Postexercise Whole-Body Protein Turnover Response to Three Levels of Protein Intake

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ABSTRACT


Purpose: This investigation examined the effect of variations in protein intake on whole-body protein turnover (WBPTO) after exercise in endurance-trained males.

Methods: Five male runners (21.3 ± 0.3 yr, 179 ± 2 cm, 70.6 ± 0.1 kg, 8.7 ± 0.4% body fat, 70.6 ± 0.1 V̇O$_{2}$peak) participated in a randomized, crossover-design diet intervention, where they consumed either a low- (0.8 g.kg$^{-1}$; LP), moderate- (1.8 g.kg$^{-1}$; MP), or high-protein (3.6 g.kg$^{-1}$; HP) diet for 4 wk. WBPTO (Ra, leucine rate of appearance; NOLD, nonoxidative leucine disposal; and Ox, leucine oxidation) were assessed after a 75-min run at 70% V̇O$_{2}$peak after each diet-intervention period.

Results: Leucine Ra (indicator of protein breakdown) and leucine Ox were greater on the HP diet than on the LP diet (Ra, 123.4 ± 6.9 vs 97.9 ± 6.0 μmol.kg$^{-1}$.h$^{-1}$; Ox, 23.9 ± 0.5 vs 17.0 ± 0.8 μmol.kg$^{-1}$.h$^{-1}$, P < 0.05). No diet differences were noted in NOLD (an indicator of protein synthesis) across diets. Plasma branched chain amino acids (BCAA) at rest were greater for MP and HP than for LP, and nonessential amino acids (NEAA) were greater for LP than MP at rest and greater than MP and HP after exercise.

Conclusion: Findings from this study show that variations in protein intake can alter plasma amino acid levels and modulate rates of WBPTO after exercise. Additionally, a lower protein intake was associated with decreased rates of WBPTO after exercise.

Key Words: LEUCINE KINETICS, ENDURANCE EXERCISE, DIETARY PROTEIN, AMINO ACIDS

Rates of whole-body protein turnover (WBPTO) are determined by the relationships between, and alterations in, protein synthesis, breakdown, and oxidation. Both level of dietary protein intake and endurance exercise have been shown to influence turnover and oxidation of proteins (4,9,13,18,20). To date, studies have employed varied levels of dietary protein, different exercise protocols, and subjects of differing training states. As a result, the role of habitual dietary protein intake in the WBPTO response to an endurance exercise bout has not been fully delineated.

Varying the level of dietary protein alters whole-body protein metabolism. Several studies have shown an increase in both the oxidation of total protein and the branched chain amino acid (BCAA) leucine (4,11) with increasing protein intake that can likely be attributed to increased amino acid availability (4). Additionally, increases in dietary protein result in greater nitrogen retention (12,13,22,25,30). However, the response for rates of synthesis and breakdown to variations in protein intake are less clear. Although increased protein turnover is routinely associated with increases in protein intake in the postabsorptive state (13,22,23,25), this is not always the case (4,11). These discrepancies may be attributable to differences in the level of dietary protein, as well as the magnitude of change in level of protein intake implemented in the study designs. We previously reported that level of dietary protein intake influences WBPTO at rest, with increasing protein intake resulting in increased rates of whole-body protein breakdown and oxidation and an increase in nitrogen balance (13). In the current study, we examined how changes in habitual protein intake influences WBPTO after an acute bout of endurance exercise, because endurance exercise clearly impacts WBPTO.

Numerous studies have shown that endurance exercise significantly impacts whole-body protein metabolism. Leucine oxidation increases during exercise in an intensity-dependent manner and may contribute 2–6% of total energy expended during an exercise session (4,9,18,26). In the postabsorptive state, whole-body protein breakdown increases during exercise (26,36), with postexercise rates either decreasing (32) or being no different than those noted at rest (27). Whole-body protein synthesis has been found to decrease (4,27,36) or not change during exercise (7,26) but to increase in the period after exercise (8,27). Other factors that influence leucine oxidation include training state (20), glycogen stores (34), and exercise...
WHOLE-BODY PROTEIN TURNOVER AFTER EXERCISE

Methods

Subjects. After project approval by the institutional review board at the University of Connecticut, five male endurance athletes aged 22–29 yr and running at least 35 miles per week were recruited from the university community and local health/running clubs to participate in the study. Each participant provided a complete medical history, training schedule, and a 3-d food record. Individuals reporting metabolic or cardiovascular abnormalities, gastrointestinal disorders (i.e., lactose intolerance), use of nutritional/sports supplements or anabolic steroids, and those who considered themselves vegetarians, were excluded from the study. Informed written consent was obtained from all subjects.

