No effects of oral ribose supplementation on repeated maximal exercise and de novo ATP resynthesis

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Op 't Eijnde, B., M. Van Leemputte, F. Brouns, G. J. Van der Vusse, V. Labarque, M. Ramaekers, R. Van Schuylenberg, P. Verbessem, H. Wijnen, and P. Hespel. No effects of oral ribose supplementation on repeated maximal exercise and de novo ATP resynthesis. J Appl Physiol 91: 2275–2281, 2001.—A double-blind randomized study was performed to evaluate the effect of oral ribose supplementation on repeated maximal exercise and ATP recovery after intermittent maximal muscle contractions. Muscle power output was measured during dynamic knee extensions with the right leg on an isokinetic dynamometer before (pretest) and after (posttest) a 6-day training period in conjunction with ribose (R, 4 doses/day at 4 g/dose, n = 10) or placebo (P, n = 9) intake. The exercise protocol consisted of two bouts (A and B) of maximal contractions, separated by 15 s of rest. Bouts A and B consisted of 15 series of 12 contractions each, separated by a 60-min rest period. During the training period, the subjects performed the same exercise protocol twice per day, with 3–5 h of rest between exercise sessions. Blood samples were collected before and after bouts A and B and 24 h after bout B. Knee-extension power outputs were ~10% higher in the posttest than in the pretest but were similar between P and R for all contraction series. The exercise increased blood lactate and plasma ammonia concentrations (P < 0.05), with no significant differences between P and R at any time. After a 6-wk washout period, in a subgroup of subjects (n = 8), needle-biopsy samples were taken from the vastus lateralis before, immediately after, and 24 h after an exercise bout similar to the pretest. ATP and total adenine nucleotide content were decreased by ~25 and 20% immediately after and 24 h after exercise in P and R. Oral ribose supplementation with 4-g doses four times a day does not beneficially impact on postexercise muscle ATP recovery and maximal intermittent exercise performance.

ergogenics; adenine nucleotides; ATP; ammonia; purine salvage

PHOSPHOCREATINE BREAKDOWN by the action of creatine kinase (CK) is the primary pathway of ATP production during short periods of strenuous exercise. However, as muscle phosphocreatine concentration rapidly drops, the rate of ADP rephosphorylation through the CK reaction is impaired, which causes intracellular ATP content to decrease and ADP content to increase (2, 4, 10, 14, 15, 17, 20, 28). A fraction of this ADP is eventually degraded to IMP by the successive actions of adenylate kinase and AMP deaminase. A small proportion of the IMP so formed is converted to inosine and further to hypoxanthine, which implies a reduction of the muscle adenine nucleotide pool (8–10, 21, 24, 28). After a single bout of maximal exercise, the nucleotide pool is rapidly replenished via the purine salvage metabolic pathway in conjunction with the purine nucleotide cycle (1, 5, 12, 18, 28). This involves the conversion of hypoxanthine to IMP and further to AMP, eventually resulting in ATP resynthesis. However, during training involving many repeated bouts of high-intensity exercise, purine salvage and nucleotide cycling may fail to compensate for the massive rate of nucleotide degradation. Thus a small fraction of the nucleosides and bases are lost from the muscle cells and need to be replenished by de novo nucleotide synthesis. However, the latter process is slow, which explains the decrease in muscle ATP content for several days after exercise (10, 20). The formation of ribose 5-phosphate through the pentose phosphate pathway sets the upper pace of adenine nucleotide synthesis. There is evidence from animal studies that this limitation can be overcome by increasing ribose delivery to the muscle. In the perfused rat hindquarter, supraphysiological concentrations of ribose (5 mM) enhanced the formation of phosphoribosyl pyrophosphate, a precursor of IMP production and, thereby, ATP synthesis (27). Furthermore, increasing ribose by intravenous ribose infusion was found to markedly enhance the recovery of myocardial ATP content as well as the functional capacity.
in various animal models of myocardial ischemia (11, 13, 16, 22, 23, 26, 29–31).

