TOPICS IN EXERCISE SCIENCE AND KINESIOLOGY

Process of Science

THE EFFECTS OF ACUTE RAUWOLSCINE (α-YOHIMBINE) INGESTION ON REPEATED WINGATE SPRINT PERFORMANCE IN HEALTHY MALES

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ABSTRACT

Topics in Exercise Science and Kinesiology Volume 4: Issue 1, Article 1, 2023. Background: Rauwolscine (RW), also known as α-Yohimbine, is an α-2-adrenergic receptor antagonist which possesses sympathomimetic properties. RW is commercially sold in pre-workout and energy supplements. However, the ergogenic potential of RW has not been determined. The purpose of this study was to investigate the effects of acute RW supplementation on repeated sprint performance. Methods: Healthy male participants (*n*=12) completed 3 × 15-second Wingate anaerobic tests (WAnT) separated by 2 minutes of active recovery. Blood lactate (La) was collected before exercise (Pre) and immediately following exercise (Post). Mean power, peak power, fatigue index, heart rate (HR), and rate of perceived exertion (RPE) were taken immediately after each WAnT. *Point of application #1:* Acute RW supplementation does not result in the enhancement of repeated anaerobic sprint performance. *Point of application #2:* HR and RPE are not altered during repeated sprints with RW ingestion. *Point of application #3:* RW ingestion results in higher La levels post-exercise despite no changes in fatigue index.

KEY WORDS: Wingate, Lactate, Power Output, Sympathomimetic

INTRODUCTION

Rauwolscine (RW), or α-yohimbine, is a chemical found in the bark of the *Pausinystalia Yohimbe* tree and other native plants in central and western Africa. In traditional folk medicine, the ground bark has been ingested to improve feelings of virility, energy, and lower fatigue (24). Recently, alkaloids extracted from the bark, including RW, have been shown to act as potent α2-adrengenic receptor antagonists which act to override the negative feedback mechanism for norepinephrine (NE) release from pre-synaptic sympathetic neurons (21). This results in greater NE spillover and exacerbated sympathetic (i.e. fight or flight) responses (12). As a consequence, RW and related alkaloids have been reported to incite alterations in blood flow (4, 20), blood

pressure (18), arousal (11), and fuel utilization (13). However, whether these effects translate to superior exercise performance remains largely unknown. Although RW remains virtually unstudied in the context of exercise, multiple investigations have shown that Yohimbine, a stereoisomer of RW which has similar properties, may enhance performance (2, 15). For example, recent evidence from our lab showed that an acute dose of Yohimbine HCl increased power output and catecholamine responses during anaerobic exercise in physically active females (2). Furthermore, the majority of RW research is decades old and focuses more on mechanisms than application (3, 12, 17, 21, 22). It is currently unknown if RW possesses similar ergogenic effects, especially in males. Therefore, the purpose of this study was the investigate the effects of acute RW ingestion on repeated Wingate sprint performance in physically active males. We hypothesized that RW ingestion would increase power output, HR, RPE, and blood lactate (La) during repeated sprints compared to a placebo.

METHODS AND RESULTS

Using a double-blinded, crossover, counterbalanced study design, physically active males (n=12; age= 20.7 yrs ± 1.9, height= 178.7 cm ± 9.7, body mass= 77.8 kg ± 11.4) completed two repeated sprint test visits each with a differing treatment: 1) Placebo (gluten-free corn starch), 2) RW (2 mg/dose; standardized to 90% RW; T6 Supplements, GA, USA). Prior to each visit, participants were asked to refrain from consuming caffeine, nicotine, and alcohol at least 12 hours before testing and vigorous exercise 24 hours prior. Participants were encouraged to keep normal dietary and sleep habits prior to each visit. For each treatment, participants ingested a single dose of the corresponding supplement 20 minutes prior to exercise. Capsules for the treatments were size and color matched in coded opaque bags to blind participants and researchers. The code was disclosed by an independent researcher not involved in data collection at the end of the study. For the testing portion of each visit, participants completed 3 × 15 second Wingate anaerobic tests (WAnT) on a cycle ergometer (Velotron, Recermate Inc, Seattle, WA, USA) as previously described by our lab (1, 2, 16). Briefly, participants were outfitted with a chest-strap HR monitor (Polar, NY, USA) and a pre-exercise blood lactate (La) measurement was taken via a lancet-induced capillary blood sample and portable lactate meter (Lactate Plus, Nova Biomedical, Biomedical, Waltham, MA, USA). Following this, participants completed a 5-minute cycling warm-up at 50 watts to a pace of 60 bpm via metronome. Immediately after, participants completed the 3 × 15 s WAnTs separated by 2 minutes of active recovery. For each WAnT, participants began by pedaling slowly against an unloaded resistance that was then ensued by a 10 s lead-in phase to allow for the attainment of maximal pedal rate. At the end of the lead-in phase, resistance was immediately applied at 7.5% of the participant's body mass and they pedaled as hard and as fast as possible for the 15 s duration. After each WAnT, rate of perceived exertion (RPE;1–10 scale), HR, and performance measures were documented. Upon the cessation of exercise (Post), another blood La measurement was repeated. All participants were verbally encouraged during the testing. Visits were separated by a minimum of a 48-hour washout period. All data were analyzed using Jamovi software (Version 0.9). A 2 × 3 [treatment × test] repeated measures ANOVA was used to analyze peak power, mean power, fatigue index, HR, and RPE. A 2 × 2 [treatment × time] repeated measures ANOVA was used to analyze La. Cohen's d effect sizes (d) were used to estimate magnitudes of

