

Dietary nitrate enhances power output during the early phases of maximal intensity sprint cycling

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Abstract

Ingestion of dietary nitrate has shown to improve endurance exercise performance, however effects on short-term maximal intensity exercise are currently unknown. The study aimed to investigate whether supplementation with dietary nitrate has an ergogenic effect on sprint cycling exercise. Following a baseline trial, ten recreationally active males consumed, in a counterbalanced fashion, 0.14 L of either nitrate-rich beetroot juice (8 mmol.NO₃-1) or placebo, the day before and 40 min prior to performing a maximal 30 s Wingate cycling test. Following nitrate supplementation there was a 'possible' increase in mean power (7.95 ± 0.55 w.kg-1) during the 30 s sprint compared to control (7.78 ± 0.61 w.kg-1) and placebo conditions (7.63 ± 0.91 w.kg-1) (47% chance; 90% CI: -0.09 - 0.43 w.kg-1). On further analysis, via division into 5 s phases; participants experienced 'likely' increases in mean power during 5-10 s (77% chance; 90% CI: -0.01 - 0.65 w.kg-1) and 10-15 s (81% chance; 90% CI: 0.05 - 0.57 w.kg-1) phases following nitrate supplementation compared to control and placebo. The consumption of dietary nitrate, seemingly enhanced power output between 5 to 15 s of maximal cycling, this occurred despite nitrate having no distinctive effect on overall cycling performance.

Keywords: cycling, sprint, dietary nitrate, power assessment, exercise physiology

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Introduction

Nitrate Oxide (NO) has a number of physiological roles and is primarily known for the maintenance of normal blood pressure and protection of the cardiovascular system (Zand et al., 2011). NO also effects muscle contractility, calcium and glucose homeostasis and myocytes differentiation (Dejam et al., 2004). Several studies have found that nitrate is a modulator of resting blood pressure and energy metabolism (Bailey et al., 2010; Kapil et al., 2010; Larsen et al., 2011). Researchers have also examined the effects of ingesting nitrate rich foods, such as green leafy vegetables and particularly beetroot, on the physiological response to exercise (Larsen et al., 2007, 2010; Bailey et al., 2009, 2010; Lansley, et al., 2011a; Cermak et al., 2012). It has been suggested that a diet rich in nitrate is a contributing factor to reduced O₂ cost during submaximal exercise (Larsen et al., 2007; Bailey et al., 2009), contributing to a higher power output ratio (Larsen et al., 2010; Lansley et al., 2011a, 2011b).

Nitrate supplementation has been reported to extend time to exhaustion by 15-25% during 70-80% gaseous exchange threshold intensity work-rate exercise (Bailey et al., 2009, 2010; Lansley et al., 2011b). However, the use of such time-to-exhaustion protocols have been questioned on the basis that they possess low ecological validity since the demands of a typical endurance event are not sufficiently simulated (Schabort et al., 1998; Atkinson & Nevill, 2001). In relation to the athletic setting, a more reliable and externally valid means of assessing performance involves protocols in which athletes are required to complete a fixed amount of work or to cover a given distance in the shortest possible time (time-trials) or to complete a maximal amount of work in a specific time-period (Foster et al., 1993; Schabort et al., 1998; Atkinson & Nevill, 2001). Such time-trials were utilised by Lansley et al. (2011a) and Cermak et al. (2012) who both found time improvements in 4 km, 10 km and 16.1 km cycling maximal efforts in a nitrate condition opposed to a placebo condition. Furthermore nitrate ingestion has been found to improve the performance of repeated 500 m rowing ergometer sprints (Bond et al., 2012).

These aforementioned performance improvements are thought to be influenced by the breakdown of nitrate (NO₃) to bioactive nitrate (NO₂) and consequently nitric oxide (NO) (Cosby et al., 2003). NO₂ and NO may modulate the efficiency of mitochondrial respiration (Larsen et al., 2011) and the cost of adenosine triphosphate (ATP) muscle force production (Bailey et al., 2010). It is thought enhanced muscle force production efficiency alongside improved delivery of O₂ to working muscles that are hypoxic; may affect the progression of fatigue and improve exercise performance (Bailey et al., 2010). ATP turnover rate is dependant via the Actomyosin ATPase and Ca²⁺ ATPase pathways, which effect sarcoplasmic reticulum Ca²⁺ pumping and membrane depolarisation, which is responsible for a small (7%) fraction of ATP turnover (Barclay et al., 2007). Intervention of nitrate supplementation may slow cross bridge cycling kinetics, modulating ATP cost of force production (Galler et al., 1997; Heunks et al., 2001), which maintains

submaximal power output for longer, with a reduction of Phosphocreatine (PCr) breakdown (Bailey et al., 2010). This could prove useful in track cycling events such as the 200 m sprint, 500 m and 1000 m time trials which have both anaerobic and aerobic demands (Craig & Norton, 2001).

