A preexercise α-lactalbumin-enriched whey protein meal preserves lipid oxidation and decreases adiposity in rats

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THE CHALLENGE OF OVERWEIGHT people involved in a weight-reducing program, as well as of athletes that have to compete in weight categories, is to face up to the energetic demand of exercise training while providing simultaneously an ergogenic aid to exercise performance. For this purpose, rats have to compete in weight categories, is to face up to the energetic demand of exercise training while providing simultaneously an ergogenic aid to exercise performance. For this purpose, rats were trained 5 days per week for 5 wk. The exercises were performed in four different situations: in the postabsorptive state (fasting) or 1 h after the ingestion of a preload that was enriched with glucose (Glc), whole milk proteins (WMP), or α-lactalbumin (CPαL). This kind of meal has the advantage of producing little insulin and thus no impairment of fat mobilization, while having the potential to bring exogenous substrates to active muscles under the form of branched-chain amino acids and glucose produced through the gluconeogenic pathway (2, 5). The production of glucose from exogenous protein should have a sparing effect on endogenous protein catabolism observed in such situations when exercise is performed during an energy deficit (11, 13). In this case, the ability of the protein source to deliver amino acids rapidly into the blood may be critical to increase amino acid availability.

The aim of the present study was to examine whether a preexercise protein-enriched meal would be able to maintain a high rate of lipid oxidation during exercise while providing simultaneously an ergogenic aid to exercise performance. For this purpose, rats were trained 5 days per week for 5 wk. The exercises were performed in four different situations: in the postabsorptive state (fasting) or 1 h after the ingestion of a preload that was enriched with glucose (Glc), whole milk proteins (WMP), or α-lactalbumin (CPαL).
MATERIALS AND METHODS

Animals and diets. All of the procedures used were in strict accordance with the French guide for the care and use of laboratory animals. Twenty-four rats (Harlan France) were housed in individual cages kept in a temperature-controlled room (24 ± 1°C) with an artificial 12:12-h light-dark cycle, lights on at 0600. The experimental diets were prepared under strict laboratory conditions by the “Unité de Préparaison des Aliments Experimentaux” (Institut National de la Recherche Agronomique-UPAE, Jouy-en-Josas, France). Whole milk protein and α-lactalbumin-enriched preparations were supplied by Lactalis (Laval, France). To prevent spillage, the food was made semi-liquid by dilution with water (1:1).

Experimental design. The following experiment, designed for a group of six rats, was repeated four times to get six rats in each group.

During the 1st wk, the six rats had free access to a semi-synthetic powdered food containing 14% of milk protein (P14-diet, Table 1). The average food intake of the first group of rats measured during this 1st wk was taken as the reference 100% ad libitum intake for all of the rats throughout the study.

During the 2nd wk, the rats were restricted to 65% of the reference ad libitum food intake (given at 1700) and started a training program 5 days/wk on a 6-lane treadmill with a 10% slope. The exercise began at 1000 and lasted 2 h. During this 1st wk of training, the exercise speed and duration were progressively increased until the rats had developed the capacity to run the 2nd h at 24 m/min. A maximal O2 consumption (VO₂ max) test at the end of the week showed that the speed of 24 m/min established as the training speed amounted to no more than 70% or less of the VO₂ max of the rats, i.e., that all the rats were able to run above 30 m/min.

During the 3rd wk, the rats were divided into four groups according to the kind of meal they received 1 h before the onset of exercise: the first (fasted) group did not receive any meal, the second (Glc) group received a glucose-enriched meal, the third group (WMP) received a whole milk protein-enriched meal, and the fourth group (CpL) received an α-lactalbumin-enriched meal. These meals amounted to 3 g (20 kJ; see Table 1) and thus reduced food restriction to 85% of the ad libitum food intake. This feeding procedure was maintained until the end of the study.