change and were interpreted with the following cutoff points: <0.2- trivial, 0.2—small; 0.5—moderate; 0.8—large [11]. All data are presented as mean ± standard deviation (SD). Significance was set a p≤ 0.05 *a priori*.

POINTS OF APPLICATION

Acute RW supplementation does not result in the enhancement of repeated anaerobic sprint performance.

Mean and peak power (watts) measurements were documented at the end of each 15 s WAnT. Findings revealed no main effect for treatment ($p=0.795$; $\eta^2 < 0.001$) or treatment × test interaction ($p=0.070$; $\eta^2 = 0.006$) effect for mean power. However, there was a main effect for test ($p<0.001$; $\eta^2 = 0.104$) whereby mean power in WAnT 1 was significantly higher than WAnT 2 ($p=0.002$; $d=1.11$) and WAnT 3 ($p<0.001$; $d= 1.18$). Furthermore, mean power during WAnT 2 was significantly higher than WAnT 3 ($p=0.002$; $d= 0.84$). Findings revealed no main effect for treatment ($p=0.821$; $\eta^2 < 0.001$) or treatment ×test interaction ($p=0.648$; $\eta^2 = 0.001$) for peak power. However, there was a main effect for test ($p<0.001$; $\eta^2 = 0.056$) whereby peak power in WAnT 1 was significantly higher than WAnT 2 ($p=0.012$; $d= 0.63$) and WAnT 3 ($p=0.013$; $d=1.09$).