The purpose of this study was to investigate the effect of nitrate supplementation on peak power (w.kg-1), mean power (w.kg-1) and fatigue index % during a 30 s Wingate maximal cycling sprint in recreationally active males. We hypothesised that nitrate ingestion would increase mean power produced and negate fatigue during a 30 s Wingate cycling sprint test.

Methodology

Participants

Ten recreationally active males (Mean \pm SD, age: 20.4 ± 0.5 years, stature: 1.82 ± 0.06 m; body mass: 75.7 ± 10.8 kg) who engaged in regular exercise and sporting activities volunteered to participate in the study, none of which were tobacco smokers (Bailey et al., 2010; Larsen et al., 2010). Following a health screening questionnaire all participants provided written informed consent to participate in the study, which was approved by the local ethics committee in line with the Helsinki Declarations for research with human volunteers.

Experimental Protocol

The study implemented a randomised single-blind repeated measures crossover design. Participants initially attended a familiarisation session where the Wingate test procedures were described and practiced. On this occasion, handlebar, seat and foot clip position on the Monark 874e (Monark Exercise AB, Varberg, Sweden) were adjusted and recorded to suit each participant to ensure accuracy. The 30 s Wingate test has previously been used to assess the power generating capabilities of elite cyclists and has been found to distinguish competitive performance ability (Tanaka et al., 1993).

For the baseline testing assessment, all participants were weighed to calculate the required mass for the Wingate test (7.5% body mass load) (Wilson et al., 2009). Participants then performed a standardised warm-up consisting of five min of pedalling (60 rpm, 60 w). Immediately following this, the 30 s Wingate test was completed, this was initiated by participants increasing rpm to maximum revolutions and the ergometer weighted load being applied (Wilson et al., 2009). Each participant received positive verbal encouragement throughout to maintain motivation and promote maximal exertion (Bampouras & Marrin, 2009). After the test was complete, the load was lifted and the participant cycled at low intensity with no weight to cool down and regulate blood flow.

Following completion of the baseline assessment, participants were then randomly assigned to ingest either 0.14 L (8 mmol.NO₃.Day⁻¹) of concentrated BR (Beet It, James White Drinks Ltd., Ipswich, UK; Cermak et al., 2012) or 0.14 L of concentrated low calorie blackcurrant juice with negligible NO₃ content as a placebo (Bailey et al., 2010). The administered placebo (blackcurrant juice) has been commonly used in research assessing effects of dietary nitrate ingestion on performance (Bailey et al., 2009, 2010; Bond et al., 2012). Treatments were taken one day before, with the final dose 40 min prior to the experimental trial (Larsen et al., 2010), after this there was a washout period of seven days (Larsen et al., 2010). Previous acute dosing protocols utilising similar total nitrate content have been found to be equally effective at eliciting physiological effects and increasing performance than more prolonged supplementation protocols (Vanhatalo et al., 2010; Kelly et al., 2013). The participants were then assigned the opposite supplement depending on the randomised order given, with the same supplementation protocol taking place. Participants were instructed to consume the exact same diet over the three testing periods (Bailey et al., 2010; Lansley et al., 2011). All assessments were performed at the same time of the day; 10 am \pm 2 h for purposes of reliability (Bailey et al., 2009, 2010).

Physical Activity and Dietary Standardisation

Participants were told to keep their weekly training schedule as consistent as possible over the course of the study. This typically involved a combination of resistance training, endurance training and sporting activities with a common frequency of four to five total exercise bouts per week. In addition participants were instructed to arrive in the laboratory in a fully rested and hydrated state, and avoid strenuous exercise 24 h prior to testing (Cermak et al., 2012). They were instructed to maintain their habitual diet over the course of the study. Participants were told to record their food intake 24 h prior to the first assessment so this could be repeated for subsequent trials, and were also told to refrain from the consumption of caffeine 6 h and alcohol 24 h prior to testing (Lansley et al., 2011b; Cermak et al., 2012). All participants were also asked to refrain from using antibacterial mouthwash and chewing gum during supplementation periods as these are known to eradicate the oral bacteria that are necessary for the conversion of nitrate to nitrite (Govoni et al., 2008).

