

Effects of Oral D-Ribose Supplementation on Anaerobic Capacity and Selected Metabolic Markers in Healthy Males

R.B. Kreider, C. Melton, M. Greenwood, C. Rasmussen, J. Lundberg, C. Earnest, and A. Almada

Oral D-ribose supplementation has been reported to increase adenine nucleotide synthesis and exercise capacity in certain clinical populations. Theoretically, increasing adenine nucleotide availability may enhance high intensity exercise capacity. This study evaluated the potential ergogenic value of D-ribose supplementation on repetitive high-intensity exercise capacity in 19 trained males. Subjects were familiarized to the testing protocol and performed two practice-testing trials before pre-supplementation testing. Each test involved warming up for 5 min on a cycle ergometer and then performing two 30-s Wingate anaerobic sprint tests on a computerized cycle ergometer separated by 3 min of rest recovery. In the pre- and post-supplementation trials, blood samples were obtained at rest, immediately following the first and second sprints, and following 5 min of recovery from exercise. Subjects were then matched according to body mass and anaerobic capacity and assigned to ingest, in a randomized and double blind manner, capsules containing either 5 g of a dextrose placebo (P) or D-ribose (R) twice daily (10 g/d) for 5 d. Subjects then performed post-supplementation tests on the 6th day. Data were analyzed by ANOVA for repeated measures. Results revealed a significant interaction ($p = .04$) in total work output. Post hoc analysis revealed that work significantly declined (-18 ± 51 J) during the second post-supplementation sprint in the P group while being maintained in the R group (-0.0 ± 31 J). No significant interactions were observed in peak power, average power, torque, fatigue index, lactate, ammonia, glucose, or uric acid. Results indicate that oral ribose supplementation (10 g/d for 5 d) does not affect anaerobic exercise capacity or metabolic markers in trained subjects as evaluated in this study.

Key Words: exercise, sport nutrition, ergogenic aids

R.B. Kreider, C. Melton, C. Rasmussen, J. Lundberg, and M. Greenwood are with the Exercise & Sport Nutrition Laboratory, Center for Exercise, Nutrition & Preventive Health, in the Department of Health, Human Performance & Recreation at Baylor University, Waco, TX 76798-7313. C. Earnest is with the Division of Epidemiology & Clinical Applications at The Cooper Institute for Aerobics Research, 12330 Preston Road, Dallas, TX 75230. A. Almada is with MetaResponse Sciences, Inc., 30131 Town Center Drive, #211, Laguna Niguel, CA 92677. Address correspondence to Dr. Kreider.

Introduction

Over the last several years, the naturally occurring carbohydrate D-ribose has been marketed as a potential nutritional ergogenic aid for athletes. The theoretical rationale has been primarily based on the role that ribose plays in energy metabolism (13). In this regard, ribose is a constituent in the *de novo* synthesis of nucleotides like adenosine triphosphate (ATP; 10, 17). Research has suggested that maintaining total adenine nucleotide (TAN) availability during intense exercise may affect the ability to sustain high intensity exercise (6, 7, 12, 14). Additionally, enhancing the ability to resynthesize TAN pools following exercise or during training may affect recovery and the ability to perform subsequent high-intensity exercise bouts (7, 15). Since only trace amounts of ribose are typically found in the diet and the ability to synthesize ribose in the body has been reported to be somewhat limited, oral ribose supplementation has been suggested as a way of enhancing the TAN pool prior to, during, and following exercise in healthy (13) and diseased populations (10, 16). Theoretically, increasing TAN may provide some ergogenic benefit during and/or following high-intensity exercise (9).