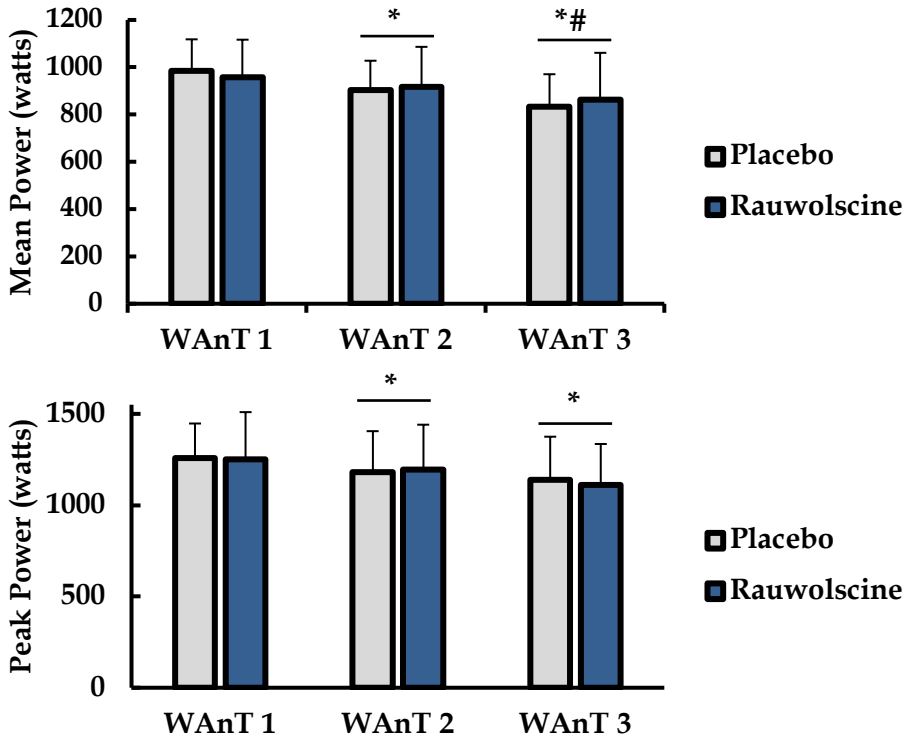

Figure 1. Comparison of mean power (watts; *top*) and peak power (watts; *bottom*) over 3 × 15 s Wingate anaerobic tests (WAnT 1, WAnT 2, WAnT 3) between placebo (grey) and rauwolscine (blue). Data are presented as mean ± SD. * indicates significantly different from WAnT 1 (p≤0.05). # indicates significantly different from WAnT 2 (p≤ 0.05).

Counter to our hypothesis, these data suggest RW has minimal to no effect on mean and peak power output during repeated sprints. The reasons for lack of differences between placebo and RW are not fully clear but may be due to dosing. Studies in animal models have shown effective dose ranges from 0.5-4.0 mg/kg (3, 7, 23). Assuming a typical human body mass of 70 kg, this would equate to an absolute dosage of 35-280 mg. Participants consumed 2 mg in the present study according to the manufacturer's instruction which may have not been a high enough dose to impart any ergogenic effects. However, it should be noted that other investigations showing performance enhancement using the enantiomer yohimbine utilized similar doses albeit the properties of oral yohimbine and RW have yet to be directly compared in humans (2). Furthermore, purity of RW was not directly measured although third party tested independent of the manufacture. Currently, the optimal dosage of RW and safety/efficacy of using relative dosages to body mass (i.e. mg/kg) in humans is unknown. However, higher doses of *yohimbe* alkaloids have been well-documented to be anxiogenic and cause overstimulation (5, 9). Thus, the use of larger doses may not be practical but the delineation of the tolerability of RW and exercise in humans is imperative for future research. These findings suggest that coaches, practitioners, and athletes should not use RW in attempts to boost anaerobic performance although future research with varying dosages and larger sample sizes will be needed to develop a more comprehensive conclusion.

HR and RPE are not altered during repeated sprints with RW ingestion.

At the end of each 15 s WAnT, HR (bpm) and RPE (1-10 scale) were collected. For HR, there was no main effect for treatment ($p=0.382$; $\eta^2 = 0.002$) nor was there an interaction ($p=0.095$; $\eta^2 = 0.009$) effect between treatment × test. However, there was a main effect for test ($p=0.033$; $\eta^2 = 0.070$). Specifically, HR during WAnT 3 was significantly higher than WAnT 1 ($p=0.031$; $d=0.41$) and WAnT 2 ($p=0.022$; $d=0.59$). For RPE, there was no main effect for treatment ($p=0.788$; $\eta^2 < 0.001$) nor was there an interaction ($p=0.815$; $\eta^2 = 0.001$) effect between treatment × test. However, there was a main effect for test ($p<0.001$; $\eta^2 = 0.433$). Specifically, RPE during WAnT 1 was significantly lower than WAnT 2 ($p<0.001$; $d=1.14$) and WAnT 3 ($p<0.001$; $d=1.80$). Furthermore, RPE during WAnT 2 was significantly lower than WAnT 3 ($p<0.001$; $d=0.66$).

Overall, HR and RPE increased with successive tests, but RW treatment did not alter either outcome compared to placebo. Given the reported sympathomimetic properties of RW (23), these findings were surprising and underlying mechanisms for the lack of changes with treatment are not entirely clear. However, these findings may manifest in the maximal nature of the exercise test. RW has been shown to increase blood pressure and other markers of sympathetic stimulation at rest and during recovery (6, 14, 17). But since participants were giving all-out effort, exercise-induced sympathetic output was likely high independent of treatment which could have preponderated any HR or RPE alterations by RW. This is further supported by previous work showing that other stimulants (i.e. caffeine) modulate sympathetic pressor responses at rest but are less impactful on hemodynamic responses during exercise (19). While speculative, RW may act in a similar manner whereby sympathomimetic actions during exercise may be mitigated by the overwhelming responses to high-intensity exercise. Collectively, these data suggest that coaches and athletes should not expect alterations in HR or RPE during repeated sprints and that theoretically, the integrity of HR and RPE measurements for determining exercise intensity likely remain intact with RW supplementation.