Statistical Analysis

All result data is expressed as mean \pm standard deviation unless stated otherwise. Monark anaerobic test software was used to assess peak power and mean power (w.kg⁻¹) and fatigue index % (% difference between peak- and end-of-test power) for the 30 s Wingate test. Mean power was analysed for the whole trial and via division into 6 x 5 s phases of the test. Data was analysed using methods that reported the uncertainty of outcomes as 90% confidence intervals, making probabilistic magnitude-based inferences concerning the true

value of outcomes using the methods described by Batterham and Hopkins (2006). This method establishes the smallest practical effect, allowing the researcher to qualify the probability of a worthwhile effect with inferential descriptors to aid interpretation (Rowlands et al., 2008). Smallest practical effect was calculated for Wingate mean power (w.kg-1) from the product of 0.2 (which represents the smallest standardised (Cohen) change in mean) times the between-participant standard deviation for baseline values of all the participants. From using the smallest practical effect value, magnitude and inference of the change in mean power was then analysed according to procedures developed by Batterham and Hopkins (2006). From these procedures, 90% confidence intervals (CI) for the changes in mean power following ingestion of nitrate are calculated. In addition practical likelihoods of harm or benefit caused to each dependent variable (mean power) from the independent variable (supplement) are established based on percentage boundaries; 0 to 0.5% indicated most unlikely; 0.5% to 5% indicated very unlikely; 5% to 25% indicated unlikely; 25% to 75% indicated possibly; 75% to 95% indicated likely; 95% to 99.5% indicated very likely; and > 99.5% indicated most likely.

Results

Peak Power and Fatigue Index

Peak power was 10.90 ± 1.21 w.kg-1 at baseline, 10.83 ± 1.28 w.kg-1 following nitrate ingestion and 11.07 ± 1.70 w.kg-1 following ingestion of placebo. Inferences confirmed that the slight mean differences between the conditions were 'negligible' or 'trivial' with no meaningful beneficial effect following supplementation of nitrate. Fatigue index was $52.6 \pm 5.4\%$ at baseline, $53.1 \pm 6.7\%$ following nitrate ingestion and $57.6 \pm 12\%$ following ingestion of placebo. Inferences once again confirmed the 'negligible' mean differences between the conditions.