The above-mentioned findings have prompted interest in the potential of oral ribose supplements to boost muscular performance in sports. Moreover, ribose supplements are being advertised to be “ergogenic” in athletic populations involved in high-intensity exercise training. Ribose is rapidly absorbed from the intestinal tract and is well tolerated, even at very high dosages (>100 g/day) (7) or during exercise (6). After absorption, ribose is rapidly and extensively metabolized, the principal fate being conversion in the liver to glucose via the pentose phosphate pathway (19). Furthermore, ribose can also be transported to muscle cells to feed the nucleotide synthesis pathway. However, there is no evidence that oral ribose intake can beneficially impact adenine nucleotide synthesis in skeletal musculature and/or enhance exercise performance in humans. Therefore, the goal of the present study was to evaluate the effect of a common oral ribose supplementation regimen on the restoration of muscle ATP after strenuous exercise, on the one hand, and on muscle force and power output during a training period involving repeated bouts of maximal exercise, on the other hand.

METHODS

Subjects. Twenty healthy male physical education students (20.6 ± 0.4 yr of age, 75.2 ± 1.6 kg body wt) gave their informed written consent to participate in the study. None of the volunteers had a specific background of sprint and/or resistance training, but all were regular participants in various sports activities involving sprint and resistance exercise. Exclusion criteria on admission were 1) prior ribose supplementation or intake of other nutritional supplements or medication and 2) any medical condition that might contraindicate heavy-resistance exercise with the right leg. The subjects were asked to avoid changes in their diet and level of physical activity during the period of the study. One subject withdrew because of a back injury, which prevented further participation in the exercise training sessions.

Study protocol. The study consisted of two phases (I and II), with a 6-wk washout period in between phases. The study protocol was approved by the local ethics committee.

In phase I, a double-blind placebo-controlled study was performed over a period of 12 days. Days 0–7 were exercise days, which were preceded by 4 days of placebo/ribose supplementation (days −4 to −1). One week before day −4, the subjects reported to the laboratory for familiarization with the exercise test procedures. On the basis of the torque measurements in this preliminary session, the subjects were assigned to two experimental groups with similar distributions for maximal isometric torque and percent muscle fatigue during the pretest of maximal isometric exercise. Subjects were assigned to the ribose-treated (R) or placebo (P) group to obtain two groups with similar distributions for maximal isometric torque, and percent muscle fatigue was measured during the pretest of phase I. On the morning of day 0, the subjects reported to the laboratory after an overnight fast and received a standardized breakfast. After 90 min, a biopsy was taken from the left vastus lateralis muscle using a Bergström needle with suction applied. To obtain the biopsy, an incision was made through the skin and muscle fascia under local anesthesia (2–3 ml of 1% lidocaine). Immediately thereafter, the subjects performed the exercise test similar to phase I. However, the maximal isometric contractions before bout A and after bout B in phase I were omitted. At 10 min before the start of bout B, an incision was made through the skin and fascia of the right vastus lateralis muscle. This incision was used to obtain a biopsy within 2–3 min after bout B. After 24 h of recovery (day 1), the subjects returned to the laboratory for a muscle biopsy, which was taken ~3 cm proximal to the postexercise biopsy site on day 2.

Diet and supplements. On the evening and morning before each exercise test, the subjects received a standardized dinner [855 kcal: 47% carbohydrate (CHO), 25% fat, 28% protein] and breakfast (320 kcal: 65% CHO, 15% fat, 20% protein), respectively. The ribose supplements (4 g) were mixed with maltodextrin (4.5 g), aspartame (250 mg), and artificial lemon flavor (23 mg) to be similar in taste and appearance to the placebo supplements (8.5 g maltodextrin, 250 mg aspartame, and 3 mg lemon flavor). At the end of the study, the subjects were asked whether they had any notion of the treatment they had received. Irrespective of the supplements received, all were unsure.

During phase I, the subjects from the R group (n = 10) received four 4-g oral ribose supplements each day (16 g/day); the other subjects (P group) received placebo supplements. On the resting days (day −4 to day −1), the subjects were instructed to ingest the supplements in 150 ml of water immediately before breakfast, lunch, and dinner and 1 h before bedtime. During the pretest (day 0) and the posttest (day 7), the subjects ingested the supplements 10 min before and at the end of exercise bout A and immediately after and 2 h after exercise bout B. During the training days (days 1–6), the subjects ingested the supplements immediately before and after each of the two daily training sessions.