Experimental design. This study was a crossover design with volunteers serving as their own controls. After initial baseline testing, subjects were randomly assigned to a diet containing either: 0.8 (low protein; LP), 1.8 (moderate protein; MP), or 3.6 (high protein; HP) grams of protein per kilogram of body weight (g kg⁻¹ d⁻¹) for 4 wk. After 4 wk on the diet, WBPTO was assessed after a 75-min run at 70% VO₂peak, using a primed continuous infusion of 1³⁵C-leucine. After an approximately 2-wk wash-out period, athletes crossed over to another diet intervention for 4 wk, and all measurements were repeated. All athletes were instructed to keep training journals that detailed their daily and weekly totals for running mileage. These were collected weekly throughout each of the diet interventions.

Anthropometric measures. Body mass and height were measured using a balance beam scale equipped with a measuring rod (Health-o-meter, Bridgeview, IL). Body weight was assessed at baseline and twice weekly throughout the study to ensure body-weight maintenance. Percent body fat was estimated through hydrostatic weighing, and values were calculated from body density according to equations by Brozek et al. (5).

VO₂peak. Before the start of the study, VO₂peak was determined via breath-by-breath analysis of expired gases during testing using an open-circuit respiratory apparatus (MedGraphics CPX/D, Medical Graphics Corporation, St. Paul, MN) on a treadmill (Quinton MedTrack ST55, Bothell, WA).

Dietary intervention. Protein intakes were set at either a low, moderate, or high protein-intake level (0.8, 1.8, or 3.6 g kg⁻¹ d⁻¹, for LP, MP, and HP, respectively). Diet interventions were designed such that percent of total calories contributed by the macronutrients approximated 60% carbohydrate, 30% fat, and 10% protein for LP; 55% carbohydrate, 30% fat, and 15% protein for MP; and 40% carbohydrate, 30% fat, and 30% protein for the HP diet. Diets were eucaloric, with an isoenergetic exchange between protein and carbohydrate. The predominant protein source at each meal was beef. Additionally, participants on the HP diet received two commercially available protein bars (Met-Rx Protein Plus, Irvine, CA) per day, providing approximately 300 kcal and 32 g of protein (15 g of carbohydrate, 8 g of fat) to increase their protein intake to the prescribed level. Menus incorporated food item exchange lists to meet the specified diet prescription for each individual and to ensure body-weight maintenance.

Participants were fed at the metabolic kitchen in the department of nutritional sciences and at a designated dining room through the department of catering at the University of Connecticut. Research assistants were present at all meals to weigh and serve the appropriate foods for each participant. Participants were not food restricted, per se; any food eaten in excess or less than that prescribed at each meal was recorded for that participant. All daily menus and baseline 3-d food records were analyzed for energy and macronutrient composition using Nutritionist Pro Software (N² Computing, Salem, OR).

Stable isotopes. Stock solutions of all stable isotopes were created and certified to be sterile and pyrogen free by the department of laboratory medicine, University of Connecticut Health Center, Farmington, CT. The stable isotopes tested were all commercially available products (Cambridge Isotope Laboratories, Cambridge, MA).

WBPTO determination. Subjects trained and competed throughout the study but refrained from exercise...
the day before the infusion protocols to minimize the influence of the last exercise session on WBPTO measures. On study days, subjects were transported to the metabolic assessment laboratory in the department of nutritional sciences at approximately 0700 h after an overnight fast (≥ 10 h). A 20-gauge Teflon catheter (7.78 cm, Jelco, Critikon, Tampa, FL) was inserted into an antecubital vein for isotope infusion. Another catheter (3.97 cm) was placed in the contralateral hand, and the hand was heated with a heating pad, for the sampling of arterialized blood.

