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Oral Adenosine-5′-triphosphate (ATP) Administration Increases Postexercise ATP Levels, Muscle Excitability, and Athletic Performance Following a Repeated Sprint Bout

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ABSTRACT

Objective: Oral adenosine-5′-triphosphate (ATP) administration has failed to increase plasma ATP levels; however, chronic supplementation with ATP has shown to increase power, strength, lean body mass, and blood flow in trained athletes. The purpose of this study was to investigate the effects of ATP supplementation on postexercise ATP levels and on muscle activation and excitability and power following a repeated sprint bout.

Methods: In a double-blind, placebo-controlled, randomized design, 42 healthy male individuals were given either 400 mg of ATP as disodium salt or placebo for 2 weeks prior to an exercise bout. During the exercise bout, muscle activation and excitability (ME, ratio of power output to muscle activation) and Wingate test peak power were measured during all sprints. ATP and metabolites were measured at baseline, after supplementation, and immediately following exercise.

Results: Oral ATP supplementation prevented a drop in ATP, adenosine-5′-diphosphate (ADP), and adenosine-5′-monophosphate (AMP) levels postexercise ($p < 0.05$). No group by time interaction was observed for muscle activation. Following the supplementation period, muscle excitability significantly decreased in later bouts 8, 9, and 10 in the placebo group (−30.5, −28.3, and −27.9%, respectively; $p < 0.02$), whereas ATP supplementation prevented the decline in later bouts. ATP significantly increased Wingate peak power in later bouts compared to baseline (bout 8: +18.3%, bout 10: +16.3%).

Conclusions: Oral ATP administration prevents exercise-induced declines in ATP and its metabolite and enhances peak power and muscular excitability, which may be beneficial for sports requiring repeated high-intensity sprinting bouts.

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ATP; athletic performance; bioavailability; power; muscular excitability

Introduction

Long-term, periodized resistance exercise training produces increased skeletal muscle size and, ultimately, force-generating capacity [1–3]. Sports nutrition scientists have attempted to increase training-induced gains through a number of protocols, which generally attempt to augment and/or speed skeletal muscle regeneration. Adenosine-5′-triphosphate's (ATP) role as the primary intracellular energy source for body tissues is well established [4]. In addition, ATP has extensive extracellular functions that are primarily mediated through purinergic (P2Y and P2X) membrane receptors ubiquitously present in many cell types [5,6]. Extracellular-mediated functions of ATP include the increase in skeletal muscle calcium permeability, the blocking of chloride efflux, and vasodilation [7,8]. ATP is a food ingredient and part of our daily diet, with meat, fish, and nuts being particularly good sources. ATP has been marketed as a dietary supplement in the United States for more than 20 years and PEAK ATP is self-affirmed generally recognized as safe at levels of up to 800 mg per day. Consequently, ATP is not included in the prohibited list of the World Anti-Doping Agency [9].

Performance studies in athletes investigating the potential ergogenic effects of orally administered ATP showed beneficial effects at a daily dose of 400 mg, specifically when combined with resistance training exercise. Low-dose ATP supplementation (150 or 225 mg enteric-coated ATP per day) for 15 days did not alter bench press strength and endurance or peak or average Wingate power. However, ATP supplementation resulted in increased total bench press lifting volume (i.e., sets•repetitions•load) as well as within-group set 1 repetitions to failure [10]. Higher dose (400 mg per day for 15 days) ATP supplementation in form of the uncoated disodium salt increased minimum peak torque for the final 2 sets of a dynamometer test [11] and postexercise blood flow (400 mg ATP as disodium salt per day for 12 weeks) [12]. The increases suggest that orally delivered ATP may reduce muscle fatigue and enable a higher force output during repeated high-intensity bouts of exercise. Long-term ATP supplementation

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(400 mg ATP as disodium salt per day for 12 weeks) in combination with resistance training exercise resulted in significant increases in 1 repetition maximum, vertical jump power, and lean body mass [13].