During phase II, on day −1, the subjects received four 4-g doses of ribose or placebo supplements similar to phase I, and then they rested in a semisupine position until the start of bout B. Capillary blood samples (heparinized glass capillaries) from a hyperemic earlobe (Forapin) and venous blood samples from an antecubital vein (Vacutainer) were taken immediately before and after bouts A and B and 24 h after bout B. From day 1 to day 6, the subjects participated in a standardized training program. On each day, they reported twice to the laboratory, with 4–6 h between visits, and performed a training session on the isokinetic dynamometer, which was similar to exercise bout A (see above). However, on day 6, the subjects performed only a single training session. On the next day (day 7) and ≥24 h after the last training session on day 6, they returned to the laboratory to perform the posttest, which was identical to the pretest.

The goal of phase II was to evaluate the impact of the intermittent exercise protocol on muscle adenine nucleotides. Sixteen of the 20 subjects who participated in phase I (20.3 ± 0.4 yr of age, 75.3 ± 1.9 kg body wt) agreed to participate in phase II. After a 6-wk washout period following phase I, a double-blind placebo-controlled study was performed over 3 days. Day 1 was a rest day, day 0 was an exercise day, and day 1 was a recovery day. Subjects were assigned to the ribose-treated (R) or placebo (P) group to obtain two groups with similar distributions for maximal isometric torque, and percent muscle fatigue was measured during the pretest of phase I. On the morning of day 0, the subjects reported to the laboratory after an overnight fast and received a standardized breakfast. After 90 min, a biopsy was taken from the left vastus lateralis muscle using a Bergström needle with suction applied. To obtain the biopsy, an incision was made through the skin and muscle fascia under local anesthesia (2–3 ml of 1% lidocaine). Immediately thereafter, the subjects performed the exercise test similar to phase I. However, the maximal isometric contractions before bout A and after bout B in phase I were omitted. At 10 min before the start of bout B, an incision was made through the skin and fascia of the right vastus lateralis muscle. This incision was used to obtain a biopsy within 2–3 min after bout B. After 24 h of recovery (day 1), the subjects returned to the laboratory for a muscle biopsy, which was taken ~3 cm proximal to the postexercise biopsy site on day 2.

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in the evening they were served a standardized dinner (see above). On day 0, they arrived at the laboratory after an overnight fast, and they received a standardized breakfast 90 min before the exercise test similar to phase I. They ingested the placebo/ribose supplements before and after exercise bout A and 2 and 4 h after exercise bout B. They also received a standardized lunch (400 kcal: 60% CHO, 16% fat, 24% protein) and dinner. On day 1, they returned to the laboratory for the 24-h recovery biopsy 2 h after a standardized breakfast and a final 4-g ribose/placebo dose.

Knee-extension torque measurements. The subjects were seated on an isokinetic dynamometer in a backward (30°)-inclined chair (hip angle 90°) with the right knee supported at the level of the popliteal cavity. The dynamometer was instrumented with a torque transducer (Lebow 1605, 0.05% accuracy level) and connected with a rigid lever arm, which was positioned lateral to the lower leg, such that its axis was aligned with the knee joint axis. The leg was strapped to the lever arm of the system above the ankle. The positions of the knee and ankle fixations and of the axis of the measuring device were noted for each individual and were set to be identical for the pretest and the posttest and for phases I and II of the study. Subjects were asked to generate, from full relaxation and as fast as possible, maximal knee extensions (isometric or concentric) and thereafter relax as fast as possible. After a standardized 5-min warm-up, the test started with three maximal isometric contractions (3 s) at a knee angle of 120° (180° being full extension), separated by 1 min of rest. Immediately thereafter, they performed two intermittent exercise bouts (A and B), which each consisted of 15 series of 12 maximal knee extensions. The knee extensions were performed at a constant velocity of 60°/s, starting from 90° knee flexion to 150° knee extension. After each contraction, the leg was returned (240°/s) passively to the starting position, from which the next contraction was immediately initiated. The contraction series (15 s) were separated by 15-s rest intervals. A 60-min rest pause separated bouts A and B. Immediately afterbout B, the subjects repeated the isometric contractions similar to those performed in bout A.