After providing baseline blood and breath samples (0 min), subjects received a bolus injection of $^{13}$C-bicarbonate (0.295 mg·kg$^{-1}$) to prime the CO$_2$ pool, and then a primed continuous infusion of L-[1-$^{13}$C] leucine (4 μmol·kg$^{-1}$.h$^{-1}$, 4.8 μmol·kg$^{-1}$.h$^{-1}$) was initiated (Razel Syringe Pump, Razel Scientific Instruments Inc., Stamford, CT) and continued for 180 min. At 45 min, subjects were moved to the treadmill, where they began their 75-min run at 70% VO$_{2peak}$. Immediately after the run (120 min), blood and breath measurements were collected at 15-min intervals until 180 min. Plasma samples were stored at −80°C for subsequent analyses. $^{13}$CO$_2$ breath samples were collected by subjects breathing into a Douglas bag for 2 min and were then transferred to 20-mL Venoject containers for future analysis. Both the plasma $^{13}$C-KIC and breath $^{13}$CO$_2$ enrichments were determined by gas chromatography and isotope ratio mass spectrometry, respectively, by a commercial laboratory (Metabolic Solutions, Nashua, NH).

Blood $^{13}$C-KIC and breath $^{13}$CO$_2$ data for the five time points (120, 135, 150, 165, and 180 min) were evaluated to confirm steady-state conditions. Steady-state conditions were assumed when the coefficient of variation of the atom percent excess (APE) values at isotopic plateau was < 10%. Data from the five time points were averaged for each subject, and group means were determined. Leucine rate of appearance (Ra; an indicator of protein breakdown), leucine oxidation (Ox), and nonoxidative leucine disposal (NOLD; an indicator of protein synthesis) were then calculated using the reciprocal pool model (15). Net leucine balance (NET) was calculated by subtracting leucine Ra from NOLD.

**Insulin analysis.** Insulin concentrations were determined in duplicate from lithium-heparin processed samples using solid-phase, double-antibody radioimmunoassay (RIA) (DSL-1600, Diagnostic Systems Laboratories, Webster, TX).

**Plasma amino acids.** Plasma amino acids were analyzed at rest and immediately after exercise for each dietary intervention by derivatizing with phenylisothiocyanate and high-performance liquid chromatography (14).

**Statistical analysis.** Primary outcome measures were indices of WBPTO (Ra, Ox, and NOLD) after 4 wk on each dietary intervention. Group means for Ra, Ox, NOLD, plasma amino acid, and insulin concentrations were compared using repeated-measures ANOVA with group differences determined using Tukey’s post hoc analysis with an alpha level of 0.05. Simple correlations between resting plasma amino acid concentrations (nonessential amino acids (NEAA), essential amino acids (EAA), and BCAA) and markers of WBPTO (Leucine Ra, NOLD, and Ox) were calculated for each diet intervention. All data were analyzed using SPSS 11.0 statistical software (SPSS Inc., Chicago, IL). All data are presented as means ± SEM.

**RESULTS**

**Subject characteristics.** A homogenous group of five fit young males participated in this study (Table 1). Baseline dietary nutrient data from 3-d food records provided by the subjects revealed that the runners consumed approximately 2831 kcal, 52% carbohydrate (5.4 g·kg$^{-1}$), 17% protein (1.7 g·kg$^{-1}$), and 31% fat before participating in the study.

**Diet intervention.** The actual macronutrient breakdown was 66% carbohydrate, 27% fat, and 7% protein for LP; 60% carbohydrate, 26% fat, and 14% protein for MP; and 48% carbohydrate, 26% fat, and 26% protein for HP. Mean protein intake in grams per kilogram body weight was 0.87, 1.78, and 3.12, and carbohydrate intake was 8.3, 7.4, and 5.4 for LP, MP, and HP, respectively. Energy intake was not significantly different across the diets (3498 ± 33, 3463 ± 87, and 3347 ± 19 kcal for LP, MP, and HP, respectively).

**Plasma insulin.** Resting plasma insulin concentrations decreased with increasing dietary protein. There were significant differences in resting insulin concentrations between LP (11.7 ± 1.5 μU·mL$^{-1}$) and both MP (5.8 ± 0.5 μU·mL$^{-1}$) and HP (6.0 ± 1.2 μU·mL$^{-1}$), P < 0.01. Postexercise values did not differ between treatments (11.5 ± 1.7, 8.9 ± 4.8, and 9.0 ± 3.1 μU·mL$^{-1}$ for LP, MP, and HP, respectively).