The simultaneous analysis of ATP and its metabolites in blood for pharmacokinetic studies has been well established [14]. Though intravenous administration of ATP is bioavailable [15], several studies have shown that oral ATP in the form of enteric-coated pellets is not systemically bioavailable under resting conditions [16,17]. Oral ATP administration in the form of enteric-coated pellets for 28 days in healthy subjects did not increase plasma ATP concentrations. However, though systemic ATP increases at rest have not been demonstrated, chronic oral administration of ATP increased portal vein ATP concentration and nucleoside uptake by erythrocytes, which resulted in an increase in ATP synthesis in the erythrocytes [24], independent of an elevation in systemic plasma ATP. Therefore, chronic oral supplementation increases the capacity to synthesize ATP within erythrocytes in response to supplementation, without elevating the resting systemic concentration in plasma [24]. Thus, we suggest an indirect mechanism of ATP supplementation whereby chronic oral administration results in a greater capacity of erythrocytes to synthesize and thus sustain plasma ATP concentrations in response to the hypoxic perturbations triggered by high-intensity exercise. These contentions are supported by recent findings from 2 laboratories showing significant increases in blood flow following human elbow extension and rat leg kicking exercise when ATP was orally administrated [12].

We speculate that though orally administered ATP is rapidly metabolized, it increases the capacity to synthesize ATP in red blood cell pools [20,21] and could potentially prevent drops of ATP levels during times of increased energy expenditure, thereby explaining the previously described performance benefits. In this work, we tested the hypothesis that short-term ATP supplementation prevents drops in postworkout ATP and ATP metabolite levels and changes in power output and mental performance during and following a repeated sprint bout.

Methods

Subjects characteristics

Forty-two ($n = 42$) healthy male individuals between the ages of 18 and 30 participated in the study and were recruited into the study in 3 cohorts. Study participation required that all subjects resistance train at least 3 times per week for the past 6 months and have a minimum of 1 year of training experience. During the testing period, subjects were instructed not to train within 72 hours of the test in order to prevent any confounding variables (fatigue, muscle damage, etc.). As suggested by Kraemer et al. [22], nutrition assessment screening was performed by a registered sports dietitian to ensure that subjects were (1) on a diet consisting of 15–20% protein, 45–55% carbohydrate, and 25–30% fat; (2) not taking performance-enhancing supplements for the last 3 weeks; (3) not smokers; (4) not taking amino acid supplements; (5) not using anabolic or catabolic hormones; and (6) not on medication or supplements known to influence any of the variables measured in the study. Each participant gave written informed consent before participating in the study. All procedures were approved by the University of Tampa's Institutional Review Board. The present study was registered at ClinicalTrials.gov (registration no. NCT02093611).

Supplementation

In a double-blind, placebo-controlled, randomized design, subjects received either ATP (400 mg of ATP as disodium salt, PEAK ATP, TSI USA, Inc., Missoula, MT) or placebo (400 mg, rice flour) for a loading period of 14 days. Prior to the loading period, all subjects performed an intense sprint protocol as described below. On day 15, after the 14-day loading period, subjects consumed 400 mg of either ATP or a placebo 30 minutes prior to repeating the same sprinting bout. The sprinting bout was performed before and after supplementation to allow for comparison prior to and after treatment on all of the dependent variables discussed below. Compliance was assessed via completed supplement logs, meetings, collection of empty supplement packets, and reviewing compliance at all workout sessions.

