The effect of acute taurine ingestion on 3km running performance in trained middle distance runners

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Abstract

Limited research exists examining the effect of taurine (TA) ingestion on human exercise performance. The aim of this study was to investigate the effect of acute ingestion of 1000 mg of TA on maximal 3km time trial (3KTT) performance in trained middle distance runners (MDR). Eight male MDR (Mean ±SD: age, 19.9 ± 1.2 yr, body mass, 69.4 ± 6.6 kg, height, 180.5 ± 7.5 cm, 800m personal best time: 2:01.0 ± 5.3 sec) completed TA and placebo (PL) trials one week apart in a double blind, randomised, crossover designed study. Participants consumed TA or PL in capsule form on arrival at the laboratory followed by a 2-hour ingestion period. At the end of the ingestion period participants commenced a maximal simulated 3KTT on a treadmill. Capillary blood lactate was measured pre- and post-3KTT. Expired gas, heart rate (HR), ratings of perceived exertion (RPE), and split times were measured at 500m intervals during the 3KTT. Ingestion of TA significantly improved 3KTT performance (TA: 10:46.6 ± 52.8s and PL: 10:58.5 ± 58.2s) ($p = 0.013$) equating to a 1.7% improvement (range -0.34 to 4.24%). Relative oxygen uptake, HR, RPE nor blood lactate differed between conditions ($p = 0.803$, $p = 0.364$, $p = 0.760$ and $p = 0.302$, respectively). Magnitude-based inference results assessing the likeliness of a beneficial influence of TA were 99.3%. However, the mechanism responsible for this improved performance is unclear. TA's potential influence on exercise metabolism may involve interaction with the muscle membrane the coordination or the force production capability of involved muscles. Further research employing more invasive techniques may elucidate TA's role in improving maximal endurance performance.

Key words: Oxygen uptake, ergogenic aids, time trial, endurance running

Introduction

The sulfonic amino acid taurine (TA), is found in its free form in large concentrations in both skeletal and cardiac muscle and brain tissue (Huxtable, 1992). The high content of TA within these major organs has stimulated much research examining its role in modulating several physiological actions including osmoregulation, calcium content regulation, oxidative stress and substrate oxidation, all of which are known to have particular relevance to exercise performance (for an extensive review refer to Huxtable, 1992). To date, very little research has been conducted on the effect of acute or prolonged TA ingestion on endurance performance in humans (Rutherford et al., 2010). Existing research has produced inconclusive evidence on the use of TA prior to exercise. Running (Lee et al., 2003) and cycling (Zhang et al., 2004) time to exhaustion has been shown to significantly improve following TA supplementation. However, acute TA administration demonstrated no benefit to cycling performance (Rutherford et al., 2010).

The precise mechanisms underpinning how TA may affect human endurance performance are still largely unclear (Galloway et al., 2008). It has previously been shown that endurance trained individuals have higher TA muscle content compared to their untrained counterparts (Blomstrand and Saltin, 1999; Graham et al., 1995), indicating the potential importance of TA in human exercise performance. However, differences in the uptake of TA into the muscle have been demonstrated between human (Galloway et al., 2008) and rodent (Yatabe et al., 2003) models preclude suggestions that human endurance performance is enhanced via increased force production resulting from enhanced calcium regulation in the sarcoplasmic reticulum (Zhang et al., 2004). Such a finding has only been demonstrated in an *in vitro* animal model (Bakker and Berg, 2002). The effect of TA

ingestion on the human neuromuscular system during exercise is yet to be investigated. It has also been suggested TA exerts metabolic effects via interaction with the muscle membrane, similarly this has not been confirmed or linked with improvements in exercise perforance. A further concept is acute TA ingestion may may enhance exercise performance by atentuating losses from the muscle during exercise given the decreased levels reported following endurance exercise in humans (Cuisinier et al., 2001, 2002; Graham et al., 1995; Ward et al., 1999). Furthermore, it is unclear whether TA alone can improve endurance performance as many studies have used TA co-ingested with other products (Alford et al., 2001; Geiss et al., 1994; Forbes et al., 2007), highlighting the requirement for more studies in humans.

Rutherford and colleagues (2010) have previously demonstrated TA ingestion to induce a metabolic effect, on fat oxidation during prolonged submaximal cycling exercise in trained individuals citing a likely effect of TA on the muscle membrane. However, this enhancememt in fat oxidation did not translate to enhanced exercise performance in a subsequent time trial. Moreover, the use of prolonged submaximal exercise before a time trial performance is not ecologically valid and may have potentially reduced the sensitivity of the time trial to detect differences between conditions. Furthermore, it is unclear whether taurine may influence a physiological effect when solely maximal endurance performance is assessed as none of the existing research in this area has employed such a design. Therefore, the aim of this study was to investigate the effect of acute TA ingestion on maximal exercise performance, oxygen uptake and rating of perceived exertion (RPE) during a simulated 3km time-trial (3KTT) on a treadmill.