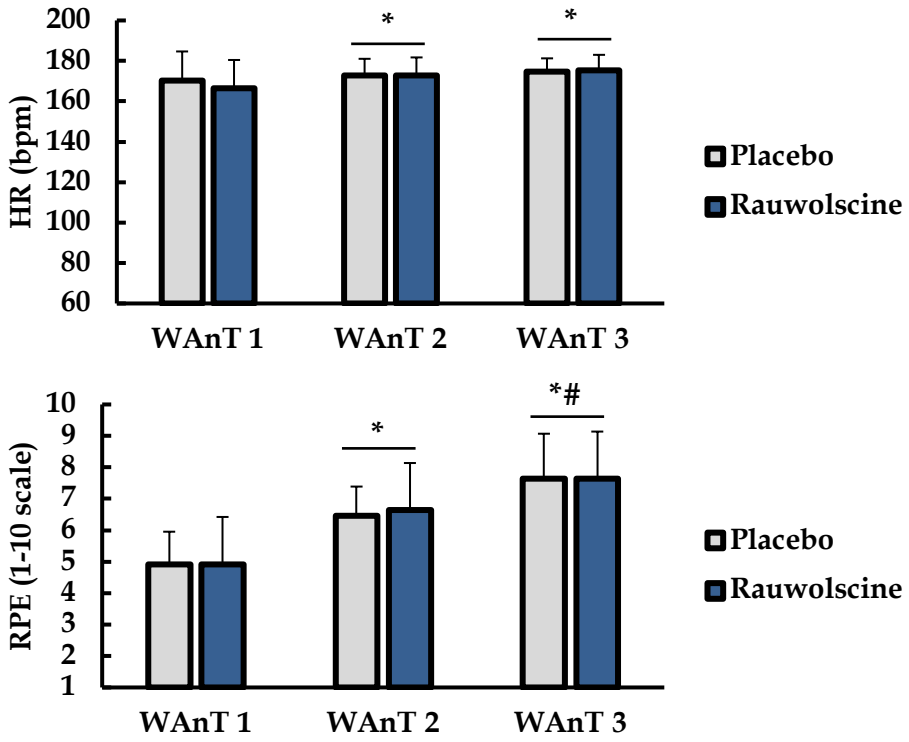

Figure 2. Comparison of HR (bpm; *top*) and RPE (1-10 scale; *bottom*) over 3 × 15 s Wingate anaerobic tests (WAnT 1, WAnT 2, WAnT 3) between placebo (grey) and rauwolscine (blue). Data are presented as mean ± SD. * indicates significantly different from WAnT 1 ($p \leq 0.05$). # indicates significantly different from WAnT 2 ($p \leq 0.05$).

RW ingestion results in higher La levels post-exercise despite no changes in fatigue index.

At the end of each 15 s WAnT, fatigue index (watts·s^{-1}) was recorded. Before (Pre) and immediately following the completion of exercise (Post), blood La (mmol/L) was also collected. Fatigue index was derived from (peak power- minimum power)/duration of sprint which yields information on power output loss during the sprint. For fatigue index, analysis revealed no main effect for treatment ($p=0.598$; $\eta^2 = 0.002$) nor was there an interaction effect ($p=0.760$; $\eta^2 = 0.002$). A main effect for test existed ($p=0.020$; $\eta^2 = 0.040$) and fatigue index during WAnT 1 was significantly lower than WAnT 2 ($p=0.027$; $d=0.46$). and WAnT 3 ($p=0.023$; $d=0.82$). For La, there was a main effect for time ($p<0.001$; $\eta^2 = 0.897$) and treatment ($p=0.008$; $\eta^2 = 0.010$). But there was not an interaction ($p=0.145$; $\eta^2 = 0.003$) effect between time × treatment. More specifically, La levels for the Pre timepoint were significantly lower than Post regardless of treatment ($p<0.001$; $d=5.78$). Furthermore, La was significantly higher with RW treatment versus placebo at the Post timepoint ($p=0.037$; $d=0.83$).

