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Effect of lactate supplementation on $\dot{V}O_{2peak}$ and onset of blood lactate accumulation: A double-blind, placebo-controlled trial

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Background: Professional and recreational athletes use ergogenic aids to enhance aerobic performance, facilitate training adaptations, and reduce recovery time. While the ergogenic effect of commonly marketed and commercially available ergogenic aids (i.e., sodium bicarbonate and β-alanine) have been investigated extensively, the effectiveness of lactate supplementation on aerobic capacity markers (i.e., $\dot{V}O_{2peak}$ and the onset blood lactate accumulation) has not been thoroughly investigated. **Objective:** The purpose of this study was to examine the effect of lactate supplementation on $\dot{V}O_{2peak}$ and the onset blood lactate accumulation (OBLA). **Methods:** Eighteen healthy individuals (14 men and 4 women, age: 24 ± 5 years, height: 171 ± 7 cm, body mass: 76 ± 17 kg) participated in a double-blind-placebo-controlled study and were randomly assigned to one of 2 groups: placebo (PLA, n = 8), or supplement (SUP, n = 10) consisting of a combination of magnesium lactate dihydrate and calcium lactate monohydrate. Prior to and following supplementation, participants performed a cycling graded exercise test to determine $\dot{V}O_{2peak}$, time to exhaustion, power output, $\dot{V}O_2$ and % $\dot{V}O_{2peak}$ associated with OBLA. **Results:** There were no statistically significant differences between PLA and SUP in direct markers of aerobic capacity (all p > .05). Heart rate at OBLA was reduced by 6 ± 6% in the PLA group post-supplementation compared to pre-supplementation (p = .03). **Conclusions:** Lactate supplementation did not present an advantage over a placebo in improving aerobic capacity. The results from this study support those by previous investigators suggesting that there is no physiological rationale for using lactate supplementation to improve aerobic capacity and/or performance.

Keywords: lactate supplementation, ergogenic aids, exercise testing, cycling, aerobic capacity

Introduction

Professional and recreational athletes use nutritional ergogenic aids to enhance aerobic performance, facilitate training adaptations, and reduce exercise recovery time. The performance enhancing effects of nutritional ergogenic aids, in the form of chemical buffers, have been attributed to increased buffering capacity in an attempt to regulate the concentration of H^+ ions. Commercially available forms of nutritional ergogenic aids include sodium bicarbonate, β-alanine, and lactate supplements. An extensive review by McNaughton, Siegler, and Midgley (2008) highlighted more than 12 investigations demonstrating that chronic sodium bicarbonate supplementation improved resistance exercise performance and aerobic endurance. Improvements of cycling time-trial performance were also observed following chronic β-alanine supplementation (Bellinger, Howe, Shing, & Fell, 2012; Bellinger & Minahan, 2016). Taken together, results from these previous investigations demonstrate that the ingestion of sodium bicarbonate and β-alanine improve sport performance due to their ability to buffer the increasing concentration of hydrogen (H^+) ions during sustained physical activity. Although these nutritional strategies seem attractive because of their potential ergogenic effect, reported side effects such as paresthesia (Hobson, Saunders, Ball, Harris, & Sale, 2012) and gastrointestinal distress (Siegler, Marshall, Bishop, Shaw, & Green, 2016) limit their applications.

While the use of sodium bicarbonate and β-alanine as ergogenic aids have been investigated extensively (Bellinger et al., 2012; Bellinger & Minahan, 2016;