Table 1. Compositions of the P14 maintenance diet and of the preexercise meals

<table>
<thead>
<tr>
<th></th>
<th>P14 Diet</th>
<th>Glc Meal</th>
<th>WMP Meal</th>
<th>CpL Meal</th>
</tr>
</thead>
<tbody>
<tr>
<td>WMP</td>
<td>140.7</td>
<td>46.7</td>
<td>714.5</td>
<td>46.7</td>
</tr>
<tr>
<td>CpL</td>
<td>0.0</td>
<td>0.0</td>
<td>0.0</td>
<td>667.5</td>
</tr>
<tr>
<td>Sucrose</td>
<td>100.5</td>
<td>33.4</td>
<td>33.4</td>
<td>33.4</td>
</tr>
<tr>
<td>Cornstarch</td>
<td>625.1</td>
<td>207.7</td>
<td>207.7</td>
<td>207.7</td>
</tr>
<tr>
<td>Glucose</td>
<td>0.0</td>
<td>667.8</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Vitamin mix</td>
<td>10.1</td>
<td>3.3</td>
<td>3.3</td>
<td>3.3</td>
</tr>
<tr>
<td>Mineral mix</td>
<td>10.1</td>
<td>3.3</td>
<td>3.3</td>
<td>3.3</td>
</tr>
<tr>
<td>Soybean oil</td>
<td>40.2</td>
<td>13.4</td>
<td>13.4</td>
<td>13.4</td>
</tr>
<tr>
<td>Cellulose</td>
<td>50.3</td>
<td>16.7</td>
<td>16.7</td>
<td>16.7</td>
</tr>
<tr>
<td>Choline</td>
<td>23.1</td>
<td>7.7</td>
<td>7.7</td>
<td>7.7</td>
</tr>
</tbody>
</table>

Values are in g/kg. In the α-lactalbumin-enriched whey protein concentrate (CpL), α-lactalbumin obtained by physical separation amounted to ≥40% of total protein. Preexercise meals were done with 1 g of the P14 diet plus 2 g of glucose (Glc meal), whole milk protein (WMP meal), or CpL (CpL meal). No additional minerals, vitamins, or choline were added.

At the end of the 3rd wk of training, all of the rats were implanted with a chronic jugular catheter, which ended in the vena cava according to an adaptation of the method of Burvin et al. (3).

During the 4th and 5th wk of training, the daily energy expenditure of the rats was measured by indirect calorimetry after they were put in turn into a metabolic cage linked to an open-circuit flow-through calorimetric device. Briefly, fresh air taken from the compressed-air circuit of the building was pulled by a vacuum pump through the metabolic cage (1.3 l/min) and then pushed to air-dryers (anhydrous CaCl₂) and a mass flowmeter (El Flow flowmeter/controller from Bronkhorst Hi-Tec, Montigny-les-Cormeilles, France) before being analyzed by O2 (Oxygr 610 from Maihak, Hamburg, Germany) and CO2 (Rubis 3000 from Cosma, Igny, France) gas analyzers. The gas analyzers were linked to a computer-controlled system of data acquisition, which collected and stored data for subsequent analysis. Spontaneous activity in the metabolic cage was recorded from three piezo-electric force-transducers (sensitivity 1 g, Kistler, Les Ulis, France) fixed under the floor of the cage. The simultaneous measurement of respiratory exchange and spontaneous activity of the rat was processed according to the method of Kalman filtering to compute separately resting and activity-related oxygen consumption (QO₂) and carbon dioxide release (QCO₂) (7, 8). On this day, the exercise was performed in a metabolic treadmill so that respiratory exchanges were recorded also during exercise.

Seven 500-μl blood samples spread over 4 days (maximum 2 samples per rat and per day) were taken from each rat, and no samples were taken during the calorimetric studies. The samples were taken 10 min before the preexercise meal, 10 min before the onset of exercise, after 30 and 90 min of exercise, and 30, 90, and 240 min after the end of exercise.

At the end of the study, the rats were killed for measurement of whole body composition and liver and muscle glycogen content.

Experimental procedure in the metabolic chamber and treadmill. The rats were housed in the metabolic chamber at 1700. A polyethylene tube supported by an articulated arm was inserted through the vena cava according to an adaptation of the method of Bronkhorst Hi-Tec, Montigny-les-Cormeilles, France) before being analyzed by O2 (Oxygr 610 from Maihak, Hamburg, Germany) and CO2 (Rubis 3000 from Cosma, Igny, France) gas analyzers. The gas analyzers were linked to a computer-controlled system of data acquisition, which collected and stored data for subsequent analysis. Spontaneous activity in the metabolic cage was recorded from three piezo-electric force transducers (sensitivity 1 g, Kistler, Les Ulis, France) fixed under the floor of the cage. The simultaneous measurement of respiratory exchange and spontaneous activity of the rat was processed according to the method of Kalman filtering to compute separately resting and activity-related oxygen consumption (QO₂) and carbon dioxide release (QCO₂) (7, 8). On this day, the exercise was performed in a metabolic treadmill so that respiratory exchanges were recorded also during exercise.

