intestinal absorption, intact casein clots in the stomach, delaying gastric emptying and the subsequent release of amino acids (Koopman et al., 2009). The fast but transient rise in plasma amino acid concentration after whey-protein ingestion leads to higher protein-synthesis and -oxidation rates (Boirie et al., 1997; Dangin et al., 2001; Dangin et al., 2003; Tang & Phillips, 2009). However, despite these differences, net protein balance during recovery from resistance-type exercise is similar after postexercise casein or whey consumption (Tipton et al., 2004). In addition to differences in digestion rate, it has been suggested that the mechanism by which whey more effectively promotes protein synthesis than casein could also be related to its slightly higher leucine content (Tang & Phillips, 2009). However, as shown by Koopman et al. (2005), the addition of leucine to a casein hydrolysate does not result in higher postexercise muscle protein-synthesis rates than the intake of casein hydrolysate only.

Besides the amount and type of protein ingested, the timing of ingestion seems to represent an important factor in stimulating postexercise muscle anabolism. Levenhaegen et al. (2001) reported an improved postexercise net protein balance after consumption of a protein, CHO, and fat supplement immediately after cessation of exercise as opposed to 3 hr later. Furthermore, recent studies suggest that CHO and protein coingestion before or during exercise may further augment postexercise muscle protein accretion (Beelen, Koopman, et al., 2008; Tipton et al., 2001). Tipton et al. (2001) showed that amino acid ingestion before, as opposed to after, exercise further augments net muscle protein accretion during recovery. The stimulating effect of amino acid supplementation before exercise on muscle protein synthesis after exercise has been attributed to a more rapid supply of amino acids to the muscle during the acute stages of postexercise recovery. However, it could also be speculated that protein ingestion before or during resistance-type exercise already stimulates muscle protein synthesis during exercise, thereby creating a longer time frame for muscle protein synthesis to be elevated. In a recent study, we confirmed that coingestion of a casein hydrolysate with CHO before and during 2 hr of intermittent resistance-type exercise stimulates muscle protein synthesis during exercise (Beelen, Koopman, et al., 2008). In addition, Fujita et al. (2009) reported that CHO and protein supplementation 1 hr before exhaustive leg resistance exercise inhibited the decrease in muscle protein-synthesis rate that was seen during exercise in a fasted state.

Because different types of protein differ in digestion rate and therefore their subsequent appearance in plasma (Koopman et al., 2009), the type and timing of protein ingestion are related. Tipton et al. (2001) previously demonstrated that amino acid uptake was greater when free essential amino acids plus CHO were ingested before resistance-type exercise than after exercise. However, the latter seems to depend also on the amino acid/protein source supplied (Tipton et al., 2007). More research is warranted to identify the optimal timing of ingestion of different protein sources to maximize muscle protein synthesis.

It has been speculated that the observed impact of protein coingestion on mixed muscle protein synthesis during exercise is merely restricted to intermittent, resistance-type exercise (Beelen, Koopman, et al., 2008; Fujita et al., 2009). It is attractive to assume that AMPK is not continually activated throughout intermittent, resistance-type exercise when the exercise is performed in the fed state. The latter could prevent its proposed inhibitory effect on muscle protein synthesis (Atherton & Rennie, 2006; Dreyer et al., 2006; Fujita et al., 2009; Koopman, Zorenc, Gransier, Cameron-Smith, & van Loon, 2006; Rose & Richter, 2009) and allow protein-synthesis rates to increase during resting periods between sets. It remains to be determined whether protein ingestion before or during exercise also increases muscle protein synthesis during continuous endurance-type exercise. Preliminary findings in our laboratory seem to indicate that even during moderate-intensity endurance-type exercise (50% Wmax), muscle protein synthesis can be stimulated in the working muscle by dietary protein coingestion before and during exercise (unpublished observations). More research is warranted to address the relevance of the potential to stimulate muscle protein synthesis during exercise.

The combined ingestion of CHO and protein/amino acids in the postexercise recovery phase can further stimulate net protein balance. Besides providing amino acids as precursors for protein synthesis, combined ingestion of CHO and protein/amino acids can elicit a strong insulinotropic response (Kaastra et al., 2006; Manders et al., 2006; Manders et al., 2005; Nuttall et al., 1984; Pallotta & Kennedy, 1968; Rabinowitz et al., 1966; van Loon, Kruijshoop, et al., 2000; van Loon, Saris, Verhaegen, & Wagenmakers, 2000). In a recent study, Koopman, Beelen, et al. (2007), showed that the coingestion of different amounts of CHO (0, 0.15, and 0.60 g · kg−1 · hr−1) with an ample amount of protein (0.3 g · kg−1 · hr−1) resulted in similar muscle protein-synthesis rates during 6 hr of postexercise recovery, despite elevated plasma insulin levels when protein was coingested with the highest amount of CHO. Although another study also indicated that muscle protein synthesis is already maximally stimulated at normal postprandial insulin concentrations (Greenhaff et al., 2008), further increasing plasma insulin might still effectively inhibit muscle proteolysis (Biolo et al., 1999; Gelfand & Barrett, 1987; Greenhaff et al., 2008), thereby increasing postexercise muscle protein net balance. Overall, the intake of ~20 g intact protein or ~9 g EAA is sufficient to stimulate
Postexercise Nutrition and Subsequent Performance