Even though performance and fatigue index remain unchanged, La levels were higher with RW ingestion compared to placebo following the completion of exercise. Although we hypothesized that La would increase to a greater degree with RW ingestion, it was expected that increases would occur as a consequence of enhanced power output and performance. Results suggest that RW resulted in a greater La level in the absence of performance enhancement which cannot be explained fully from our data alone. As previously mentioned, RW and related alkaloids induce heightened sympathetic responses

which lead to increased catecholamine release (23). Catecholamines have been well described to mediate fiber-type recruitment and glycolytic activity (10). RW may have resulted in greater catecholamine release compared to placebo resulting in a more reliance on energy from glycolysis thus resulting in greater La levels. The lack of performance changes may also be partially due to the greater La formation as well. It is plausible that pH alterations from La formation may have resulted in some level of fatigue thereby negating benefits from RW. While La has been previously shown to decrease with YHM ingestion in females, it is possible that current disparities may manifest in biological sex differences in that males have been implicated to rely more heavily on glycolysis during maximal exercise (22). While increased lactate concentrations are associated with declines in exercise performance acutely (8), is also believed to possibly contribute to anabolic signaling that mediates muscle protein synthesis. While speculative, the increased lactate concentrations following RW ingestion may be become deleterious in an acute sense if exercise persists but may also be advantageous during training focusing on maximizing skeletal muscle adaptations. However, the reader is cautioned that no long-term outcomes were determined by the current investigation and chronic regimens and possible habituation to RW have yet to be studied. Future research should investigate the possible ergogenic effects of chronic RW in combination with other anaerobic training (i.e., resistance training) on muscle mass accumulation. Since La responses during exercise are highly dependent on training status and sample population, these factors may alter responses to RW supplementation differently which warrants considerable study for the future.

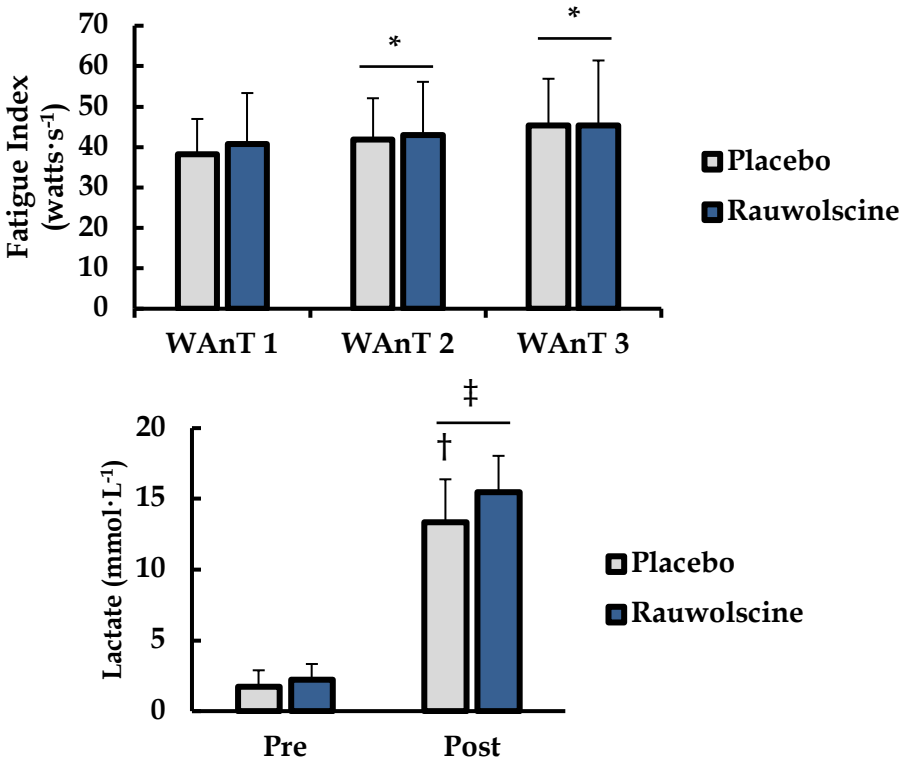

Figure 3. Comparison of Fatigue index (watts·s^{-1}; *top*) over 3 × 15 s Wingate anaerobic tests (WAnT 1, WAnT 2, WAnT 3) and lactate (mmol·L^{-1}; *bottom*) pre- and post-exercise between placebo (grey) and rauwolscine (blue). Data are presented as mean ± SD. * indicates significantly different from WAnT 1 ($p \leq 0.05$). ‡ indicates significantly different from Pre ($p \leq 0.05$). † indicates significantly different from Rauwolscine ($p \leq 0.05$).

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EQUIPMENT UTILIZED

(T6 Supplements, GA, USA).

(Velotron, Racermate Inc, WA, USA)

(Lactate Plus, Nova Biomedical, Biomedical, MA, USA).

Polar H10 Heart Rate Monitor Chest Strap (Polar Electro, NY, USA)