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At the end of the study, the rats were killed for measurement of whole body composition and liver and muscle glycogen content.

Blood sample procedures. Twenty microliters of an antiprotease solution composed of EDTA and aprotinin (Trasylol, 10,000 units; Bayer, Leverkusen, Germany) were added to each 500-μl blood sample. The plasma was then separated by centrifugation (15 min at 3,000 g), frozen, and stored at −80°C. After each blood sample, the catheter was washed with citrated physiological serum [Citric Acid anhydrous + Citric Acid Trisodium Salt Dihydrate Sigma Ultra (Sigma
Diagnostics, St. Quentin Fallavier, France) + NaCl (9.0 g/l solution; B. Braun Medical, Boulogne, France).

Assays. Urea in urine and plasma was assayed with the Bun (Endpoint) Urea Nitrogen Kit from Sigma Diagnostics. Glucose and lactate were assayed with the Glucose RTU and Lactate PAP kits (bioMérieux, Lyon, France). Triacylglycerol and glycerol were assayed with the triglyceride (GPO-Trinder) kit from Sigma Diagnostics. Free fatty acids were assayed using the NEFA C kit from Wako (Wako Chemicals, Richmond, VA).

Tissue samples and glycogen dosage. Rats were deeply anesthetized with an overdose of anesthetic (pentobarbital sodium, 48 mg/kg) and exsanguinated by section of the abdominal aorta and vena cava. Liver and hindlimb muscles were immediately dissected out, frozen in liquid nitrogen, and stored at −80°C for further glycogen dosage by the method described by Lo et al. (16). Then, the main organs and the remainder of the body, i.e., muscle mass plus skeleton (excluding tail and feet), was weighed and classified as “carrion.” Lean body mass (LBM) in this study was taken as total body mass minus adipose tissues (10).

Substrate oxidation. Protein oxidation (Pox) between 1700 and 0900 the next day was computed from the amount of nitrogen released in urine urea. During the periexercise period, i.e., 0900–1600, changes in Pox were computed by using simultaneously urinary urea production and changes in plasma urea concentrations. Changes in whole body urea were extrapolated from changes in plasma urea with the assumptions that urea diffuses freely in the body water compartment and that body water accounts for 66.8% of body weight (18). Urea production was thus computed according to the following formula

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UP = [d(\text{PU}) \cdot \text{TBW}] + [(\text{UU}) \cdot \text{UV}]
\]

With UP as urea production (mg), d(UP) as change in plasma urea (g/l) and used when available, TBW as total body water (ml), UU as urinary urea (g/l), and UV as urine volume (ml). Pox (N·6.25), measured at various intervals following this method, was used to establish a kinetic of protein oxidation by polynomial regression. The Pox values derived from the polynomial functions were used to compute the nonprotein values of \( \text{Q}_{\text{O}2} \) and \( \text{Q}_{\text{CO}2} \) and the corresponding rates of glucose (Gox) and lipid oxidation (Lox) during the calorimetric studies. To normalize the presentation of the data in terms of energy produced, the rates of Pox, Gox, and Lox have been converted from milligrams per minute to watts, with the assumption of 16.74 kJ/g for glucose, 18.84 kJ/g for protein, and 37.67 kJ/g for lipid.

Statistical analysis. All values are expressed as means ± SE. ANOVA and repeated-measures ANOVA were used to assess differences between groups and were completed by a post hoc Scheffé test when appropriate. A probability of \( P < 0.05 \) was chosen as the criterion for acceptance of a statistical significance. The statistical tests were performed with the SAS program (SAS Institute, Cary, NC).

RESULTS

Body weight and body composition. Body weight increased more in the CPαL, WMP, and Glc groups than
in the fasted group (Fig. 1). Analysis of body composition showed that LBM was significantly smaller in the fasted rats than in the three other groups (Table 2). All of the rats showed a reduced adiposity (5.7–7.8% FAT) compared with sedentary ad libitum P14-fed Wistar rats of similar body weight, in which fat usually amounts to 20% of TBW (10). This lower adiposity was mainly the result of food restriction, because in a preliminary study done on 24 rats put on the same dietary schedule but without exercise, we observed that %FAT was reduced nearly as much as in the present study and was similar between the groups (8.0 ± 0.7% for fasted, 8.3 ± 1.1% for Glc, 9.2 ± 0.7% for WMP, and 8.7 ± 0.8% for CPαL rats). In contrast, in the rats of the present study, fat mass adjusted for body weight (adiposity index, %FAT) was smaller in the fasted and CPαL rats than in the WMP rats. In the exercised rats, carcass weight adjusted for body weight (%CARC) was larger in the fasted rats than in the WMP and Glc rats and larger in the CPαL rats than in the WMP rats. The size of the vastus lateralis (part of the quadriceps femoris), which works as an extensor of the shank and was strongly requested during the run, was the largest in the CPαL rats (data not shown, P < 0.05). No changes were observed on the other components of body weight.