As mentioned before, nutritional support during acute postexercise recovery is especially important for athletes who need to perform multiple training or competition sessions on the same or successive days and who need to maintain performance during the subsequent exercise sessions. Several studies have examined exercise performance after a short period of postexercise recovery from prolonged endurance-type exercise during which either CHO (Betts, Williams, Duffy, & Gunner, 2007; Casey et al., 2000; Fallowfield, Williams, & Singh, 1995; Tsintzas et al., 2003; Wong & Williams, 2000) or CHO and protein (Berardi, Noreen, & Lemon, 2008; Betts et al., 2007; Karp et al., 2006; Millard-Stafford et al., 2005; Williams, Raven, Fogt, & Ivy, 2003) were ingested. Although CHO ingestion during 4 hr of recovery from a first exercise session has been shown to improve exercise performance during a second exercise bout (Casey et al., 2000; Fallowfield et al., 1995), there is some inconsistency regarding the amount that needs to be ingested (Betts et al., 2007; Tsintzas et al., 2003; Williams et al., 2003; Wong & Williams, 2000). Wong and Williams showed no difference in exercise performance after the ingestion of 0.15 versus 0.52 g · kg⁻¹ · hr⁻¹ CHO during 4 hr of recovery from a first bout of endurance-type exercise.

To investigate the rationale behind the findings of Wong and Williams (2000), Tsintzas et al. (2003) repeated the study and reported similar muscle glycogen utilization during a second exercise bout, despite an increased muscle glycogen-synthesis rate after the ingestion of 0.52 versus 0.15 g · kg⁻¹ · hr⁻¹ CHO. This implies that muscle glycogen content is not necessarily the more important factor determining subsequent performance capacity. However, the amount of CHO that was ingested during recovery in these studies (Tsintzas et al., 2003; Wong & Williams, 2000) was much lower than the recommended 1.2 g · kg⁻¹ · hr⁻¹ (Howarth et al., 2009; Jentjens et al., 2001; van Hall et al., 2000; van Loon, Saris, Kruifshoop, & Wagenmakers, 2000) and therefore resulted in muscle glycogen-synthesis rates of only ~19 mmol · kg dw⁻¹ · hr⁻¹ compared with ~45 mmol · kg dw⁻¹ · hr⁻¹ usually reported after ingestion of greater amounts of CHO (Casey et al., 1995; Jentjens et al., 2001; van Hall et al., 2000; van Loon, Saris, Kruifshoop, & Wagenmakers, 2000). In accordance with the increased muscle glycogen-synthesis rates van Loon, Saris, Kruifshoop, and Wagenmakers (2000) showed with the ingestion of 1.2 g · kg⁻¹ · hr⁻¹ CHO or 0.8 g · kg⁻¹ · hr⁻¹ CHO plus 0.4 g · kg⁻¹ · hr⁻¹ protein, Betts et al. (2007) reported improved performance during a second exercise session when CHO intake during 4 hr of recovery from the first session was increased from 0.8 to 1.1 g · kg⁻¹ · hr⁻¹. Furthermore, the addition of 0.3 g · kg⁻¹ · hr⁻¹ protein to 0.8 g · kg⁻¹ · hr⁻¹ CHO resulted in a performance effect similar to that of intake of 1.1 g · kg⁻¹ · hr⁻¹ CHO. In addition, Berardi et al. (2008) showed greater recovery of performance relative to an earlier exercise session when 0.8 g · kg⁻¹ · hr⁻¹ CHO plus 0.4 g · kg⁻¹ · hr⁻¹ protein was ingested during the intervening 6-hr recovery period than with the ingestion of 1.2 g · kg⁻¹ · hr⁻¹ CHO only. Therefore, it seems likely to assume that for optimal performance during a second exercise session, athletes should consume a amount of CHO during recovery from the first session that maximally stimulates postexercise muscle glycogen synthesis (~1.0–1.2 g · kg⁻¹ · hr⁻¹). However, it remains to be determined whether the rate of muscle glycogen repletion during short-term recovery is directly related to performance during a second exercise session and whether the addition of protein or specific amino acids can further enhance exercise performance by mechanisms other than accelerating muscle glycogen synthesis.