Sprint protocol

The sprint protocol utilized in this study was adapted from Mendez-Villanueva et al. [23] and reported that muscle excitability decreased in a linear fashion after 10 6-second sprints on an air-braked, front-access cycle ergometer, with 30 seconds' rest between sprints. The current study conversely utilized a Wingate Ergomedic 894 (Monark, Vansbro, Sweden), allowing the weight to drop at 175 revolutions per minute (RMP), thus giving credence to increasing the rest time between bouts to 45 seconds. Subjects were familiarized with the Wingate prior to the first testing session. For familiarization, subjects came on 3 separate occasions and performed a 6-second Wingate in order to understand the mechanics as well as instill a neural familiarity with the protocol. Muscle power was assessed during maximal Wingate cycle ergometer sprinting (Monark, Vansbro, Sweden). During the cycling test, the volunteer was instructed to cycle against a predetermined resistance (7.5% of body mass) as fast as possible for 6 seconds. The saddle height was adjusted for each individual in order to produce a 5–10° knee flexion while the foot was in the low position of the central void. A standardized verbal stimulus was provided to the participant. Power output was recorded in real time by a computer connected to the Monark standard cycle ergometer (Monark model 894e, Monark, Vansbro, Sweden) during a 6-second sprint test. Wingate peak power (PP) was recorded using Monark Anaerobic test software (Monark Anaerobic Wingate Software, Version 1.0, Monark). The intraclass correlation (ICC) of muscle peak power was $r = 0.96$.

Muscle activation and excitability

Muscle activation was measured by detecting the electrical activity in muscle as determined by the average activation in millivolts. Delsys wireless electromyography sensors were used for the current study and placed on the vastus lateralis in order to look at the muscle activation. Muscle activation was recorded on the first 6-second sprint as well as every proceeding sprint.

After additional high-pass filtering (at 20 Hz) to eliminate movement artifacts, the root mean square of the signal was calculated from each sprint. For each sprint, the root mean square was normalized to the first sprint value, which will be assigned the value of 100%. Muscle excitability was defined as the ratio of power output to muscle activation. It was assumed that greater output (power) relative to input (activity) was relative to the excitable state of the muscle.

Assessment of ATP changes in blood

Baseline whole blood sample was taken on day 0 (no supplementation), day 15 in a fasted state, and 30 minutes following supplementation as well as immediately following the exercise stimulus. Venous blood was collected from the antecubital vein using a 21-gauge needle into a 4 ml EDTA tube (BD Vacutainer, Becton, Dickinson and Company, Franklin Lakes, NJ). For whole blood ATP determinations, the freshly drawn blood was added to ice-cold 7% trichloroacetic acid (w/v), which ess then kept on ice for 30 minutes [24]. Once the 7% trichloroacetic acid was added to the blood (immediately upon withdrawal), all metabolic activities stopped. The ATP and other metabolites in whole blood were collected and measured by high-performance liquid chromatography, with an intra- and interassay variance after that was less than 6 and 15%, respectively [14].

Reaction time

Reaction time and vertical jump peak power were only measured in a subgroup ($n = 30$). Reaction time was quantified using the Dynavision D2 Visuomotor Training Device (Dynavision International LLC, West Chester, OH). Subjects completed the reaction time test immediately before the Wingate trial (30 minutes post-ATP ingestion, immediately after vertical jump) and immediately after completing their postmeasure vertical jump. The Dynavision apparatus was specifically designed to measure and train visual scanning, visual attention in focal and peripheral fields, visuomotor reactions and coordination, and basic cognitive skills. For reaction time, subjects began while holding down one telegraph key. The subjects were then required to strike another telegraph key 30 cm away when the signal light was illuminated. The task imposes demands on integrated visual reaction time and movement time. The dependent variable is mean response time (in milliseconds) of the 5 test trials. The ICC of the mean response time was $r = 0.89$.

Vertical jump power

Immediately before the reaction time test (30 minutes postingestion), vertical jump peak power was assessed by taking the average of 3 attempts using a Tendo unit. Immediately prior to and after finishing their last Wingate sprint, subjects immediately performed 3 vertical jumps (average of 3 being taken as the post measure). The ICC of the vertical jump PP test was $r = 0.97$.