Metabolic rate and respiratory quotient. Resting metabolic rate (RMR) measured at 0800, i.e., 15 h after the meal was given at 1700, has been taken as the basal metabolic rate (BMR) of the rats. BMR was higher in the CPαL group (2.44 ± 0.14 W) than in the other groups (1.82 ± 0.13 W for fasted, 1.95 ± 0.14 W for WMP, and 2.1 ± 0.09 W for Glc rats; P < 0.05). Ingestion of the preexercise meals promoted an increase in BMR during the hour preceding the exercise because of the thermogenic effect of the meal (Fig. 2A). This increase was not different among the three meals. On the other hand, glucose ingestion promoted a marked increase in respiratory quotient (RQ) (Fig. 2B). After the exercise, all of the rats recovered rapidly their preexercise BMR; i.e., no excess postexercise QO2 was observed.

Rates of oxidation of protein, glucose, and lipid. Before introduction of the preexercise meal, Pox, Gox, and Lox were low and similar in the four groups (Fig. 3). As illustrated by the RQ increase (Fig. 2B), the preexercise glucose-enriched meal induced an increase in Gox, a decrease in Lox, and a slight decrease in Pox. The WMP and CPαL preexercise meal increased Pox to ~0.35 W. During the exercise bout, Pox remained elevated in the WMP group but did not increase further despite the increased metabolic demand. In contrast, Pox continued to increase during exercise in the CPαL group and reached 0.65 W at the end of exercise, i.e., two times more than in the WMP group and four times more than in the Glc and fasted groups. A protein-sparing effect was observed in the Glc group in which

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**Fig. 2.** Metabolic rate (A) and respiratory quotient (B) before, during, and after exercise. White arrow, preexercise meal time; dashed lines, exercise. PRE, preexercise; EX, exercise; RECOV, recovery. *P < 0.05 vs. fasted; †P < 0.05 vs. Glc.
Pox was the lowest. The relative Pox participation to energy expenditure during exercise was significantly higher in the CPαL group (8.7%) than in all of the other groups (P < 0.05), and higher in the WMP (6.4%) group than in the fasted (3.6%) or glucose (2.0%)-fed groups (P < 0.05).

During exercise, the participation of Lox to whole body energy expenditure was as large in the WMP- and CPαL-fed rats as in the fasted ones, whereas the glucose-fed rats relied significantly more on glucose oxidation and less on lipid oxidation, not only during exercise but also during recovery. Interestingly, only minor and transient differences in Gox and Lox could be observed among the fasted, WMP, and CPαL groups throughout the exercise and recovery periods, indicating that the ingestion of the protein meals did not significantly affect the utilization of glucose and lipids. During the 2-h recovery period, however, the participation of Pox to whole body energy peaked up to 23.3% of whole body energy expenditure in the CPαL group and to only 15.5% in the WMP group (P < 0.05). In addition, the maximum of this rise occurred early after exercise (30 min) in the CPαL group and much later (150 min) in the WMP group.

Fig. 3. Changes in protein (Pox, A), glucose (Gox, B), and lipid (Lox, C) oxidation. †P < 0.05 vs. Glc.
Plasma substrates. Ingestion of the preexercise glucose-enriched meal promoted a rapid increase in plasma glucose concentration that was apparent after 30 min of exercise ([glucose] at t 90 min > [glucose] at t 50 min, P < 0.05) and remained above plasma glucose values observed in the fasted, WMP, and CPαL groups throughout the exercise period (Fig. 4). However, a rather large variability in the individual responses prevented this difference from reaching significance. In contrast, plasma glucose was the same in the fasted, WMP, and CPαL groups throughout. In agreement with the changes observed in plasma glucose, plasma lactate remained low in the fasted WMP and CPαL groups and was increased in the Glc group.