Fig. 1. Effect of ribose intake on power output during maximal intermittent muscle contractions. Values are means ± SE of 9 (placebo, □) and 10 (ribose, ■) observations. During the pretest (A) and posttest (B), subjects performed 2 bouts (bouts A and B) of maximal intermittent muscle contractions. Each bout consisted of 15 series of 12 maximal knee extensions on an isokinetic dynamometer, with 15 s of rest between each series. A 1-h rest interval separated bouts A and B. Each data point represents power output for the former or the latter 3 contractions of each series. A 6-day training period separated the pretest and posttest. Mean power was calculated for the 15 contraction series of bouts A and B.

Fig. 2. Effect of ribose intake on mean power output during the intermittent exercise test. Values are means ± SE of 9 (placebo, open bars) and 10 (ribose, solid bars) observations. Each bout consisted of 15 series of 12 maximal knee extensions on an isokinetic dynamometer, with 15 s of rest between each series. A 1-h rest interval separated bouts A and B. A 6-day training period separated the pretest and posttest.
Table 1. Muscle adenine nucleotides during and after intermittent muscle contractions

<table>
<thead>
<tr>
<th></th>
<th>Preexercise</th>
<th>Postexercise</th>
<th>24 h Recovery</th>
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<tbody>
<tr>
<td></td>
<td>Placebo</td>
<td>Ribose</td>
<td>Placebo</td>
</tr>
<tr>
<td>ATP, μmol/g dry wt</td>
<td>16.0 ± 0.6</td>
<td>17.1 ± 0.5</td>
<td>12.7 ± 1.1*</td>
</tr>
<tr>
<td>ADP, μmol/g dry wt</td>
<td>1.92 ± 0.12</td>
<td>2.23 ± 0.20</td>
<td>1.78 ± 0.18</td>
</tr>
<tr>
<td>AMP, μmol/g dry wt</td>
<td>36 ± 2</td>
<td>44 ± 5</td>
<td>38 ± 6</td>
</tr>
<tr>
<td>IMP, μmol/g dry wt</td>
<td>0.19 ± 0.05</td>
<td>0.45 ± 0.15</td>
<td>0.32 ± 0.12</td>
</tr>
<tr>
<td>TAN, μmol/g dry wt</td>
<td>18.2 ± 0.7</td>
<td>19.8 ± 0.5</td>
<td>14.9 ± 1.3†</td>
</tr>
<tr>
<td>NAD, μmol/g dry wt</td>
<td>1.04 ± 0.06</td>
<td>1.16 ± 0.11</td>
<td>1.02 ± 0.08</td>
</tr>
</tbody>
</table>

Values are means ± SE of 8 observations. TAN, total adenine nucleotides. Subjects performed 2 bouts of maximal intermittent exercise with 1 h of rest between bouts. Each bout consisted of 15 series of 12 maximal knee extensions with the right leg on an isokinetic dynamometer, with 15 s of rest between each series. Adenine nucleotides were measured in needle-biopsy samples from the vastus lateralis muscle before exercise and 2–3 min and 24 h after the last exercise bout. Significantly different from corresponding baseline value: *P < 0.05; †P < 0.10.
torque was ~25% lower after than before the pretest, with no differences between P and R. In the posttest, isometric torques were higher than in the pretest, but again they were similar between groups. Muscle power outputs were also measured during every training session from day 1 to day 6. Power outputs progressively increased throughout the week but were similar between P and R at all times.

In phase II, the subjects performed exercise bouts A and B on one occasion. Mean power outputs in P were 125.1 ± 6.0 and 124.3 ± 6.1 W for bouts A and B, respectively, and were similar in R (113.8 ± 9.2 and 110.5 ± 9.3 W for bouts A and B, respectively).

**Muscle adenine nucleotides.** Adenine nucleotides were measured only in phase II of the study, before and after the maximal intermittent exercise test. ATP, ADP, AMP, and IMP concentrations before exercise were similar between P and R (Table 1). The exercise decreased muscle ATP content by 20–25% in both groups (P < 0.05). After 24 h of recovery, ATP content had slightly increased (not significant) in P and R but was still significantly lower (P < 0.05) than the corresponding baseline values. Because muscle ADP, AMP, and IMP contents were not significantly increased 2–3 min after exercise, muscle TAN content paralleled the exercise-induced changes in ATP. Thus TAN was decreased (P < 0.05) by 20–25% at 2–3 min after exercise and 15–20% after 24 h of recovery in either group (P < 0.05).