In support of the potential ergogenic value, several studies have evaluated the effects of oral ribose supplementation on ATP availability in various clinical populations. For example, Wagner and colleagues (16) reported that oral ribose supplementation (3 g every 10 min beginning 1 hour prior to exercise and concluding at the completion of a maximal exercise test) increased ATP availability in patients with myoadenylate deaminase deficiency (an enzyme deficiency that interferes with the ability to resynthesize ATP during exercise). Pliml and coworkers (10) reported that taking 60 g/d of ribose (15 g taken four times per day) for 3 d increased exercise time to onset of ischemia during an exercise stress test (performed after the 3-d loading period) in patients with severe coronary artery disease. Additionally, Gross and associates (5) reported that ribose supplementation (2 g ingested every 5 min during a 30-min submaximal cycling exercise bout) blunted the normal increases in plasma hypoxanthine in healthy subjects, suggesting less of a decrease in TAN. However, these researchers also reported that ribose supplementation increased plasma hypoxanthine levels in the single patient with myoadenylate deaminase deficiency evaluated in this study suggesting a possibly an ergolytic effect. Although more data is needed, these findings suggest that ribose supplementation may affect energy metabolism and provide some clinical benefit under certain conditions.

The potential ergogenic value of ribose supplementation in athletes is less clear. Op't Eijnde and colleagues (9) recently reported that ribose supplementation (16 g/d for 6 d) during training did not significantly effect power output while performing a series of repetitive maximal effort knee extension exercises or *de novo* ATP resynthesis rates. Remaining studies have only been reported in abstract form to date. Analysis of these preliminary studies indicates that ribose supplementation (10–24 g/d for 1.5–30 d) has limited effects on repetitive sprint performance (2, 11); may improve the number of repetitions performed during 10 sets to failure in the bench press, but has no significant effects on body composition or one-repetition maximum (1RM) bench press performance during training (1); and, may attenuate the decline in TAN following sprint exercise and training (4). Based on this analysis, it is clear that much more research is needed to evaluate the potential ergogenic value of ribose supplementation before any definitive conclusions can be drawn. The purpose of this study was to examine the potential ergogenic value of oral ribose

supplementation (10 g/d for 5 d) on repeated bouts of sprint exercise in trained athletes and to determine whether ribose supplementation may affect selected metabolic markers during and/or following short-term recovery.

Methods

Subjects

Twenty-eight resistance-trained subjects volunteered to participate in this study. Subjects were informed as to the experimental procedures and signed informed consent statements in adherence with the human subject's guidelines of The University of Memphis and the American College of Sports Medicine. In order to participate in the study, subjects had to: (a) be an experienced resistance-trained athlete (>1 yr) who was currently training at least 3 hrs/wk with a program that included high intensity strength training and sprint conditioning; (b) not have taken any ergogenic aid or prescription drug that may have influenced anaerobic exercise capacity for 8 weeks prior to the start of the study (e.g., creatine, ribose, sodium phosphate, sodium bicarbonate, ergogenic levels of caffeine); and (c) sign statements indicating that they had no current or past history of anabolic steroid use.

Subjects who volunteered to participate in this study underwent two familiarization/ practice exercise sessions in order to become accustomed to the exercise testing to be employed in the study. Eight subjects who initially volunteered to participate in the study decided not to continue due to the difficulty in performing the anaerobic capacity sprint tests. The remaining 20 subjects were matched according to body mass and sprint capacity and participated in pre-supplementation testing. One subject was unable to complete the trial due to difficulty in scheduling the post-test session. Therefore, 19 subjects completed all aspects of the study. Subjects were descriptively (mean \pm SD) 23 \pm 3 yrs, 91 \pm 19 kg, 71 \pm 3 in., and 14.8 \pm 2% body fat.

Experimental Design

Subjects reported to the lab in order to sign informed consent statements, complete medical and training history questionnaires, and become familiarized with the testing protocol. The subjects were then scheduled to participate in two practice testing trials in order to establish reliability in performing the exercise protocol. For each testing session, subjects were instructed to refrain from exercise for 48 h and to fast for 4 h prior to testing. In addition, subjects were asked to maintain a similar diet on the day prior to each testing session. After the practice trials were completed, the subjects were scheduled for pre-testing. Once reporting to the lab, the subjects were weighed and had body composition determined for descriptive purposes. Subjects then donated a resting blood samples. Pre-exercise blood samples were typically obtained within 10 min of reporting to the lab. Subjects warmed up on a bicycle ergometer for 5 min at a standardized workload (2 kg, 60 rev/min). Subjects then performed two 30-s Wingate anaerobic capacity tests separated by 3 min of passive rest recovery. Blood samples were obtained immediately after the first and second sprints as well as after 5 min of recovery of the second sprint.