**Plasma amino acids.** Plasma amino acid concentrations were influenced by level of protein intake at rest and after exercise (Table 2). At rest, BCAA were higher for HP and MP compared with LP, and NEAA were lower on MP than LP (P < 0.05). After exercise, NEAA were higher for LP than for both MP and HP (P < 0.05). There was also a significant negative correlation between baseline NEAA and leucine Ra after exercise (r = −0.7, P < 0.01). Therefore, higher plasma NEAA concentrations at rest after 4 wk of dietary intervention were correlated with decreased leucine Ra (whole-body protein breakdown) after exercise.

**TABLE 1. Subject characteristics (N = 5).**

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>21.3 ± 0.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>179.1 ± 1.6</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>70.8 ± 0.1</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>8.7 ± 0.4</td>
</tr>
<tr>
<td>VO$_{2peak}$ (mL·kg$^{-1}$·min$^{-1}$)</td>
<td>70.6 ± 0.1</td>
</tr>
<tr>
<td>Running distance (miles per week)</td>
<td>52 ± 2</td>
</tr>
</tbody>
</table>

Values are mean ± SE.
**WBPTO.** Leucine $Ra$ for LP ($97.9 \pm 6.0 \text{ mmol·kg}^{-1} \cdot \text{h}^{-1}$) was significantly lower ($P < 0.05$) than HP ($123.4 \pm 6.9 \text{ mmol·kg}^{-1} \cdot \text{h}^{-1}$) (Fig. 1). No differences in $Ra$ between MP (120.8 $\pm$ 4.4 mmol·kg$^{-1}$·h$^{-1}$) and LP were seen. However, there was a trend for greater $Ra$ on MP than LP ($P = 0.06$). Leucine $Ox$ was significantly lower ($P < 0.05$) for LP ($17.0 \pm 0.8 \text{ mmol·kg}^{-1} \cdot \text{h}^{-1}$) than for HP ($23.9 \pm 0.5 \text{ mmol·kg}^{-1} \cdot \text{h}^{-1}$). No differences in $Ox$ between MP (20.7 $\pm$ 2.7 mmol·kg$^{-1}$·h$^{-1}$) and LP or HP were found. There were no differences noted in NOLD across all three interventions (82.5 $\pm$ 5.7, 95.9 $\pm$ 5.2, and 98.4 $\pm$ 6.9 mmol·kg$^{-1}$·h$^{-1}$ for LP, MP, and HP, respectively). NET was more negative for HP compared with LP ($P < 0.05$).

**DISCUSSION**

The present investigation is unique, given its purpose to determine whether habitual changes in level of dietary protein could alter rates of WBPTO after an acute endurance exercise bout. The trained male runners in this study consumed three different levels of protein spanning the wide range of protein intakes recommended in the current DRI (16). The highest level of protein reflects the amount of protein in contemporary diet plans (i.e., 40:30:30 macronutrient composition), whereas the middle level was based on recommendations for protein intake for endurance athletes (19,29). The lowest level of protein is representative of the current recommended dietary allowance (RDA) for this age group. In theory, an increase in dietary protein will alter the plasma amino acid pool size and composition and, therefore, influence rates of protein turnover. We have previously reported that alterations in level of dietary protein influence rates of WBPTO at rest (13) and skeletal muscle fractional synthetic rates after exercise (3). The current study was designed to examine how altering the level of dietary protein influences WBPTO in response to an acute endurance exercise bout.

The results of the present investigation indicate that variations in habitual protein intake influence measurements of WBPTO after a prolonged endurance run (75 min at 70% $\dot{V}O_{2\text{peak}}$) in trained male runners. We observed the highest level of protein to result in elevated rates of leucine $Ra$ (protein breakdown) and leucine $Ox$ (oxidation) compared with the lowest level of protein. The increase in $Ra$ and $Ox$ on the HP diet after exercise are similar to what we have observed at rest, as shown in a previously published report that was part of this investigation (13).