**Blood and plasma metabolites.** The intermittent knee-extension exercise bouts each increased (P < 0.05) blood lactate from ~0.8 mmol/l before to ~4–5 mmol/l immediately after exercise (Table 2). However, at any time, blood lactate concentrations were similar between groups. By analogy, exercise bouts A and B each increased (P < 0.05) plasma ammonia concentration to the same degree in P and R (Table 2). Blood lactate and plasma ammonia also were similar for the pretest and posttest. Plasma CK was increased in P and R 24 h after the pretest (P < 0.05) but not after the posttest (Table 3). Neither the pretest nor the posttest significantly changed plasma uric acid concentration in either experimental condition. Plasma ribose concentration was only assayed in R immediately before bouts A and B of the pretest. Initial plasma ribose concentration was 7 (range 0–17) µmol/l and increased to 93 (range 19–303) µmol/l before bout B. No side effects related to the ribose supplementation were reported over the duration of the study.

**DISCUSSION**

This study evaluated the effect of oral ribose supplementation on muscle ATP concentration after a single exhaustive intermittent exercise session and on muscle force and power output during a training period involving many repeated maximal intermittent exercise bouts. It was hypothesized that ribose administration enhances postexercise ATP recovery by stimulating de novo ATP synthesis and, via this mechanism, increases muscle performance during high-intensity intermittent training. The exercise test caused a significant net loss of adenine nucleotides from the quadriceps muscle, as evidenced by a ~20% lower ATP content 24 h after exercise (Table 1). Conversely, published literature indicates that successive days of repeated high-intensity training (10, 20), but not single sprint-exercise ses-

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**Table 1. Plasma uric acid and creatine kinase during and after intermittent muscle contractions**

<table>
<thead>
<tr>
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<th>Pretest</th>
<th>Posttest</th>
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<tr>
<td></td>
<td>Pre-A</td>
<td>+24 h</td>
</tr>
<tr>
<td>Creatine kinase, U/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>287 ± 107</td>
<td>390 ± 84*</td>
</tr>
<tr>
<td>Ribose</td>
<td>270 ± 69</td>
<td>554 ± 142*</td>
</tr>
<tr>
<td>Uric acid, mmol/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>0.40 ± 0.04</td>
<td>0.35 ± 0.02*</td>
</tr>
<tr>
<td>Ribose</td>
<td>0.39 ± 0.03</td>
<td>0.39 ± 0.04</td>
</tr>
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</table>

Values are means ± SE of 9–10 observations. During the pretest and posttest, subjects performed 2 bouts (A and B) of maximal exercise. Each bout consisted of 15 series of 12 maximal knee extensions on an isokinetic dynamometer, with 15 s of rest between each series. A 1-h rest interval separated bouts A and B. Blood samples were taken from an antecubital vein immediately before and after bouts A and B and 24 h after bout B (+24 h). A 6-day training period separated the pretest and posttest. *Significantly different from Pre-A, P < 0.05.

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**Table 2. Blood lactate, ammonia, and glucose during and after intermittent muscle contractions**

<table>
<thead>
<tr>
<th></th>
<th>Pretest</th>
<th>Posttest</th>
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<tbody>
<tr>
<td></td>
<td>Pre-A</td>
<td>Post-A</td>
</tr>
<tr>
<td>Lactate, mmol/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>0.77 ± 0.05</td>
<td>3.79 ± 0.39*</td>
</tr>
<tr>
<td>Ribose</td>
<td>0.81 ± 0.08</td>
<td>4.91 ± 0.59*</td>
</tr>
<tr>
<td>Ammonia, µmol/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>53.1 ± 3.73</td>
<td>99.9 ± 10.6*</td>
</tr>
<tr>
<td>Ribose</td>
<td>64.2 ± 9.82</td>
<td>121 ± 12*</td>
</tr>
<tr>
<td>Glucose, mmol/l</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Placebo</td>
<td>5.2 ± 0.2</td>
<td>4.8 ± 0.2</td>
</tr>
<tr>
<td>Ribose</td>
<td>5.0 ± 0.2</td>
<td>4.6 ± 0.3</td>
</tr>
</tbody>
</table>