Statistical analysis

Shapiro-Wilk testing confirmed that dependent variables were normally distributed. An analysis of variance with repeated measures was used to scrutinize the effects of supplementation on dependent variables assuming group (placebo and ATP) and time as fixed factors. Because there were high interindividual variations in the pretest data for metabolites concentration, ATP, adenosine-5′-diphosphate (ADP), adenosine-5′-monophosphate (AMP), ΣATP-ADP-AMP, and ΣATP+AMP, we used delta values between day 15 post-Wingate − day 15 baseline values as the dependent variable. Day 15 baseline values were used as a covariate. Whenever, a significant F-value was obtained, a post hoc test with a Tukeý's adjustment was performed for multiple comparisons purposes. Within-group effect sizes (ESs; pre-to-post changes) were calculated using Cohen's *d*. Finally, when appropriate, we presented the mean value and confidence intervals of the absolute difference (CI_{diff}) because this approach allows us to know how much the supplementation changed the variables investigated, rather than only the level of statistical significance. In this regard, the confidence interval includes the *value* range in which the true population mean of the difference is likely to be. The significance level was previously set at $p < 0.05$. Results are expressed as mean ± standard deviation. SAS 9.2 and GraphPad Prism 6 (GraphPad Software, San Diego, CA, USA) were used to perform statistical analyses.

Results

Baseline results for anthropometry, body composition, and age are presented in Table 1. No significant between-group differences were detected at baseline for any of the variables ($p > 0.05$). No adverse events were observed during the 2 = week supplementation period.

Metabolites concentration

No significant differences between groups in metabolite concentrations were detected prior to the supplementation period, day 15 in a fasted state, and 30 minutes following supplementation on Day 15 ($p > 0.05$). Analysis of covariance revealed that absolute changes in ATP, ADP, and AMP from baseline to postexercise were greater in the ATP group than in the placebo group ($p < 0.05$; Fig. 1).

In addition, the analysis of covariance revealed that absolute changes for the sum of ATP-ADP-AMP (171.8 ± 54.5 vs −72.5 ± 88.0), ATP-ADP (179.2 ± 50.7 vs −58.4 ± 53.0), and ATP-AMP (24.3 ± 10.4 vs −30.0 ± 14.8) concentrations were greater than the placebo group ($p < 0.04$; Fig. 2).

Power changes in the wingate test

Changes for Wingate peak power output (PPO) are presented in Table 2. No significant differences between groups in PPO

Table 1. Baseline characteristics of the participants.

Variable	Placebo ($n = 21$)	ATP ($n = 21$)
Age (years)	20.2 ± 2.6	20.6 ± 2.5
Body mass (kg)	82.4 ± 7.2	82.0 ± 6.9
Lean body mass (kg)	64.2 ± 5.0	63.6 ± 5.1
Fat mass (kg)	12.9 ± 2.4	12.5 ± 2.4

ATP = adenosine-5′-triphosphate.

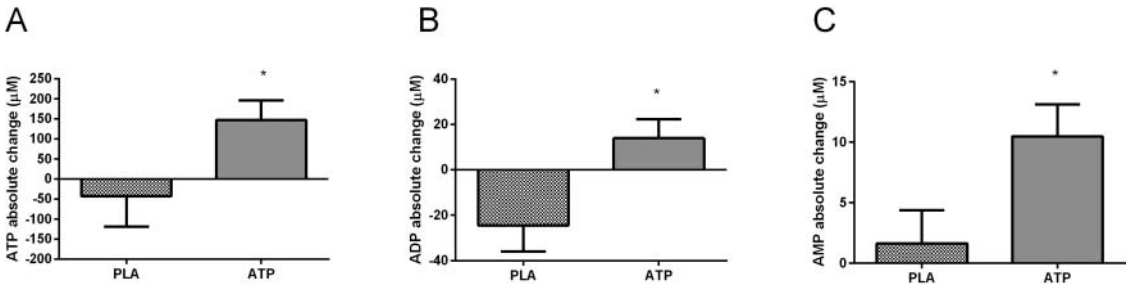

Figure 1. Delta changes in (A) ATP, (B) ADP, and (C) AMP levels from day 15 presprint to 30 minutes postsprint. *Indicates $p < 0.05$ different from placebo group.