Subjects were matched according to body mass and anaerobic capacity (peak power, average power, and total work). In a double blind and randomized manner, subjects were assigned to supplement their normal diet with 10 g/d of powdered

D-ribose (Bioenergy, Minneapolis, MN) or a dextrose placebo. The supplements were encapsulated in 0.5-g colored capsules, placed in bottles, and independently labeled by a cooperating pharmacist for double blind administration. Subjects ingested 10 capsules in the morning (5 g) and 10 capsules in the evening (5 g) for 5 days (total of 50 g). Subject compliance in taking the supplements was verified by collecting empty supplement bottles and by questionnaire. On the day following the 5-d supplementation period, subjects reported back to the lab to undergo post-intervention testing. Testing sessions were performed at the same time of day between trials. Post-supplementation testing was performed in an identical fashion as the pre-tests described above with the exception that body composition was not measured.

Procedures

Total body mass was measured on a calibrated digital scale with a precision of ± 0.02 kg (Sterling Scale Co., Southfield, MI). Pre-supplementation body composition was determined for descriptive purposes using standard skinfold techniques (8).

Approximately 200–400 microliters of whole blood was collected into a microcapillary tube from a dry, pre-warmed finger using standard finger stick phlebotomy techniques. The microcapillary tube was centrifuged at 1,500 rev/min for 10 min using a standard centrifuge. Serum was separated from the microcentrifuge tube and assayed for ammonia, lactate, glucose, and uric acid using an Ektachem DT60 II chemistry analyzer (Johnson and Johnson, Rochester, NY). Inter-assay variances for low and high controls were 5, 10; 0, 0; 0.5, 0.7; and 0, 0.5% for NH_3 , lactate, glucose, and uric acid, respectively. The observed variances on low and high controls for NH_3 , lactate, glucose, and uric acid were less than the within-day normal coefficients of variation reported by the manufacturer on these chemistries (4.5–10.6%, 13.3–1.1%, 1.6–1.2%, and 1.5–1.9%, respectively).

Anaerobic capacity tests were performed on a computerized $\text{CardiO}_2^{\text{TM}}$ cycle ergometer equipped with toe clips at a standardized work rate of 7.5 J/kg/rev (ErgometR_x Corp., St. Paul, MN). Seat position was standardized between trials. The ergometer was connected via an RS232 parallel interface to a Dell 466/Le Optiplex computer (Dell Computer Corp., Austin, TX) using ErgometR_x CardioscribeTM and ExerscribeTM software (ErgometR_x Corp., St. Paul, MN). Crank frequency was measured using a crystal referenced optic encoder with a precision range of 0 to 200 rev/min and an accuracy of ± 1 rev/min. Pedal torque was determined by a calibrated strain gauge with a range of 0 to 2,000 W and an accuracy of $\pm 1\%$. Data were collected and downloaded into the computer at 0.5-s intervals. The test-to-test reliability of performing this anaerobic capacity test in our lab was $\pm 3\%$, $r = 0.96$. The Wingate anaerobic capacity test has been reported to be a reliable and valid method of assessing anaerobic capacity (3).

Statistical Analysis

Data were analyzed by repeated measures analysis of variance (ANOVA) using SPSS for Windows Version 10.05 software (SPSS Inc., Chicago, IL). Data were considered significantly different when the probability of error was 0.05 or less. Tukey post hoc procedures were employed when a significant interaction was observed. Data are presented as means \pm standard deviations of means.