Values are means ± SE of 9–10 observations. During the pretest and posttest, subjects performed 2 bouts (A and B) of maximal exercise. Each bout consisted of 15 series of 12 maximal knee extensions on an isokinetic dynamometer, with 15 s of rest between each series. A 1-h rest interval separated bouts A and B. Blood samples were taken from a hyperemic earlobe (lactate and glucose) and from an antecubital vein (ammonia) immediately before and after bouts A and B and 24 h after bout B (+24 h). A 6-day training period separated the pretest and posttest. *Significantly different from Pre-A, P < 0.05.
sions, can cause muscle ATP content to be decreased for $>24$ h after exercise. However, the number of intermittent sprint bouts performed in the present study (30 “sprints” involving 12 maximal knee extensions each) was much higher than in any earlier study (1, 9, 15, 17, 21, 28). Clear evidence has been provided that the loss of purines from muscle is enhanced with increasing number of successive sprints to be performed (21). In addition, the strain imposed on the quadriceps muscle, the site of biopsy sampling, during maximal unilateral knee extensions is conceivably higher than that during whole body sprint exercise (18, 21, 28). Thus, despite the fact that the exercise involved only a small muscle mass, it caused a nearly fourfold rise of systemic arterial lactate concentration. In addition, plasma ammonia, an extracellular marker of muscle adenine nucleotide catabolism, was significantly increased (Table 2). However, administration of oral ribose supplements did not alter the exercise-induced changes of muscle ATP content immediately after or $24$ h after exercise. The reason for the low ATP values measured in the present study is unclear. However, NAD concentrations measured in the muscle biopsies were normal and constant at all times, which proves the validity of the ATP and TAN changes measured. Thus four doses of oral ribose at 4 g each within a 4-h window after the exercise test clearly failed to beneficially impact purine nucleotide metabolism during the strenuous exercise conditions used in the present study.

If ribose administration does not facilitate postexercise adenine nucleotide resynthesis, then exercise capacity cannot be improved via this mechanism. We measured maximal torque and power output during an 8-day training period that consisted of the pretest (day 0) and the posttest (day 7), with 6 days of high-intensity training between test days (2 daily training sessions). For the pretest, the training days, and the posttest, muscle power outputs were similar between the P and R group. Ribose intake did not alter mean power production for either of the two intermittent exercise bouts (A and B), which were separated by 1 h of rest (Fig. 1). In addition, it failed to enhance power recovery during the short rest intervals (15 s) between the maximal contraction series within each bout (Fig. 2). Furthermore, maximal isometric force was similar between R and P before exercise and after fatigue due to the intermittent exercise test (Fig. 3). Thus this study clearly shows that intake of ribose at 16 g/day, in the conditions of the present study, did not enhance maximal muscle force and power production during maximal intermittent muscle contractions.

Zarzeczny and co-workers (27) previously demonstrated that increasing perfusate ribose concentration from ~0 to ~5 mmol/l increased nucleotide synthesis rate in perfused rat skeletal muscles. Such a high plasma ribose concentration conceivably cannot be established in humans by oral ribose intake. First, the ribose intakes required would be beyond the limits of gastrointestinal tolerance. Second, because of the very rapid clearance of plasma ribose (19), it is very difficult to obtain high and stable plasma ribose levels by oral ribose ingestion. (32). In the present study, subjects ingested 4 g of ribose immediately before and after a 15-min intermittent exercise bout. Plasma ribose concentration measured 1 h after the last dose on average was <0.1 mmol/l, which is conceivably too low to significantly enhance muscle ribose uptake to stimulate purine nucleotide synthesis. We cannot exclude the possibility that substantially higher ribose intake rates than used here, if well tolerated, might enhance postexercise ATP recovery in humans. However, our findings provide strong evidence to suggest that the ribose doses used by athletes result in plasma ribose levels that are too low to allow for an ergogenic action. Our ribose administration regimen (4 doses at 4 g each) was even higher than that recommended for most, if not all, commercial ribose preparations.

In conclusion, oral ribose supplementation at 16 g (4 doses at 4 g each) per day does not beneficially impact muscle ATP recovery and muscle force and power output during repeated days of maximal intermittent exercise training.

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REFERENCES