were detected prior to the supplementation period ($p > 0.05$). A significant main time effect was observed for PPO ($p < 0.0001$), which demonstrated a similar performance decrement for both baseline and postsupplement periods (e.g., baseline: placebo, −41.2% and ATP, −41.0%; post: placebo, −36.8% and ATP, −28.0%). However, CI_{diff} revealed that only ATP significantly increased PPO pre-to-post at bout 8: mean 102.6 W, 95% CI_{diff}, 21.6 to 183.5 W; bout 10: mean 90.8 W, 95% CI_{diff}, 9.8 to 171.8 W.

Muscle activation and muscle excitability

Results for muscle activation during Wingate bouts are presented in Fig. 3. No significant between-group differences in muscle activation were detected prior to supplementation period ($p > 0.05$). A trend for a main time effect on muscle activation was observed for placebo and ATP groups ($p = 0.07$).

Herein, the ratio between muscle power outputs to muscle activation is defined as muscle excitability (ME), and the results are presented in Fig. 4. No significant between-group differences in muscle excitability were detected prior to the supplementation period ($p > 0.05$). ATP supplementation significantly increases ME in early bouts (bout 2, +21.5%; $p < 0.02$) and prevented declines in ME in later bouts, and the placebo group significantly decreased muscle excitability in sprints 8, 9, and 10 compared to sprint 1 (−30.5, −28.3, and −27.9%, respectively; $p < 0.02$).

Reaction time and vertical jump power

No significant differences between groups in reaction time were detected prior to the supplementation period or after supplementation ($p > 0.05$). No significant differences between groups in vertical jump power were detected prior to the supplementation period ($p > 0.05$). A significant main time effect was observed for vertical jump power ($p < 0.0002$) that demonstrated an increase at day 15 for all time points. However, there were no differences between groups.

Discussion

The purpose of this study was to investigate the effects of ATP supplementation on postworkout ATP metabolite levels as well as power output, muscle activation, and excitability during and following a repeated sprint bout. Our primary findings were that ATP supplementation demonstrated greater metabolite changes following the sprinting bout in the oral ATP supplementation in comparison to placebo. Moreover, ATP supplementation resulted in improved repeated sprint ability and these effects were amplified with the degree of fatigue induced by the sprinting bout. Our findings suggest that ATP increased muscular excitability in early bouts and prevented the decline in later bouts.

Effects of oral ATP on metabolite concentration levels

The current study indicates that chronic combined with acute oral supplementation is able to sustain ATP levels in the blood following an intense exercise bout. Previous research has only looked at a resting condition; however, the mechanism by which oral ATP is taken up would not be detected in a static state. Thus, it is possible that red blood cells in portal blood rapidly increase the uptake of adenosine as ATP is degraded in the blood and enhance its endogenous synthesis.

Rapaport and Fontaine [20,21] were able to show that the systemic and oral administrations of ATP result in the expansions of liver, blood (red blood cells), and blood plasma (extracellular) pools of ATP. Following the digestive process, ATP degrades rapidly into adenosine and inorganic phosphate,