Results

Side Effects

Subjects tolerated the supplementation protocol well, with no reports of medical problems/symptoms in post-study questionnaires administered in a blinded manner.

Sprint Performance

Table 1 presents mean power and work data observed for the placebo and ribose groups. A significant Group \times Time interaction ($p = .04$) was observed in total work among groups. Post hoc analysis revealed that total work values in the ribose group were significantly greater than the placebo group during the second sprint test following supplementation. This difference was due to a decrease in performance in the placebo group during the second sprint rather than a significant increase in total work in response to ribose supplementation. No significant differences were observed in peak power, average power, time to peak power, torque, or fatigue index between the groups. Figure 1 presents the average power values observed for each 5-s interval during the sprint tests for the placebo and ribose groups. No significant Group \times Time interactions were observed among groups during pre- or post-supplementation sprints.

Metabolic Markers

Figure 2 presents ammonia, lactate, glucose, and uric acid values observed for the placebo and ribose groups during the pre and post-supplementation sprint trials. Exercise significantly increased ($p < .0001$) lactate, ammonia, glucose, and uric acid levels. However, no significant Group \times Time interactions were observed among groups in these metabolic markers.

Discussion

This study evaluated whether ribose supplementation (10 g/d for 5 d) affects repetitive sprint performance and/or metabolic markers in resistance trained athletes. Our rationale in designing this study was to examine whether several days of ribose supplementation at levels that were typically 3–5 times greater than that marketed as a potential ergogenic aid at the time of this study affected the ability to perform successive bouts of exhaustive exercise with limited rest recovery. Although we could have evaluated various combinations of sprint lengths and/or recovery times, we decided to evaluate the effects of ribose supplementation on performing two standard 30-s Wingate anaerobic capacity tests with 3 min of rest recovery between sprints. Our rationale in doing so was to ensure that the exercise bouts: (a) evaluated maximal anaerobic power using a valid, reliable, and well accepted test of anaerobic capacity (3); (b) would likely promote adenine nucleotide degradation and muscular fatigue (6, 14); and (c) would challenge the ability to recover from exhaustive exercise at typical work to rest ratios utilized by trained athletes (e.g., 1:3-6).

Given the theoretical rationale of ribose supplementation, we felt that if daily ribose supplementation enhanced ATP availability during and/or following exhaustive exercise, the athletes should have been able to increase peak power and/or total

Table 1 Anaerobic Capacity Variables for the Placebo (P) and Ribose (R) Groups

Variable	Group	Day 0		Day 6		P
		Sprint 1	Sprint 2	Sprint 1	Sprint 2	
Total work (J)	P	258 (35)	184 (47)	237 (50)	165 (57)	G = .80; T = .001; G×T = .04
	R	241 (31)	196 (37)	227 (26)	196 (27)†	
Peak power (W)	P	1021 (208)	895 (189)	1041 (188)	908 (187)	G = .56; T = .001; G×T = .29
	R	1016 (138)	828 (68)	1028 (145)	836 (70)	
Average power (W)	P	726 (122)	566 (113)	722 (138)	548 (99)	G = .71; T = .001; G×T = .42
	R	704 (75)	549 (60)	700 (98)	545 (68)	
Time to peak power (s)	P	3.67 (3.1)	2.94 (1.3)	2.67 (1.0)	2.44 (1.3)	G = .69; T = .07; G×T = .50
	R	2.85 (1.1)	3.25 (2.0)	2.25 (1.2)	2.55 (0.8)	
Torque (N·m)	P	76.2 (14)	71.3 (11)	70.6 (10)	70.6 (10)	G = .95; T = .003; G×T = .50
	R	79.5 (11)	75.9 (10)	78.9 (12)	74.8 (9)	
Fatigue index (%)	P	60.3 (10)	71.7 (11)	58.8 (6)	73.0 (13)	G = .95; T = .13; G×T = .24
	R	64.6 (10)	70.1 (13)	61.8 (10)	68.4 (15)	

Note. Data are means (standard deviations). G = group alpha level, T = time alpha level, G×T = Group × Time alpha level. † represents $p < .05$ difference from the P group.