Future Research

The application of specific postexercise nutritional strategies to improve recovery represents a major factor in allowing athletes to maintain performance capacity and improve the adaptive response to exercise training. Most recovery studies have assessed the impact of nutritional supplementation after a single bout of exercise performed in the overnight fasted state. However, this is generally not representative of a normal exercise training or competition routine in which athletes generally follow standard precompetition dietary guidelines. In addition, most recreational athletes exercise in the evening and have dinner before or after training, which means that a large part of postexercise recovery generally takes place during overnight sleep. In this respect, the impact of postexercise nutrition has hardly been investigated. We recently assessed the impact of CHO and protein ingestion on muscle protein synthesis during exercise performed in the evening and during subsequent recovery during the night (Beelen, Tielen, et al., 2008). Although muscle protein synthesis during exercise was higher with CHO and protein ingestion than with placebo, muscle protein synthesis during overnight recovery was similar between treatments. However, when we calculated whole-body protein-turnover rates, protein synthesis was higher in the CHO and protein experiment than with placebo for the first 3 hr of overnight recovery. During this period only, plasma amino acid concentrations were greater in the CHO plus protein experiment. Increased extracellular amino acid availability is a major stimulus for protein synthesis, provided with either a continuous or an intermittent supplementation regimen.
(Bohe et al., 2003; Wolfe, 2002). However, ingesting subsequent boluses of protein at regular time intervals is not a practical approach during overnight recovery. In this respect, a slowly digested protein may offer an opportunity to increase plasma amino acid concentrations over a longer period. Future research is needed to establish the impact of different protein sources and the timing and mode of dietary protein administration on subsequent muscle protein synthesis during overnight recovery.

Another opportunity to maximize the muscle’s adaptive response to exercise training is to increase the time frame of muscle protein synthesis by providing protein and/or amino acids during the exercise session. We recently showed that muscle protein-synthesis rates are increased during resistance-type exercise when a protein hydrolysate is coingested with CHO. And even during continuous endurance-type exercise, nutritional supplementation seems to increase muscle protein-synthesis rates (Beelen et al., unpublished data). However, the mechanisms by which nutritional supplementation stimulates muscle protein synthesis during exercise remain to be elucidated.

With regard to muscle glycogen, future research should focus on the practical implications of nutritional interventions to stimulate postexercise muscle glycogen recovery. Previous studies have shown that postexercise muscle glycogen resynthesis is maximally stimulated when large amounts of CHO are ingested frequently. However, the practicality of this feeding regimen is questionable, especially for athletes who have to perform a second exercise session on the same day. Gastrointestinal complications are common with the ingestion of such high amounts of CHO (Jeukendrup, 2004; Van Nieuwenhoven et al., 2000). Dietary strategies should seek to maximize the uptake of exogenous CHO in exercised muscle. Strategies to increase endogenous insulin release, like protein and/or amino acids, or modulate gastrointestinal glucose uptake could further augment the availability of exogenous CHO for postexercise muscle glycogen synthesis.

Therefore, future studies should aim to identify the preferred composition of postexercise sports nutrition to optimize muscle glycogen repletion and augment muscle protein synthesis that can be implemented in practical, sport-specific settings.

**Practical Guidelines and Conclusions**

Postexercise nutrition is important to replenish endogenous substrate stores and to facilitate skeletal-muscle damage repair and reconditioning. Endurance athletes generally focus on the intake of CHO to restore muscle glycogen, whereas resistance-type athletes are mainly interested in the postexercise ingestion of protein to allow skeletal-muscle reconditioning and increase muscle mass. However, both types of athletes will benefit from the combined ingestion of CHO and protein. Postexercise CHO ingestion has been well established as the most important factor determining the rate of muscle glycogen synthesis. Coingestion of protein and/or amino acids does not seem to further increase the rate of muscle glycogen synthesis when postexercise CHO intake exceeds 1.2 g · kg\(^{-1}\) · hr\(^{-1}\). However, from a practical point of view it is not always feasible to ingest such large amounts of CHO. The combined ingestion of a small amount of protein (0.2–0.4 g · kg\(^{-1}\) · hr\(^{-1}\)) with a lesser amount of CHO (0.8 g · kg\(^{-1}\) · hr\(^{-1}\)) stimulates endogenous insulin release and accelerates muscle glycogen repletion. The latter might represent a more practical approach to optimize postexercise glycogen repletion, because it has been shown to result in rates of muscle glycogen-synthesis similar to those with the intake of larger amounts of CHO.

Furthermore, postexercise protein and/or amino acid administration is warranted to stimulate mixed muscle protein synthesis, inhibit protein breakdown, and allow net muscle protein accretion. The latter is believed to be required to optimize skeletal-muscle-damage repair and allow muscle-tissue reconditioning. The consumption of ~20 g intact protein, or an equivalent of ~9 g EAA, has been reported to be sufficient to maximize muscle protein-synthesis rates during the first few hours of postexercise recovery. Ingestion of such relatively small amounts of dietary protein five or six times daily might support maximal muscle protein-synthesis rates throughout the day. The consumption of CHO and protein during the early phases of postexercise recovery has been shown to positively affect subsequent exercise performance and could be of specific benefit for athletes involved in multiple training or competition sessions on the same or successive days.

**References**


induced impairment of insulin action but not insulin signaling in human skeletal muscle is reduced by exercise. *Diabetes, 51*(3), 583–590.