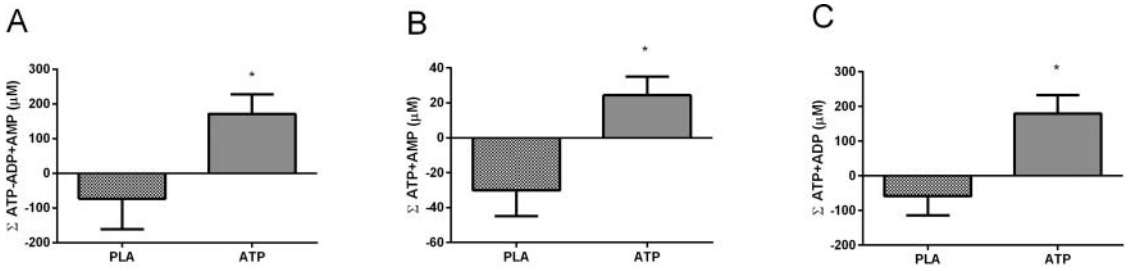

Figure 2. Delta change values for the sum of (A) ATP, ADP, and AMP; (B) ATP and AMP; and (C) ATP and ADP from day 15 baseline to day 15 postsprint. *Indicates $p <$ 0.05 different from placebo group.

Table 2. Peak power outputs and resultant effect sizes differences between days 1 and day 15.

	Placebo			ATP		
PPO (W)	Pre	Post	ES	Pre	Post	ES
Bout 1	897.4 ± 154.0	903.6 ± 152.1	0.04	944.0 ± 189.7	909.8 ± 150.7	−0.18
Bout 2	872.8 ± 164.4	870.3 ± 170.8	−0.01	893.3 ± 123.6	899.7 ± 130.0	0.04
Bout 3	798.6 ± 157.9	797.9 ± 166.9	0.00	825.8 ± 145.5	860.8 ± 144.7	0.23
Bout 4	726.8 ± 172.5	759.3 ± 177.3	0.18	785.6 ± 161.6	833.5 ± 176.6	0.29
Bout 5	684.9 ± 193.7	698.9 ± 172.0	0.07	712.1 ± 194.2	759.0 ± 164.6	0.24
Bout 6	647.2 ± 184.9	673.1 ± 182.0	0.13	649.5 ± 171.3	708.9 ± 162.4	0.34
Bout 7	624.0 ± 182.1	642.6 ± 171.6	0.10	617.0 ± 155.1	672.6 ± 155.0	0.35
Bout 8	567.8 ± 163.6	600.6 ± 151.4	0.20	560.4 ± 128.3	663.0 ± 128.7*	0.79
Bout 9	535.1 ± 168.2	585.1 ± 145.6	0.29	563.4 ± 136.9	620.4 ± 116.4	0.41
Bout 10	527.6 ± 153.4	571.0 ± 140.4	0.28	556.3 ± 144.1	647.2 ± 137.9*	0.63

ATP = adenosine-5′-triphosphate, PPO = peak power output, ES = effect size; W = Watts.
*Indicates $p < 0.05$ different from baseline.

which then are absorbed in the small intestine, entering the portal circulation where they are incorporated into liver ATP pools [24]. Previous studies showed a lack of intact bioavailability for orally administrated ATP at rest [16,17]. However, we posit that ATP supplementation and its resultant metabolites work indirectly to increase the synthesis of ATP during high-intensity exercise. Specifically, chronic oral administration of ATP increased portal vein ATP concentration and nucleoside uptake by erythrocytes, which resulted in an increase in ATP synthesis in the erythrocytes [24], independent of an increase in systemic plasma ATP. Therefore, chronic oral supplementation increases the capacity to synthesize ATP within erythrocytes in response to supplementation, without elevating the resting systemic concentration in plasma [24]. Thus, we suggest an indirect mechanism of ATP supplementation whereby chronic oral administration results in a greater capacity of erythrocytes to synthesize and thus sustain plasma ATP concentrations in response to the hypoxic perturbations triggered by high-intensity exercise.

This study was able to demonstrate that after 15 days of oral ATP supplementation, 30 minutes post-high-intensity exercise, the absolute change for combined ATP, ADP, and AMP concentrations was significantly greater than the placebo group. Though the exact mechanism of oral ATP absorption is still not fully understood, ATP and its metabolites may stimulate intracellular ATP by interacting with specific ATP and adenosine receptors on cell surfaces through a signaling effect [25]. The combination of high-intensity exercise and oral ATP may create a higher demand for increasing ATP synthesis in red blood cells, allowing a higher concentration of blood ATP in these subjects.