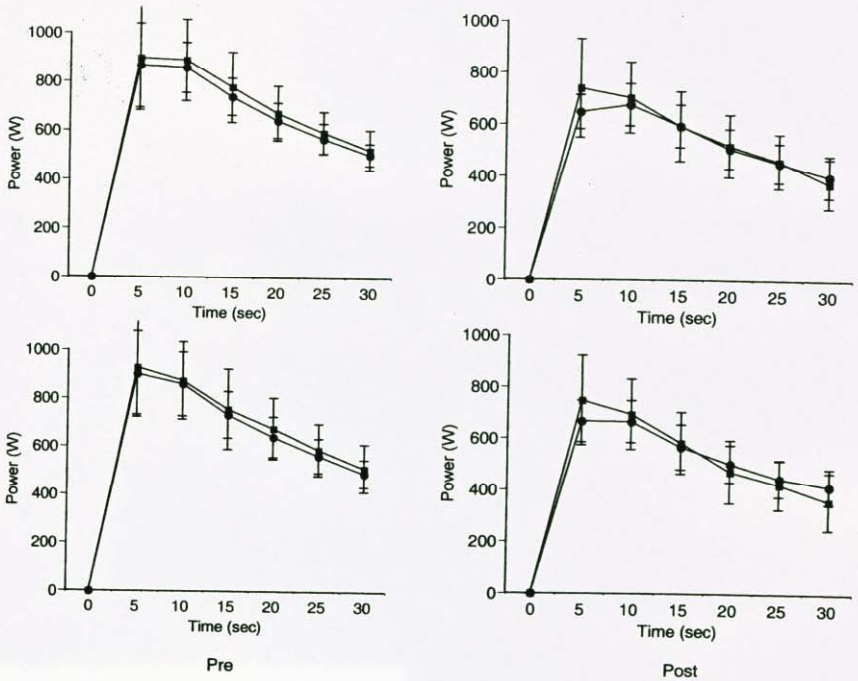


Figure 1 — Power output values observed at each 5-s interval for the placebo (∇) and ribose (\bullet) groups in sprint 1 (top panel) and sprint 2 (lower panel) for pre- and post-supplementation tests. Data are means and standard deviations.

work during the anaerobic capacity tests evaluated. Moreover, if ribose supplementation provides an ergogenic attenuation in the decline in the TAN pool during exercise and/or enhances adenine nucleotide resynthesis following exercise, differences in selected metabolic responses to exercise (i.e., ammonia, lactate, uric acid, and/or glucose) should have been evident at the completion of the first sprint, second sprint, and/or within 5 min of recovery from the final bout of exercise (i.e., about 8.5 min after performing the initial sprint). Although it is possible that differences between groups may have been more evident if we evaluated a longer period of recovery and/or attempted to monitor the peak levels of these metabolites, it was our view that such differences would be less likely to affect performance of athletes performing successive bouts of intense exercise with typical work to rest ratios (e.g., 1:3-6). Consequently, we felt that the experiment employed provided a good initial assessment of the proposed ergogenic value of ribose supplementation on repetitive sprint performance in athletes.

Analysis of work output, blood lactate, and ammonia responses to the exercise tests employed in this study indicated that this design was successful in promoting fatigue. In this regard, the subjects experienced a 60–73% decrease in power output during the 30-s sprint tests and a 23–25% decrease in total work performed from the first to second sprints. In addition, the exercise protocol significantly