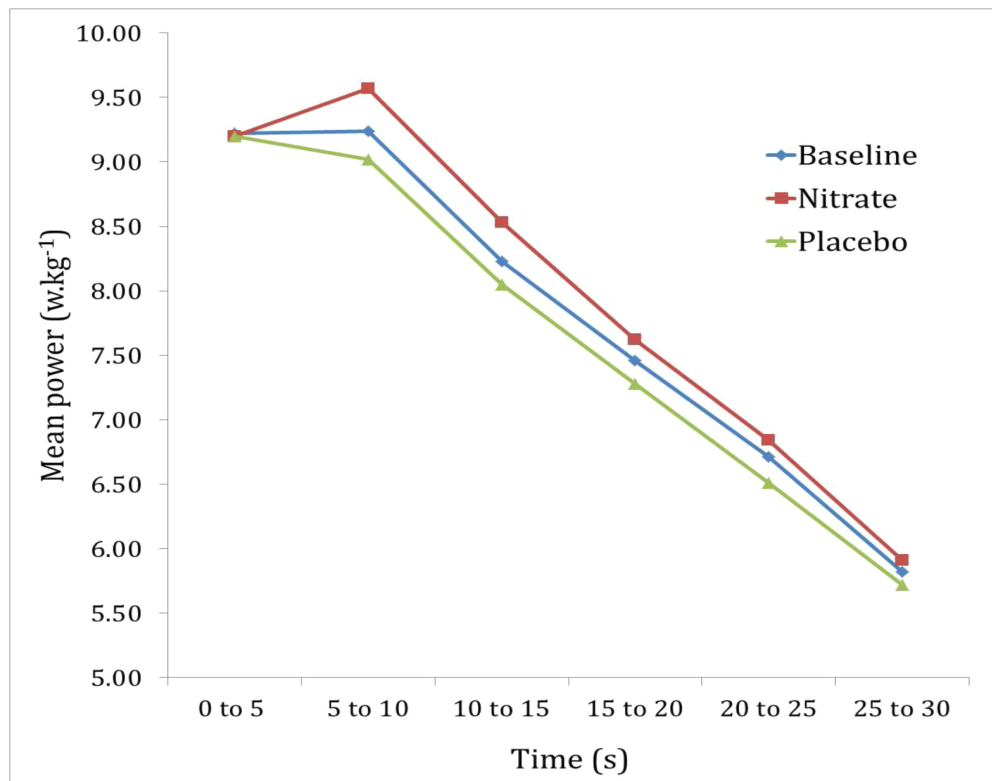
Mean Power

Following nitrate supplementation there were 'possible' and 'likely' increases in whole trial mean power (7.95 ± 0.55 w.kg-1) during the 30 s sprint compared to baseline (7.78 ± 0.61 w.kg-1) and placebo (7.63 ± 0.91 w.kg-1) respectively (Table 1). The power trace shows analysis of the trial, via division into 5 s phases (Figure 1). The trace shows a tendency for higher mean power following nitrate ingestion in all phases except for the initial 0 to 5 s of the test. However, practically meaningful increases in power output following nitrate ingestion in comparison to baseline were only 'likely' to occur between 5 to 10 s and 10 to 15 s of the test.

Table 1. Effect of nitrate on mean power output (w.kg⁻¹) during the 30 s Wingate test

Phase of 30 s sprint	Comparison	Mean effect (90% CI) w.kg ⁻¹	% Chance / Qualitative inference
0-5 s	Nitrate vs. Baseline	-0.01 (-0.37 to 0.35)	Unclear
	Nitrate vs. Placebo	0.01 (-0.83 to 0.85)	Unclear
5-10 s	Nitrate vs. Baseline	0.32 (-0.01 to 0.65)	83% Increase likely
	Nitrate vs. Placebo	0.55 (0.2 to 0.9)	97% Increase very likely
10-15 s	Nitrate vs. Baseline	0.31 (0.05 to 0.57)	89% Increase likely
	Nitrate vs. Placebo	0.49 (0.11 to 0.87)	94% Increase likely
15-20 s	Nitrate vs. Baseline	0.16 (-0.11 to 0.43)	60% Increase possible
	Nitrate vs. Placebo	0.34 (-0.05 to 0.73)	84% Increase likely
20-25 s	Nitrate vs. Baseline	0.14 (-0.17 to 0.45)	55% Increase possible
	Nitrate vs. Placebo	0.33 (0.00 to 0.66)	87% Increase likely
25-30 s	Nitrate vs. Baseline	0.09 (-0.25 to 0.43)	Unclear
	Nitrate vs. Placebo	0.19 (-0.14 to 0.52)	61% Increase possible
Whole trial (0-30 s)	Nitrate vs. Baseline	0.17 (-0.09 to 0.43)	63% Increase possible
	Nitrate vs. Placebo	0.32 (-0.01 to 0.65)	85% Increase likely

Figure 1. Power trace comparing mean power (w.kg⁻¹) for every 5 s of the 30 s Wingate tests in baseline, supplementation and placebo conditions



Discussion

The principle finding of this study was that supplementation of 0.14 L (8 mmol.NO₃.Day⁻¹) of concentrated beetroot juice over a period of two days seemingly enhanced mean power output during the 5-15 s phase of the 30 s Wingate maximal cycling sprint test. This interesting finding occurred despite nitrate ingestion having no distinctive effect on mean power during the overall 30 s Wingate test. There has been evidence to suggest supplementation of nitrate can improve performance of high intensity exercise (Bailey et al., 2009, 2010; Larsen et al., 2010; Bond et al., 2012) which implements the use of the ATP-PC system. The two ATPase pathways: Actomyosin ATPase and Ca²⁺ ATPase are thought to have an increased efficiency under the influence of NO. Evidence suggests NO slows cross-bridge cycling kinetics (Galler et al., 1997) and reduces Ca²⁺ cycling (Viner et al., 2002) suggesting NO modulates ATP cost of force production. Phosphocreatine is also thought to be effected by resulting in a reversible impediment of creatine kinase via s-nitrosylation (Arstall et al., 1998) and reduced PCr degradation, suggesting high intensity exercise can be sustained for longer before these metabolites are depleted (Bailey et al., 2010). The power trace (Figure 1) suggests mean power was marginally higher under the influence of nitrate compared to baseline and placebo conditions, suggesting these efficiency mechanisms may have played a part in a small performance improvement. These small performance improvements were only visibly evident during the 5-15 s phase of the 30 s Wingate test, when the ATP-PC energy system is primarily utilised (Hargreaves et al., 1998). This could explain why no distinctive effect was found during the full 30 s Wingate test as primary liberation and subsequent depletion of ATP-PC occurred during the initial 15 s (Hargreaves et al., 1998). During the later phases of the maximal intensity cycle sprint, inferences show 'possible' and 'unclear' chances of nitrate positively increasing mean power, this can be further interpreted through the power trace (Figure 1), as the mean power results standardise across conditions as the test duration increases. A reason for this may be depletion of PCr stores, creating a slowing effect as the next phase of energy liberation is primarily sourced through the less efficient glycolytic energy system. The 'likely' (83-89% chance) performance increase during the 5-15 s period of the cycle sprint can be seen as a practically useful finding. This finding bears relevance to elite track cycling, as small margins (< 1% performance time) in elite sport can dramatically affect race placing within a field of competitors (Hopkins et al., 1999; Smith & Hopkins, 2011). Subsequently, an acute protocol of dietary nitrate ingestion, involving two boluses of 0.14 L (8 mmol.NO₃.Day⁻¹) of concentrated BR, taken 24 h and shortly (~ 40 min) before exercise can form a recommended practice for track cyclists.