Effects of ATP supplementation on peak power and muscle excitability

The ability to rapidly produce and sustain power is the hallmark of many sports [26,27]. ATP's mechanisms of action are well established in the literature and could lead to enhancements in performance. First, ATP is carried in red blood cells and when oxygen concentration is low in a working muscle region, the red blood cell deforms, resulting in a cascade of events that lead to ATP release via pannexin channels [5,6]. Once released, ATP binds to purinergic (P2X and P2Y) receptors on endothelial cells [7,8]. Binding results in the endothelial cells releasing nitric oxide via endothelial nitric oxide synthase, prostacyclin, and endothelium-derived hyperpolarizing factor [28], all 3 of which affect the smooth muscle of the vasculature via cyclic guanosine monophosphate, cyclic adenosine monophosphate, and hyperpolarization, respectively [5]. These 3 factors cause vasodilation and the increased diameter of the blood vessel allows for an increase in blood flow and nutrient and oxygen delivery ultimately maintaining energy status in the cell particularly under fatiguing contractions [29].

Initial studies on the impact of nutrient supplementation to boost ATP levels were conducted by Maresh et al. [30]. The researchers administered a cocktail containing amino acid, choline, and ribose to subjects prior to a 30-second Wingate bout. Not directly administering ATP this cocktail did increase preworkout plasma levels of ATP and mean power output on the Wingate. Building on their work, researchers began to supplement directly with ATP. The first studies with direct oral ATP found low to nonsignificant performance improvements likely due to dose, amount, and timing of ingestion relative to exercise. However, Jordan et al. observed increases in within-group 1 repetition maximum (nearly 7%) and lifting volume (18.5%) with 225 mg of ATP in only 14 days of supplementation with untrained individuals who did nothing in the interim [10].

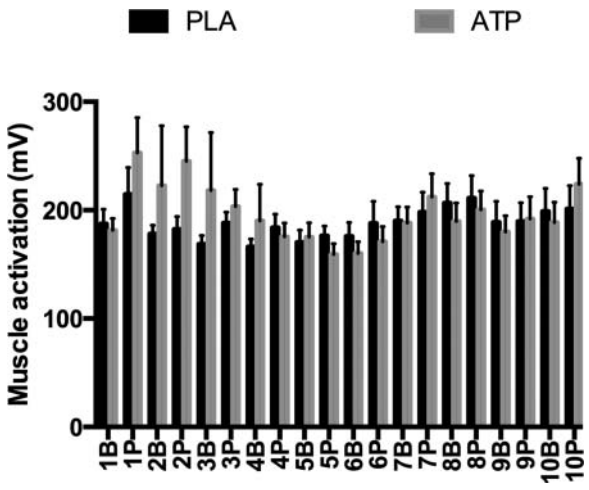

Figure 3. Skeletal muscle activation as measured through electromyography in millivolts on days 1 and 15 from sprints 1 through 10 (B = baseline, P = postexercise).