No significant differences were observed in plasma triacylglycerol concentrations among the four groups (Fig. 5). A significant decrease in plasma glycerol concentration was observed between the ingestion of the meal and the onset of exercise in the Glc group, indicating that the glucose-enriched meal promoted a rapid inhibition of lipolysis. Such a phenomenon was not observed in the groups fed the WMP or CPαL meals. The onset of exercise induced an increase in plasma glycerol in all groups. After the completion of the exercise, plasma glycerol tended to be higher in the fasted group than in the three other groups, as demonstrated by a significantly higher value in this group at t = 300 min. Plasma free fatty acid concentrations were larger in the fasted, WMP, and CPαL groups than in the Glc group during exercise and larger in the fasted group than in the Glc group after exercise.

Hepatic and hindlimb muscle glycogen content. Before the exercise, glycogen content was higher in the liver of both the fasted and Glc rats than in that of the WMP and CPαL rats, and higher in the red vastus lateralis of the glucose-fed than of the fasted and CPαL-fed rats (Table 3). Liver glycogen was more mobilized by exercise in the fasted and Glc groups than in the WMP and CPαL groups. CPαL rats, in particular, used very low amounts of liver glycogen: 7, 4.4, and 2.6 times less than the fasted, Glc, and WMP rats, respectively. Fewer differences were observed according to the utilization of muscle glycogen; however, overall, the WMP-fed rats used less muscle glycogen than the rats of the other groups.

DISCUSSION

In this study, we compared the effects of three different meals on the rates of oxidation of glucose, lipid, and protein and on the long-term consequences on body weight and body composition. The main observations are that 1) the preexercise protein meals maintained a high rate of lipid oxidation during exercise equivalent to fasted conditions; 2) the preexercise CPαL meal
appeared more efficient to sustain LBM at the expense of fat mass.

Ingestion of the Glc meal 1 h before exercise increased Gox and decreased Pox and Lox. This phenomenon was induced by the fact that, immediately after the meal, lipolysis was reduced, leading to a rapid increase in the participation of Gox in basal metabolism and a subsequent decrease in fat oxidation (12). The influence of the preexercise rate of Gox on the subsequent rate of Gox during exercise has already been suggested (9) and is probably part of the mechanism that links the composition of the usual diet with fat mass.

Another potential mechanism for the enhanced Gox in the Glc-fed rats is the observation that, in these rats, liver as well as muscle glycogen was higher than in the other groups, a phenomenon that has also been shown to increase Gox during exercise (4, 15). These rats, in contrast to the fasted ones, which had some difficulties in completing the 2 h of exercise (this is also revealed in Fig. 2 by the fact that their metabolic rate tended to decrease before the end of the exercise), also completed their daily exercise program without revealing any sign of tiredness, showing that the Glc meal improved their endurance (6). The negative effect, however, was that part of the extra energy brought about by the Glc meal was not used but was stored in adipocytes. Therefore, when the main goal of exercise training is to reduce body adiposity, the ingestion of carbohydrates before exercise may be counterproductive.

In contrast to Glc, ingestion of the protein-rich meals before exercise did not significantly affect Gox and Lox compared with the fasted condition. In addition, Pox was increased and the reliance on endogenous glycogen reserves reduced (20). Comparison of the metabolic responses of the protein-fed and Glc-fed rats clearly indicates that the primum movens of this difference was initiated before the onset of exercise by the fact that neither of the protein meals reduced preexercise lipid oxidation, and both increased amino acid oxidation. Two observations suggest that Pox was stimulated specifically by the amino acids brought by the meal: first, none of the fasted and Glc-fed rats increased their rate of amino acid oxidation during exercise; second, Pox was already increased in the CPaL and WMP rats before the onset of exercise. These rats also ran with more ease than the fasted ones, and the CPaL-fed ones in particular exhibited a certain will to run, as testified by their high and sustained metabolic rates during the exercise. This last observation argues in favor of the fact that both protein meals, but particularly the CPaL meal, brought exogenous amino acids that could be used during exercise to face the energy demand of the run.