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Jourkesh, Ahmaidi, Keikha, Sadri, & Ojagi, 2011; McNaughton et al., 2008), there is mixed support for the use of lactate supplementation to improve aerobic performance (Bryner, Hornsby, Chetlin, Ullrich, & Yeater, 1998; Morris, Shafer, Fairbrother, & Woodall, 2011; Northgraves, Peart, Jordan, & Vince, 2014; Peveler & Palmer, 2012; Swensen, Crater, Bassett, & Howley, 1994). According to the lactate shuttle (LS) theory, lactate can be utilized in the Cori cycle of the liver and/or skeletal muscle, to synthesize glycogen or be converted into pyruvate and enter aerobic metabolism (Brooks, 2000). Specifically, the LS theory proposes that lactate formed by skeletal muscles can be oxidized within the mitochondria involving the presence of mitochondrial lactate dehydrogenase – the protein responsible for lactate oxidation and reduction. Thus, lactate can be directly utilized as an energy source by skeletal muscle mitochondria in situations of physical exercise. Indeed, Morris et al. (2011) reported that consuming 120 mg/kg of body mass of lactate increased blood bicarbonate levels and improved exercise performance during high-intensity cycling ergometry to exhaustion. However, previous investigators who examined the effect of lactate supplementation on simulated cycling time trial performances, have been unable to provide evidence to support the LS theory (Bryner et al., 1998; Northgraves et al., 2014; Peveler & Palmer, 2012; Swensen et al., 1994). These mixed findings may be due to differences in exercise intensity, duration, and testing modalities (i.e., single exercise bouts versus repeat trials, time to exhaustion versus time trials). For example, a self-pacing strategy commonly utilized during cycling time trials would require participants to self-select cadence/pedaling rate. Pedaling rate (if only a single crank length is used) has been shown to account for the majority of the variability in metabolic cost during cycling (McDaniel, Durstine, Hand, & Martin, 2002). Consequently, it is important to account for the contribution of pedaling rate during cycling trials. It remains unclear if previous investigators, who implemented cycling as a test modality (Morris et al., 2011; Northgraves et al., 2014; Peveler & Palmer, 2012), allowed their study participants to self-select pedaling rates and/or gear ratios since those pedaling rates or gear ratios were not reported.

Given the link between aerobic capacity and aerobic performance has been demonstrated to be strong enough for previous investigators to search specifically for training interventions optimizing increases in aerobic capacity to improve aerobic performance (Fay, Londeree, LaFontaine, & Volek, 1989; Lehmann, Berg, Kapp, Wessinghage, & Keul, 1983; Maughan & Leiper, 1983; Midgley, McNaughton, & Wilkinson, 2006; Saltin & Astrand, 1967), the effectiveness of lactate supplementation on direct aerobic capacity markers (i.e., $\dot{V}O_{2peak}$ and the onset blood lactate accumulation) has not been thoroughly investigated. Therefore, the purpose of this study was to examine the ergogenic effect of lactate supplementation on $\dot{V}O_{2peak}$ and the onset blood lactate accumulation (OBLA). We hypothesized that lactate supplementation would improve $\dot{V}O_{2peak}$ and delay OBLA during a graded exercise test (GXT).

Methods

Participants

Eighteen healthy participants (14 men: age = 23 ± 4 years, height = 175 ± 5 cm, body mass = 84 ± 11 kg, $\dot{V}O_{2peak}$ = 51 ± 10 ml/kg/min; 4 women: age = 25 ± 7 years, height = 162 ± 3 cm, body mass = 61 ± 3 kg, $\dot{V}O_{2peak}$ = 46 ± 4 ml/kg/min) volunteered for this investigation. These volunteers consisted of individuals characterized by de Pauw et al. (2013) as being recreationally active under performance level 2 in the classification of participant groups in sport science research exhibiting $\dot{V}O_{2peak}$ values ranging from 45–54.9 ml/kg/min, being consistently active for at least 1 year but less than 5 years (Rhea, 2004). All participants were free of food allergies and were not on any medications. The participants were asked to abstain from exercise, alcohol, and supplement use 48 hours prior to each experimental visit. Experimental procedures used in this investigation were approved by the Central Connecticut State University Human Studies Council (F15060). All participants provided written informed consent prior to engaging in experimental procedures. An *a priori* power analysis performed using G*Power (Version 3.1.9.2; www.psycho.uni-duesseldorf.de/abteilungen/aap/gpower3/) utilizing an estimated effect size of .39 (corresponding to a partial eta squared of .13), with an α level of .05 and a power of .8 (1–β) estimated that a total sample size of 16 participants (8 in each group) would suffice (Faul, Erdfelder, Lang, & Buchner, 2007).