Given that acute endurance exercise influences whole-body protein use by increasing the oxidation of amino acids (4,9,11,26,27,36), it has been routinely suggested that individuals who endurance train may require more dietary protein than their sedentary counterparts. Evans and colleagues (10) have demonstrated that a single endurance exercise bout (2 h at 55% $\dot{V}O_{2\text{peak}}$) is associated with the oxidation of as much as 86% of the daily requirement for leucine. Although this may not impact protein requirements for the recreational athlete, individuals who train daily at high intensities may have increased requirements because of chronic losses of this essential amino acid. Indeed, several previous studies of endurance-exercising individuals found that subjects were in negative nitrogen balance when consuming the RDA for protein (12,22,26,30). Therefore, the premise for the current study was to see how the habitual consumption of varied levels of protein would influence WBPTO variables and contribute to the ongoing discussion as to whether recommendations for increased dietary protein are warranted for endurance athletes.

The only consistent finding in previous studies characterizing changes in WBPTO in response to varied levels of protein is an increase in the oxidation of total protein and leucine at higher protein intakes (4,11) (Fig. 1). The responses for rates of synthesis and breakdown to variations in protein intake are less clear. Although it has repeatedly been

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**TABLE 2.** Plasma amino acids (mM) at rest and after a 75-min run at 70% $\dot{V}O_{2\text{peak}}$ (post) in five trained male runners (mean $\pm$ SE).

<table>
<thead>
<tr>
<th></th>
<th>Total NEAA</th>
<th>Total EAA</th>
<th>Total BCAA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Post</td>
<td>Rest</td>
</tr>
<tr>
<td>LP</td>
<td>1535.6 $\pm$ 63.3</td>
<td>1442 $\pm$ 56.7</td>
<td>693.6 $\pm$ 35.2</td>
</tr>
<tr>
<td>MP</td>
<td>1247.0 $\pm$ 63.1*</td>
<td>1125.7 $\pm$ 52.5**</td>
<td>751.7 $\pm$ 29.6</td>
</tr>
<tr>
<td>HP</td>
<td>1309.6 $\pm$ 90.6</td>
<td>1087.6 $\pm$ 87.5**</td>
<td>777.9 $\pm$ 40.8</td>
</tr>
</tbody>
</table>

LP, low protein; MP, moderate protein; HP, high protein; NEAA, nonessential amino acids; EAA, essential amino acids; BCAA, branched chain amino acids.

* Different from LP at rest, $P < 0.05$; ** different from LP at post, $P < 0.01$.

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**FIGURE 1**—Whole-body postexercise protein turnover (WBPTO) after 4 wk on either a low-(LP, 0.8 g kg$^{-1}$·d$^{-1}$), moderate- (MP, 1.8 g kg$^{-1}$·d$^{-1}$), or high-protein (HP, 3.6 g kg$^{-1}$·d$^{-1}$) diet. $Ra$ (leucine rate of appearance), NOLD (nonoxidative leucine disposal), $Ox$ (leucine oxidation), NET (net leucine balance: NOLD-$Ra$). * Different from LP, $P < 0.05$.
shown that with increasing protein intakes, there is an increase in protein turnover in the postabsorptive state (13,22,23,25), this is not always the case (4,11). Conversely, when protein intakes are suboptimal, the adaptive response is a downregulation in whole-body protein flux (21,23). A reduction in body-protein breakdown, in particular, has been identified as the primary response to optimize protein economy when intake is inadequate (21). These previous reports included dietary protein intakes in the range of 0.1–2.5 g·kg⁻¹·d⁻¹. This study (including Gaine et al. (13)) is the first to include a protein intake of > 3.0 g·kg⁻¹·d⁻¹. In both the present study (after exercise) and our previous report (at rest) (13), we documented that variations in levels of protein do alter rates of WBPTO, specifically resulting in changes in protein breakdown and leucine Ox.

In the present study, NET is simply the inverse of leucine Ox. We were not anticipating a positive NET balance on any of the diets, because leucine kinetics were applied during a fasted state. However, because we proposed that habitual consumption of varied levels of protein might impact amino acid pool size and, subsequently, amino acid availability, we were interested in whether NET would be less when these runners were chronically consuming protein in excess of the RDA. The finding of a more negative NET with HP was not anticipated. However, we believe that the more negative net noted with HP likely indicates consumption of protein and, hence, amino acids (i.e., leucine) in excess of what was needed for maintenance, repair, or synthesis of protein in these runners. As a result, leucine Ox was greatest and NET was most negative for HP compared with LP. These athletes were allowed sufficient time to adapt to each of the diets with regard to level of dietary protein intake (i.e., 4 wk). Although these young men were weight stable throughout the entire study, the diet interventions were considered too short for any changes in body composition that would provide insight as to whether accommodation may have occurred with the lower protein intake (6). Therefore, we believe that the greater leucine Ox and more negative NET observed with HP is a result of nutrient (specifically, protein) excess. The specific events responsible for these effects, along with the physiological outcomes, are not clear at this time.