There was an expectation that the nitrate intervention would positively influence fatigue index during the 30 s Wingate test as this measure is related to mean power. However only 'negligible' mean differences existed across conditions. It was thought that performance in this variable would be effected by ATP-PC system efficiency, and decline in PCr degradation previously reported through the mechanisms as

outlined for mean power (Bailey et al., 2010). These exercise efficiencies could have contributed to the maintenance of power throughout the test due to reduced PCr degradation (Bailey et al., 2010), slowing of cross bridge kinetics and reduced Ca²⁺ cycling (Galler et al., 1997; Viner et al., 2002) leading to lower fatigue index compared to baseline and placebo conditions, however this was not evident.

Nitrate supplementation was found to have no distinct impact on peak power during the maximal intensity cycle sprint. It is theorised that nitrate supplementation lowers blood pressure (Larsen et al., 2007; Bailey et al., 2009) which is thought to be due to vasodilator activity (Cosby et al., 2003). Increased blood flow to skeletal muscle could promote optimal levels of nutrients and oxygen (Cabrales et al., 2006), leading to an improved contractile state, promoting increased muscle force. Nitrate supplementation has shown to improve performance in maximal exercise (Larsen et al., 2010; Bond et al., 2012). These findings are based on improved mechanism efficiency during high intensity exercise however; this may not be meaningful to peak power as it is the single highest power value recorded during the whole test, not the efficiency of exercise throughout. These assumptions were not supported by peak power results, indicating vasodilation and mechanisms found to improve ATP and PCr efficiency following nitrate supplementation had no effect on peak power. Limitations were evident within the study, in an attempt to enhance ecological validity participants were allowed to consume their normal diet 24 h before the first testing session, and were asked to repeat the same diet before all following tests. These variables are difficult to control and require honesty from the participants. Failure to adhere to the restrictions could have led to inconsistency in performance, damaging the validity of test results. Another limitation could be the participant sample of recreationally active males. Arguably the use of trained or elite cyclists would have been more applicable as they possess more sport specific skills and experience which are reproducible during a 30 s Wingate test. This could lead to improved reliability of test results offering a realistic view of the possible effects of a nitrate intervention on anaerobic cycling performance.

Conclusion

The study aimed to investigate whether supplementation with dietary nitrate has an ergogenic effect on sprint cycling exercise. Following nitrate ingestion, there was a 'likely' chance of an increase in mean power during the 5-15 s phase of the maximal intensity sprint, this occurred despite nitrate having no distinctive effect on overall 30 s cycling performance. The findings lead support to the theory that nitrate supplementation can improve mean power during high intensity exercise, and potentially be transferred into a sporting context. Even small increases in exercise performance could represent a meaningful marginal difference between success and failure within elite sport. It is possible nitrate could emerge as an ergogenic aid for maximal intensity sprint cycle events as cyclists seek to gain any tangible advantage over their competitive opposition. Further research is required to support the observed effects, and possibly recognise

nitrate as an ergogenic aid for anaerobic cycling activity. To enhance the validity, practicality and reliability of future research a participant cohort of trained sprint cyclists is suggested.

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