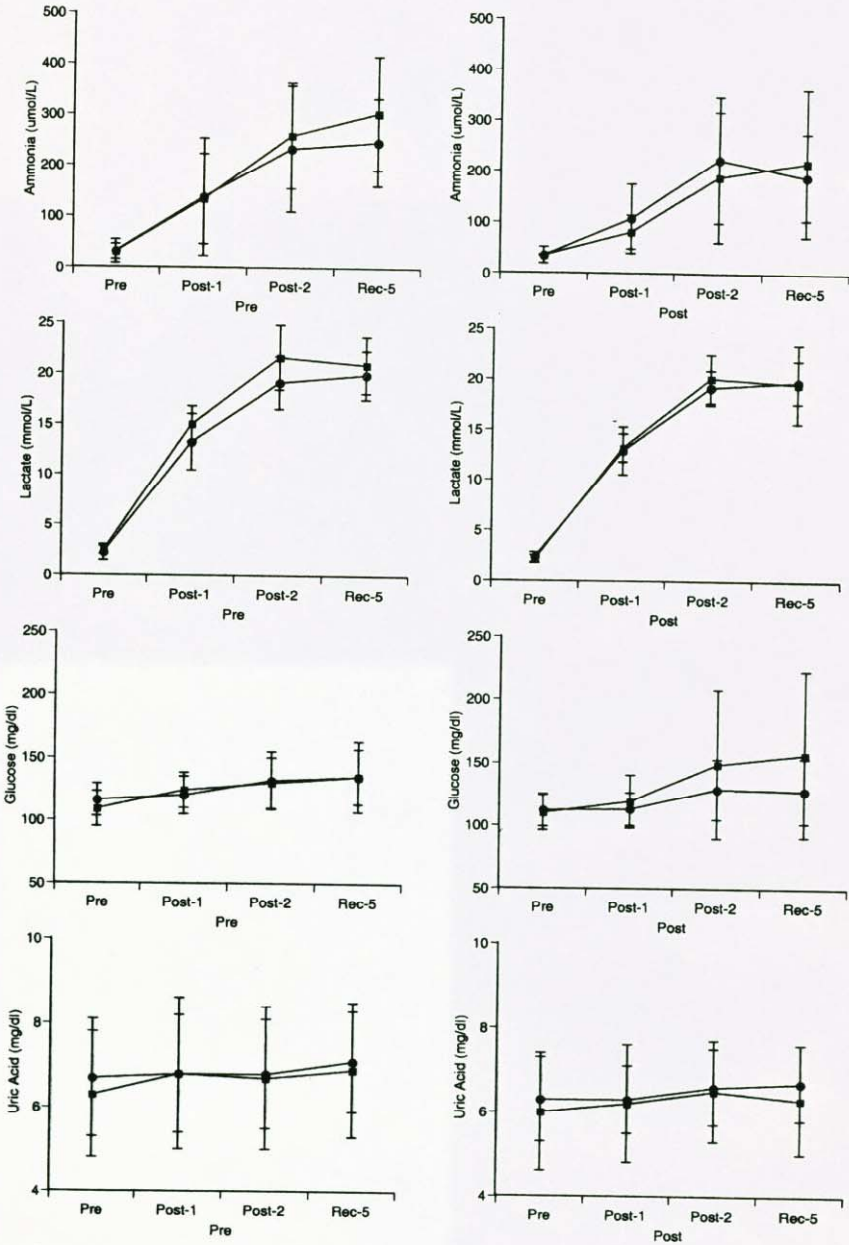


Figure 2— Ammonia, lactic acid, glucose, and uric acid values observed for the placebo (V_T) and ribose (S_H) groups prior to and following supplementation. Data are means and standard deviations.

increased lactate (about 6 fold), ammonia (about 8 fold), glucose (20–36%), and uric acid (6–8%). Ammonia is commonly used as a marker of adenine nucleotide degradation. Comparatively, the magnitude of change in blood lactate and ammonia levels observed in the present study was 2 to 5 times greater than that reported by Op't Eijnde and colleagues (9) when performing 15×12 maximal contractions of isokinetic knee extensions that promoted a 20–25% decrease in TAN. Although muscle biopsies were not obtained in this preliminary study, these findings suggest that the exercise protocol employed was not only metabolically challenging but also likely promoted a significant decrease in TAN.