Interestingly, CPaL-fed rats gained more weight but fixed most of this extra weight in their lean tissues, whereas Glc-fed and WMP-fed rats gained mainly fat. This result suggests that increasing the rate of lipid oxidation during and after exercise is not, per se, a sufficient mechanism to favor the selective mobilization of adipose reserves in the long term. Clearly, compensatory mechanisms must have developed away from the exercise to induce tiny modifications in the fate of the nutrients ingested with the night meal that allowed the WMP-fed rats to recover during the night the excess lipids used during exercise. The major difference in the metabolic response to CPaL and WMP was the time course of utilization of the amino acids brought by the meal. The higher solubility of the CPaL meal allowed Pox to increase steadily during exercise, whereas Pox leveled in the WMP-fed rats. This observation indicates that the delivery of amino acids by the gut was less reduced in the CPaL-fed than in the WMP-fed rats. As a result, the CPaL-fed rats ended the

| Table 3. Glycogen content of liver and hindlimb muscles before and after the run |
|------------------------|-----------------|-----------------|-----------------|
|                        | Fasted          | Glc             | WMP             | CPaL             |
| Liver                  |                 |                 |                 |                 |
| Before                 | 50.9 ± 9.7(a)   | 58.6 ± 11.1(a)  | 20.5 ± 13.93(ab)| 11.91 ± 7.28(b) |
| After                  | 11.8 ± 8.8*(ab)| 34.0 ± 5.77(a)  | 6.18 ± 1.17(b)  | 6.34 ± 5.11(b)  |
| Used                   | 24.6            | 14.32           | 5.57            |                 |
| Gastrocnemius          |                 |                 |                 |                 |
| Before                 | 6.76 ± 0.58(a)  | 9.03 ± 1.04(a)  | 7.89 ± 3.94(a)  | 7.72 ± 1.37(a)  |
| After                  | 3.65 ± 1.24(a)  | 4.75 ± 0.96*(a)| 5.49 ± 0.55(a)  | 2.83 ± 1.38*(a) |
| Used                   | 4.28            | 2.40            | 4.89            |                 |
| Soleus                 |                 |                 |                 |                 |
| Before                 | 5.42 ± 1.99(a)  | 6.25 ± 1.79(a)  | 5.85 ± 2.57(a)  | 5.32 ± 0.24(a)  |
| After                  | 1.88 ± 0.94(ab) | 3.52 ± 0.74(a)  | 4.20 ± 0.66(a)  | 1.38 ± 0.35*(b) |
| Used                   | 3.54            | 1.65            | 3.94            |                 |
| White vastus lateralis|                 |                 |                 |                 |
| Before                 | 3.38 ± 1.15(a)  | 1.25 ± 0.13(a)  | 1.71 ± 0.69(a)  | 2.46 ± 1.63(a)  |
| After                  | 0.60 ± 0.11(a)  | 1.23 ± 0.32(a)  | 1.25 ± 0.35(a)  | 0.81 ± 0.19(a)  |
| Used                   | 2.78            | 0.46            | 1.65            |                 |
| Red vastus lateralis  |                 |                 |                 |                 |
| Before                 | 2.22 ± 0.61(a)  | 4.63 ± 0.79(b)  | 2.23 ± 1.54(ab) | 1.45 ± 0.12(a)  |
| After                  | 1.06 ± 0.11(a)  | 2.72 ± 0.81(a)  | 1.88 ± 0.35(a)  | 0.99 ± 0.01*(a) |
| Used                   | 1.16            | 0.35            | 0.46            |                 |

Results are means ± SE expressed in mg of glycogen/g of tissue. "Used" gives the net utilization of glycogen stores (mg/g) after 2 h of exercise. Different letters in parentheses indicate a significant difference (P < 0.05); * P < 0.05, before vs. after.
exercise with a rate of Pox about two times higher than the rate in the WMP-fed ones. In addition, this high Pox rate continued after the completion of exercise, whereas by only 2 h after the termination of exercise, Pox increased again in the WMP-fed rats. All together, these data indicate that amino acid availability was larger during and early after exercise in the CPoL-fed rats. It is thus tempting to hypothesize that this kinetic played in favor of a better fixation of the exogenous amino acids in muscles and LBM in the CPoL-fed rats, in particular because more amino acids were available immediately after exercise, i.e., at a critical time to maximally enhance the processes of restoration of proteins degraded during exercise (17, 21, 22).

In conclusion, this study revealed that ingestion of a protein meal before exercise improved lipid oxidation but that this phenomenon was not as sufficient to reduce adiposity in the long term as it was in the CPoL rats. Other mechanisms must thus be looked at to understand the specific effect of the CPoL protein in this study. The data suggest that the kinetic of delivery of dietary amino acids by the digestive tract played a critical role, but the role played by the amino acid composition of CPoL also deserves further investigation.

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REFERENCES