Experimental design

This study utilized a double-blind-placebo-controlled experimental design. Participants and testers assessing experimental outcomes were blinded to the treatment until the study ended. Participants were instructed to arrive for testing in a rested state having refrained from strenuous exercise at least 1 day before each experimental visit. Participants were also instructed to refrain from ingesting any form of food 1 hour before each experimental visit and to maintain their normal diet and recreational activity between experimental visits. On the first experimental visit, participants reported to

the laboratory for anthropometric measurements and performed a baseline GXT on an electronically braked cycle ergometer (Corival cpet, Lode BV, Groningen, Netherlands). On their second experimental visit, participants were randomly assigned to consume either a lactate supplement (SUP) or a placebo (PLA) by coin toss. The SUP group included seven males and three females, and the PLA group included seven males and one female (participant characteristics are presented in Table 1). Participants in the SUP group received a lactate supplement containing a combination of calcium lactate monohydrate and magnesium lactate dihydrate (SportsLegs; Sports Specifics, Inc., Chagrin Falls, OH, USA) according to the manufacturer's recommended dosage of 1 capsule (containing 371.7 mg of lactate) per 22.7 kg of body mass. In contrast, participants in the PLA group received a placebo containing defatted rice bran flour. Both the lactate supplement and placebo were ingested on an empty stomach 1 hour before performing the GXT according to manufacturer's direction. The participants were scheduled for post-testing no less than 3 days apart from their first visit to ensure that they were well recovered and free from exercise-induced muscular soreness and/or fatigue.

Graded exercise test

Participants performed a continuous graded exercise test on an electronically braked ergometer (Corival cpet, Lode BV, Groningen, Netherlands) where aerobic performance in the form of peak oxygen consumption ($\dot{V}O_{2peak}$) and onset of blood lactate accumulation OBLA were determined. Inspired/expired gasses were measured using open circuit spirometry to determine respiratory exchange ratio (RER), $\dot{V}CO_2$, and $\dot{V}O_2$, averaged over 15-s intervals (True Max 2400, Parvo Medics, Sandy, UT, USA). After a 5-min warm-up at 100 W, the GXT commenced with the participants pedaling at 90 rpm for 3 min at 100 W. The workrate was then increased 25 W every 3 min until volitional failure. $\dot{V}O_2$ and RER were calculated at 15-s intervals, and $\dot{V}O_{2peak}$ was calculated as the average of the highest two consecutive $\dot{V}O_2$ measurements. During each stage, heart rate, blood lactate, and rating of perceived exertion (RPE) were measured. Heart rate was measured continuously using a heart rate monitor (Polar FT1, Polar Electro, Kempele, Finland). Blood was drawn during the last minute of each stage and lactate concentrations were measured using a Lactate Plus lactate analyzer (Lactate Plus, NOVA Biomedical, Waltham, MA, USA). A fixed blood lactate value of 4.0 mmol/L was used as an indicator of OBLA. This fixed lactate measurement was utilized based on the validity of this index in different exercise modes (Denadai, Gomide, & Greco, 2005; Heck et al., 1985; Jones & Doust, 1998). Additionally, OBLA coupled with the GXT 3-min stage increment has been demonstrated to produce valid results of physiological response to incremental exercise in individuals of different training status (Bentley, McNaughton, & Batterham, 2001; Coyle, 1995; Denadai, Figueira, Favaro, & Gonçalves, 2004).

Table 1
Participant descriptive characteristics (mean ± SD)

	SUP (n = 10)	PLA (n = 8)
Age (years)	24 ± 5	23 ± 4
Height (cm)	168 ± 5	173 ± 7
Body mass (kg)	73 ± 19	81 ± 13
Body mass index (kg/m^2)	26 ± 6	27 ± 3

Statistical analyses

Separate 2 (group) × 2 (time) mixed repeated measures analysis of variance (ANOVA) procedures were performed on $\dot{V}O_{2peak}$, time to exhaustion (TTE), OBLA power, HR at OBLA, $\dot{V}O_2$ at OBLA, and RPE at OBLA. If any of the ANOVA procedures indicated a significant main effect or significant interaction effect, then subsequent *post hoc* pair-wise comparisons (Bonferroni correction for multiple comparisons) were used to determine where differences occurred. Data were analyzed using SPSS statistical analysis software (Version 22; SPSS, Inc, Chicago, IL, USA). Effect sizes were reported by partial eta squared (η_p^2), where magnitudes of .02, .13, and .26 were interpreted as small, medium, and large effects, respectively (Bakeman, 2005; Cohen, 1988). All data are presented as mean ± standard deviation and α was set to .05.

Results

Outliers were assessed by boxplot, normality was assessed using the Shapiro-Wilk's normality test and homogeneity of variances was assessed by Levene's test. There were no outliers, data was normally distributed (all $p > .05$) and there was homogeneity of variances (all $p > .05$).