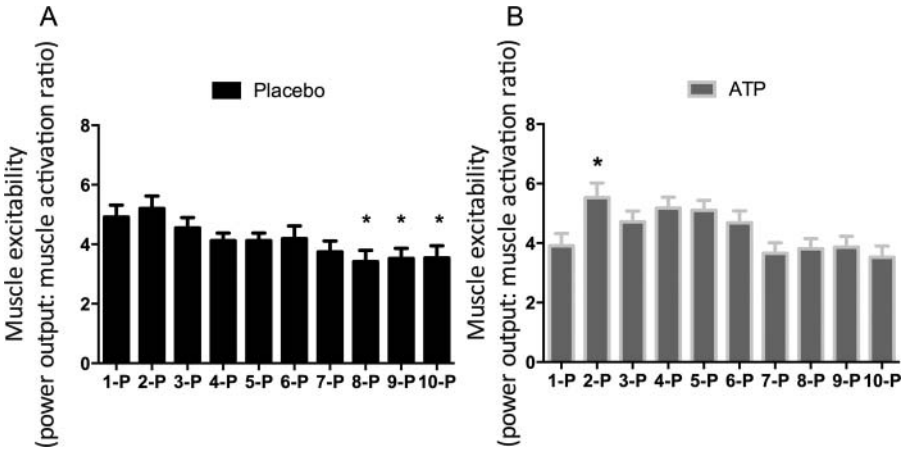

Figure 4. Muscle excitability. ME significantly decreased in the control group in later bouts, whereas PEAK ATP increased ME in early bouts and prevented the decrease in later bouts. *Indicates $p < 0.05$ different from sprint 1 (P = postexercise).

Recently, at a higher dose (400 mg), Rathmacher et al. observed improvement in low peak torque in set 2 with 400 mg of ATP compared to the placebo in only 15 days [11]. More recently, our lab observed increases in strength and hypertrophy when giving ATP over a 12-week period of time, both supplemented with ATP daily and 30 minutes prior to exercise [13]. In the current study, we found that oral ATP supplementation sustained a relatively higher power output on the final sprints of our exhaustive exercise bout but not during the initial sprints. This is in contrast to supplements like creatine that enhance power both under resting and fatigued conditions. These results may be explained by the enhanced blood flow and oxygen delivery as described previously [12]. In addition, ATP actually is known to upregulate Acetylcholine (ACH) receptors, so it is plausible that chronic ATP supplementation may increase skeletal muscle excitability/activation by making the cell more sensitive to ACH release [31,32].

In the current study, there were no significant differences in muscle activation; however, an improvement in the ratio between power and muscle activation (muscle excitability) was seen. The mechanism by which ATP enhanced performance and muscle excitability is likely due to peripheral and not central mechanisms. Based on previous research, these mechanisms might occur via increases in blood flow during fatiguing conditions [12]. Future research should seek to elucidate the predominant mechanisms by which oral ATP supplementation is working.

Effects of oral ATP on reaction time and vertical jump power

Previous research from our lab demonstrated an increase in vertical jump power following 12 weeks of ATP supplementation in combination with a controlled training program [13]. The current study did not confirm the beneficial effect of ATP on vertical jump power and the different outcome may be explained by shorter supplementation duration (12 weeks vs 2 weeks) and the lack training intervention. Lastly, the current investigation found no differences between ATP or placebo supplementation for reaction time. Oral ATP alone may not be effective in tasks that are not demanding enough to deplete ATP levels.

Limitations

Due to the heightened level of intensity, energy demand, and movement pattern of the Wingate test protocol, the data may not be directly applicable to all types of sports. In addition, though the blood data show that orally supplemented ATP is bioavailable under hypoxic conditions, further research is still warranted to determine minimum supplementation periods and needed doses to elicit increases in power and performance. Finally, it is conceivable that ATP supplementation that blunts fatigue may have lowered or altered blood lactate levels. However, because we did not include this variable, we feel that it is a limitation to our study.

Conclusion

Oral ATP administration increased postexercise ATP, ADP, and AMP levels and improved repeated sprint ability, and these effects were amplified with the degree of fatigue induced by the sprinting bout. Muscle excitability increased in the ATP group during early bouts, and ATP supplementation prevented the decrease in muscle excitability observed in the placebo group during later bouts. Athletes and strength and conditioning practitioners looking to enhance repeated sprint ability (i.e., basketball, hockey, etc.) may see beneficial effects in power and muscle excitability from supplementing with oral ATP.

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Author contributions

M.P., J.A.R., J.M.W., R.P.L., and R.J. designed the study. M.H.S., K.A.S., and J.M.P. supervised and trained subjects and participated in data acquisition. J.A.R. performed the ATP and ATP metabolite analyses. D.S. performed the statistical analysis. All authors discussed the results. R.J. and J. M.W. wrote the article.

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