Results of the present study do not support contentions that ribose supplementation may serve as an effective ergogenic aid for athletes. In this regard, although a significant interaction was observed between groups in sprint total work, differences between groups were related to a decrease in performance in the placebo group rather than an improvement in performance in the ribose group. No significant differences were observed from pre- to post-supplementation values in the ribose group. In fact, the work performed after ribose supplementation was nearly identical to pre-supplementation values suggesting that ribose supplementation had no impact on exercise capacity as examined in this study. Further, although the exercise protocol resulted in significant increases in lactate, ammonia, glucose, and uric acid concentrations, no significant differences were observed between the control and ribose supplemented groups in these metabolic markers. These findings suggest that the ribose supplementation protocol employed did not alter metabolic responses to exercise and/or short-term recovery.

Present findings support several other recent studies indicating that ribose supplementation does not significantly affect performance or metabolic markers. For example, recent preliminary reports indicate that ribose supplementation (~ 10 – 20 g/d for 1.5–5 d) does not significantly effect repetitive cycling performance (e.g., 6– 15×10 -s sprints with 60-s recovery; 2, 4). In addition, Op't Eijnde and colleagues (9) reported that ribose supplementation during training (16 g/d for 6 d) did not significantly effect power output when performing two bouts of isokinetic knee extensions (i.e., 15 sets of 12 maximal contractions with 15 s recovery), plasma metabolites (i.e., blood lactate, ammonia, uric acid, glucose, or creatine kinase), or muscle adenine nucleotides (i.e., ATP, ADP, AMP, IMP, or TAN). Collectively, results of the present study and these reports do not support contentions that ribose supplementation affects high intensity exercise capacity in healthy individuals.

Present findings contrast recent preliminary reports suggesting a possible ergogenic value of ribose supplementation in healthy individuals. For example, Berardi and associates (2) reported that ribose supplementation (4×8 -g doses for 36-hours prior to exercise) increased peak power (2.2–7%) and mean power (2–10%) during four of six cycling sprints (6×10 -s cycle sprints with 60 s of rest recovery) in a subgroup of 8 healthy subjects. Although these differences were not statistically significant, effect size calculations revealed a strong treatment effect, suggesting a possible ergogenic benefit. Researchers also reported that although ribose supplementation (20 g/d for 3 d prior to training, during a 5-d training period, and for 3 d following training) did not significantly affect cycling sprint performance (15×10 -s sprints), it partially attenuated the decrease in the TAN pool following the final sprint session and during a 65-h recovery period in comparison to the placebo (4, 11). Finally, Antonio and colleagues (1) reported that ribose supplementation (10 g/d administered in 5-g doses prior to and following training bouts)

promoted a greater increase in the number of repetitions performed during 10 sets to failure on the bench press in comparison to the placebo group (i.e., 30% vs. 7%).

It is our view that a significant amount of basic and applied research needs to be conducted on ribose supplementation before any recommendations can be made about its ergogenic value for athletes. Additional research should determine optimal dosages and timing of ribose supplementation. In this regard, it is presently unknown whether timing of ribose supplementation may affect the ergogenic value of ribose. It is possible that ergogenic benefits may be more pronounced if athletes ingest ribose prior to, during, and/or following exercise rather than 2–3 times throughout the day. It is also possible that larger and/or more frequent doses of ribose may need to be ingested in order to promote an ergogenic benefit in healthy individuals. Additional research should evaluate the acute metabolic changes that occur in response to ribose supplementation in order to understand the metabolic fates, dosage, and timing issues. Research is also needed to determine the types of exercise and supplementation protocols that may or may not be of ergogenic benefit for athletes. It is possible that studies to date have yet to examine the type, intensity, and/or duration of exercise that may benefit from ribose supplementation. Finally, it is possible that ribose supplementation may be more beneficial during training. Nevertheless, based on results of the present study, it appears that ribose supplementation (10 g/d for 5 d) does not affect performance and/or metabolic responses to repetitive high intensity exercise as examined in this study.