Pre- and post-supplementation exercise parameters for the SUP and PLA groups are presented in Table 2. There were no statistically significant main effect of group in $\dot{V}O_{2peak}$ ($F(1, 16) = 0.28$, $p = .61$, $\eta_p^2 = .02$), TTE ($F(1, 16) = 0.24$, $p = .63$, $\eta_p^2 = .02$), OBLA power ($F(1, 16) = 0.58$, $p = .46$, $\eta_p^2 = .04$), $\dot{V}O_2$ at OBLA ($F(1, 16) = 1.15$, $p = .30$, $\eta_p^2 = .07$), and RPE at OBLA ($F(1, 16) = 0.07$, $p = .79$, $\eta_p^2 = .01$). Similarly, there

Table 2
Pre- and post-supplementation exercise parameters (mean ± SD)

	SUP (n = 10)		PLA (n = 8)	
Exercise parameter	Pre	Post	Pre	Post
$\dot{V}O_{2peak}$ (ml/kg/min)	51 ± 11	51 ± 8	48 ± 5	50 ± 5
TTE (s)	1169 ± 256	1191 ± 231	1241 ± 107	1213 ± 104
OBLA power (W)	133 ± 32	150 ± 37	153 ± 26	153 ± 32
HR at OBLA (beats/min)	151 ± 19	159 ± 9	157 ± 13	148 ± 14*
$\dot{V}O_2$ at OBLA (ml/kg/min)	31 ± 7	33 ± 10	29 ± 3	28 ± 4
RPE at OBLA	11 ± 3	13 ± 3	11 ± 2	11 ± 2

Note. SUP = supplement group; PLA = placebo group; TTE = time to exhaustion; OBLA = onset of blood lactate accumulation; HR = heart rate; RPE = rating of perceived exertion. *Significantly different from pre-supplementation PLA ($p < .05$).

were no statistically significant main effect of time in $\dot{V}O_{2peak}$ ($F(1, 16) = 1.34$, $p = .26$, $\eta_p^2 = .08$), TTE ($F(1, 16) = 0.03$, $p = .87$, $\eta_p^2 = .002$), OBLA power ($F(1, 16) = 4.30$, $p = .06$, $\eta_p^2 = .21$), $\dot{V}O_2$ at OBLA ($F(1, 16) = 0.13$, $p = .73$, $\eta_p^2 = .01$), and RPE at OBLA ($F(1, 16) = 2.38$, $p = .14$, $\eta_p^2 = .13$). However, there was a significant group × time interaction on HR at OBLA ($F(1, 16) = 7.58$, $p = .01$, $\eta_p^2 = .32$). Subsequent post hoc analyses indicated that the HR at OBLA was reduced by 6 ± 6 % in the PLA group post-supplementation compared to pre-supplementation ($p = .03$).

Discussion

Our main finding was that lactate supplementation had no ergogenic effect on $\dot{V}O_{2peak}$ and OBLA. To the best of our knowledge, this study was the first to examine the effect of lactate supplementation on direct measures of aerobic capacity. Although OBLA power in the SUP group was not statistically different post-supplementation compared to pre-supplementation, the higher OBLA power observed post-supplementation allowed us to speculate on a trend of increasing OBLA power. This potential difference in OBLA power may explain anecdotal reports of lactate supplementation effectiveness. Taken together, the results of this study support those by previous investigators (Northgraves et al., 2014; Peveler & Palmer, 2012) who reported no significant improvements in aerobic performance following supplementation with similar forms of exogenous lactate. Peveler and Palmer (2012) found no improvements to 20-km time trial cycling performance following lactate supplementation. Northgraves et al. (2014) examined the relationship between lactate supplementation and a 40-km cycling time trial performance. Interestingly, while there was a significant reduction in heart rate at OBLA in the placebo group, there were no measurable improvements in $\dot{V}O_{2peak}$ and OBLA. Because the placebo effect, ranging from magnitudes of 1% to 6%, has been implicated in improvements in sports performance (Beedie & Foad, 2009; Beedie, Foad, & Coleman, 2008; Clark, Hopkins, Hawley, & Burke, 2000; Kalasountas, Reed, & Fitzpatrick, 2007), it is plausible this reduction in heart rate, could be a result of a placebo effect rather than an ergogenic effect.