It remains to be determined whether similar findings with regard to WBPTO would result if a different amino acid tracer, such as phenylalanine, were employed using these same interventions. Wolfe and colleagues (35) have provided evidence that there can be a disparity in data generated by simultaneous use of isotopic tracers in testing the hypothesis that leucine metabolism would be representative of that of other essential amino acids during a bout of exercise. However, others have found that different tracers provide similar information with regard to protein use (24,31). Short et al. (28) employed both leucine and phenylalanine tracers in examining the effects of endurance exercise on whole-body proteolysis and found a decrease in whole-body proteolysis with age, regardless of tracer. Bennett et al. (2) simultaneously infused leucine and phenylalanine tracers to determine whether independent methodologies would provide consistent information regarding protein and amino acid metabolism. To different degrees, they observed increases in whole-body leucine flux and decreases in whole-body protein breakdown with both isotopes. In total, these studies provide evidence for using isotopes of leucine for modeling WBPTO.

In addition to changes at the level of WBPTO, we observed changes in plasma amino acids across the diets. Plasma BCAA at rest were greater for both the MP and HP diets than on LP, with postexercise values not differing across interventions. There was, however, a significant decrease in plasma leucine from rest to postexercise for MP and HP, with no change noted for LP. Given that leucine Ox remained higher during the postexercise recovery period on HP despite no differences in leucine or total BCAA, immediately after exercise, a greater BCOAD activity in skeletal muscle in response to higher protein intakes may have occurred. These data may suggest an increased reliance on AA for fuel during and after exercise on MP and HP. Whether this is beneficial to the athlete is not clear, but a greater reliance on amino acids for energy may contribute to the sparing of glycogen during exercise.

From a practical perspective, protein intakes above 1.8 g·kg⁻¹·d⁻¹ present the concern for maintaining sufficient dietary carbohydrates for replenishment of muscle glycogen as well as issues regarding proper hydration in competitive endurance athletes. In terms of performance, these issues remain a priority. Although it is generally recommended that endurance athletes consume 6–10 g of carbohydrate per kilogram of body weight (1), on the HP diet, despite an intake of approximately 3500 kcal, subjects were consuming only 5.6 g·kg⁻¹. However, on both the LP and MP diets, subjects were able to meet carbohydrate recommendations, with intakes of 8.3 and 7.5 g·kg⁻¹, respectively. The lower carbohydrate intake on the HP diet may have negatively impacted glycogen stores and subsequently resulted in an increase in the oxidation of amino acids for fuel. An inverse relationship between glycogen stores and BCAA oxidation has been reported previously (34).

In summary, this study placed special emphasis on the control of energy and protein intakes to characterize changes in protein use after an endurance exercise bout in response to varied levels of dietary protein in highly trained runners. Our findings suggest a reduction in protein turnover with protein intakes approximating the current RDA compared with a higher protein intake. It has been suggested that decreased protein turnover resulting from protein intakes below 1.0 g·kg⁻¹·d⁻¹ may compromise “physiologically relevant processes” specific to endurance exercise training (29). Along with observations of a...
negative nitrogen balance in endurance athletes habitually consuming protein at rates of 0.8 g·kg$^{-1}$·d$^{-1}$ (13,26), this level of protein may be inadequate for this population. Maintenance of WBPTO is important for endurance athletes, who, by virtue of their training schedules and competitive season, are essentially recovering daily. Having sufficient amino acids available to support the maintenance and repair of lean body mass is critical, and habitual protein intake is one factor that affects amino acid availability for these processes. However, because we did not observe any difference in Ra, NOLD, or Ox between the MP and HP diets, it seems that intakes greater than 1.8 g·kg$^{-1}$·d$^{-1}$ offer no further advantage to endurance athletes with regard to whole-body protein use.

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REFERENCES