References

1. Antonio, J., D. Falk, and D. Gammeren. Ribose administration in recreational body-builders. *Med. Sci. Sports Exerc.* 33(5):S166, 2001. (Abstract)
2. Berardi, J., T. Ziegenfuss, and B. Hall. Effects of ribose supplementation on sprint performance: a pilot study. *Med. Sci. Sports Exerc.* 32(5):S260, 2000. (Abstract)
3. Brown, L.E., and J.P. Weir. ASEP Procedures Recommendation I: accurate assessment of muscular strength and power. *J. Exerc. Physiol. Online.* 4(3):1-21, 2001.
4. Gallagher, P.M., D.L. Williamson, M.P. Godard, J.R. Witter, and S.W. Trappe. Effects of ribose supplementation on adenine nucleotide concentration in skeletal muscle following high-intensity cycle exercise. *Med. Sci. Sports Exerc.* 33(5):S167, 2001. (Abstract)
5. Gross, M., B. Kormann, and N. Zollner. Ribose administration during exercise: effects on substrates and products of energy metabolism in healthy subjects and a patient with myoadenylate deaminase deficiency. *Klin. Wochenschr.* 69:151-155, 1991.
6. Hargreaves, M., M.J. McKenna, D.G. Jenkins, S.A. Warmington, J.L. Li, R.J. Snow, and M.A. Febbraio. Muscle metabolites and performance during high-intensity, intermittent exercise. *J. Appl. Physiol.* 84:1687-1691, 1998.
7. Hellsten-Westing, Y., B. Norman, P.D. Balsom, and B. Sjodin. Decreased resting levels of adenine nucleotides in human skeletal muscle after high-intensity training. *J. Appl. Physiol.* 74:2523-2528, 1993.
8. Jackson, A.S. and M.L. Pollock. Practical assessment of body composition. *Phys. Sport Med.* 13:76-90, 1985.
9. 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.

10. Pliml, W., T. von Arnim, A. Stablein, H. Hofmann, H.G. Zimmer, and E. Erdmann. Effects of ribose on exercise-induced ischaemia in stable coronary artery disease. *Lancet*. 340:507-510, 1992.
11. Raue, U., P.M. Gallagher, D.L. Williamson, M.P. Godard, and S.W. Trappe. Effects of ribose supplementation on performance during repeated high-intensity cycle sprints. *Med. Sci. Sports Exerc.* 33(5):S44, 2001. (Abstract)
12. Stathis, C.G., M.A. Febbraio, M.F. Carey, and R.J. Snow. Influence of sprint training on human skeletal muscle purine nucleotide metabolism. *J. Appl. Physiol.* 76:1802-1809, 1994.
13. Stout, J.R. Maintaining muscle cell energy: The possible role of ribose. In: *Sports Supplements*, J. Antonio and J.R. Stout (Eds.). Philadelphia: Lippincott, Williams, and Wilkins, 2001, pp. 248-249.
14. Tullson, P.C., and R.L. Terjung. Adenine nucleotide synthesis in exercising and endurance-trained skeletal muscle. *Am. J. Physiol.* 261:C342-347, 1991.
15. Tullson, P.C., J. Bangsbo, Y. Hellsten, and E.A. Richter. IMP metabolism in human skeletal muscle after exhaustive exercise. *J. Appl. Physiol.* 78:146-152, 1995.
16. Wagner, D.R., U. Gresser, and N. Zollner. Effects of oral ribose on muscle metabolism during bicycle ergometer in AMPD-deficient patients. *Ann. Nutr. Metab.* 35:297-302, 1991.
17. Zimmer, H.G. Restitution of myocardial adenine nucleotides: acceleration by administration of ribose. *J. Physiol.* 76:769-775, 1980.

Acknowledgments

We would like to thank the subjects who participated in this study and the laboratory assistants in the Exercise & Sport Nutrition Laboratory at the University of Memphis who assisted in data acquisition and analysis.