The low dosage of lactate supplementation in our present study and previous investigations (Northgraves et al., 2014; Peveler & Palmer, 2012) might explain the lack of detectable changes in aerobic capacity and/or performance. Participants in our study and those of previous investigations (Northgraves et al., 2014; Peveler & Palmer, 2012) consumed a mean dosage equivalent to 17.3 mg/kg of body mass of lactate. In contrast, previous investigators (de Salles Painelli et al., 2014; Morris et al., 2011) who administered higher dosages have reported slight but significant improvement in exercise performance (Morris et al., 2011) and increases in extracellular buffering capacity (de Salles Painelli et al., 2014). Specifically, Morris et al. (2011) administered 120 mg/kg of body mass of lactate and observed significant increases in time to exhaustion and total work following lactate supplementation. These investigators (Morris et al., 2011) reported concomitant increases in blood bicarbonate levels, peaking at 80–100 min following ingestion of calcium lactate, and speculated that the elevated blood bicarbonate levels likely contributed to improve exercise performance. de Salles Painelli et al. (2014) investigated the effect of two different doses (150 and 300 mg/kg of body mass) on blood pH, bicarbonate and lactate responses, and reported very discrete increases in blood pH and bicarbonate, without improvements in repeated high-intensity cycling performance. Taken together, these

previous investigations support the use of a higher lactate supplementation dosage (de Salles Painelli et al., 2014; Morris et al., 2011; Van Montfoort, Van Dieren, Hopkins, & Shearman, 2004). Implicitly, a future direction for our laboratory will be to consider the contributions of dosages different from the manufacturer's recommendation and the timing of ingestion during exercise testing, in order to determine the optimal dosage of lactate supplementation on metabolic cost during submaximal cycling. Feasibly, it is likely that athletes would use this supplement in accordance to the manufacturer's recommendation when consuming supplements based on safety and prohibitive cost of supplementation.

Other plausible factors contributing to the non-significant aerobic capacity responses observed in our present study and those of previous investigators (Northgraves et al., 2014; Peveler & Palmer, 2012) might be the lack of lactate dehydrogenase located within the mitochondria, and lack of bioavailability after supplementation. Indeed, previous investigators were unable to detect lactate dehydrogenase within the mitochondria in both human and animal studies (Bryner et al., 1998; Northgraves et al., 2014; Peveler & Palmer, 2012; Sahlin, Fernström, Svensson, & Tonkonogi, 2002; Swensen et al., 1994; Yoshida et al., 2007). Similarly, previous investigators were unable to observe increases in bioavailability following supplementation with exogenous lactate (Brouns, Fogelholm, van Hall, Wagenmakers, & Saris, 1995; Péronnet, Burelle, Massicotte, Lavoie, & Hillaire-Marcel, 1997; Swensen et al., 1994). Taken together, the findings of these previous investigations provide strong evidence for the lack of support for the use of oral lactate as an energy substrate during high intensity exercise.

A potential limitation in our investigation was the utilization of the fixed 4.0 mmol/L lactate level as a marker of OBLA. It has been argued that this method of identifying the lactate inflection point for OBLA results in poor reproducibility of the anaerobic threshold (Aunola & Rusko, 1984) as it is insensitive to interindividual training status (Faude, Kindermann, & Meyer, 2009). However, previous investigators (Denadai et al., 2005, 2004; Heck et al., 1985; Jones & Doust, 1998) have reported good validity of work-rate at a blood lactate concentration of 4 mmol/L as an indirect index of maximal lactate steady state ($MLSS_{work\text{-}rate}$) in different exercise modes, with experimental results indicating that OBLA is a valid indirect index of $MLSS_{work\text{-}rate}$ during cycling exercise regardless of the aerobic training status of the participants. Implicitly, these equivocal reports on the validity of our current method for determining OBLA limits its applicability as an indicator of working capacity and thus warrants cautious interpretation of our results.

Conclusion

In conclusion, the main finding of our study was that lactate supplementation did not present an advantage over a placebo in improving aerobic capacity in healthy individuals. The results from this study support those by previous investigators suggesting that there is no physiological rationale for using the manufacturer's recommended dosage of lactate supplementation to improve aerobic capacity and/or performance.

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Conflict of interest

There were no conflicts of interest.

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