Methods

Eight well-trained competitive male middle distance runners (Mean ± Standard Deviation: age, 19.9 ± 1.2 yr, body mass, 69.4 ± 6.6 kg, height, 180.5 ± 7.5 cm, sum of seven site skinfolds: 45.2 ± 5.9mm, 800m personal best time: 2:01.0 ± 5.3 sec) participated in the present study following the submission of their informed consent. Participant training at the time of recruitment consisted of a minimum of 45 miles of running per week including a minimum of two organised training sessions per week with their respective athletics clubs, Prior to participant recruitment ethical approval was obtained from the local ethics committee. All testing was conducted during their non-competitive phase of training.

Experimental Design

Participants reported to the laboratory on two occasions to complete self-paced maximal 3KTT, in a randomised, cross-over, double-blind design. A 3KTT was selected as this is the upper distance limit of the middle distance running discipline, The 3KTT have also been commonly used in the recent research literature investigating different interventions on endurance trained running populations (Rodriguez et al., 2007; Robertson et al., 2010; Julian et al., 2004). The 3KTT has previously been reported to have a coefficient of variation of 1.2% (Julian et al., 2004). Each 3KTT was separated by one week in order to allow a sufficient washout period following TA administration (Galloway et al., 2008). Test sessions were completed at the same time of day to account for the effects of circadian variation on human exercise performance (Atkinson and Reilly, 1996). Participants maintained their scheduled training in the week leading up to each test session. Participants refrained from any form of physical exercise, caffeine or pharmacological treatment for the 48-h preceding the trials.

Dietary intake 48-h before the initial test session was recorded. Participants were asked to replicate this pattern of consumption for the remaining experimental session.

On arrival at the laboratory, participants consumed either a placebo (PL) or a 1000 mg commercially available TA capsule (TA) (Holland and Barrett, UK). The TA dose was equivalent to the amount that is commonly ingested in a standard serving of Red Bull™ (Alford et al., 2001) and was the taurine dosage was equivalent to 2.5 times the maximum daily quantity reported in normal human dietary analysis (Laidlaw et al., 1990; Rana and Sanders, 1986; Shao and Hathcock, 2008). The placebo capsule was a blank capsule that had no content. A 2-hour ingestion period has previously been shown to be required to achieve peak plasma TA levels (Galloway et al., 2008). Therefore, Pre-3KTT Capsules were ingested whole with 250 ml water 2-hours before the start of testing. in order to achieve peak plasma TA at the start of the time trial. Participants completed a 10-minute standardised pace warm-up (2-minutes at 10km.h^{-1}, 3-minutes at 12km.h^{-1} and 5-minutes at 14km.h^{-1}. The standardised warm up was completed in the final 15-minutes of the 2-hour ingestion period. The 3KTT was completed on a Woodway Pro-Series treadmill (Woodway, Weil am Rhein, Germany) at a 0.0% gradient. The treadmill was maintained and calibrated in accordance with manufacturer guidelines. Participants were provided feedback on the distance covered during each 3KTT only and were not informed of overall performance time, or heart rate (HR) data, until after the completion of the second test session. All participants had extensive prior experience of high-speed treadmill running and prior experience of completing competitive 3km time trials and therefore effective personal pacing strategies. During the 3KTT participants adjusted pace via buttons located on the display of the treadmill. Participants were familiarised with how to adjust speed duirng the warm-up for each session and were permitted to adjust speed how and whenever they saw fit during the time trial. Respiratory gases (Medgraphics, CPX-D, St. Paul, MN, USA), HR (Polar A1 HR monitor, Polar Electro OY, Kempele, Finland) and RPE (Borg, 1998) were measured at 500m intervals. In addition, 500 m split times during the 3KTT and overall time to complete the time trial were recorded. Fingertip capillary blood lactate samples (Lactate Pro Analyser, Arkray Inc, Kyoto, Japan) were taken at the end of the 2-hour ingestion period before the 3KTT commenced and immediately after its completion.

Statistical analysis

All values presented are mean ± standard deviation. Normality of data was confirmed following assessmet of Q–Q plots. Paired t-tests were employed to analyse overall 3KTT performance and overall 3KTT speed between conditions. A 2 x 6 (condition vs. 500m interval during the 3KTT) general linear model repeated measures analysis of variance (ANOVA) with Bonferroni adjustment post hoc comparisons was used to analyse 500 m split times, HR, oxygen uptake, RPE and blood lactate data. A statistical significance level of $p< 0.05$ was selected to define statistically significant differences. Paired T-tests and ANOVAs were completed using Minitab 15 statistical software (Minitab Ltd., Coventry, UK). In accordance with recommendations for reporting the statistics in physiology related journals (Batterham and Hopkins, 2006; McGlory and Morton, 2010) confidence intervals (95% CI) and magnitude based inference values were calculated. For the primary outcome variable of exercise performance, differences in competitive world (IAAF, 2012) and English National (ESAA, 2012) male 3000m finals were calculated from the years 2010, 2008 and 2006. Meaningful worthwhile change was defined as the mean difference in performance time between individual positions for the first four finishers in these world

and national competitions (e.g. difference between 1st and 2nd, 2nd and 3rd and 3rd and 4th). Elite and National level performance differences between positions were 0.30 ± 0.11% and 0.44 ± 0.23%. To provide further assessment of the effect of acute TA ingestion on overall 3KTT performance the meaningful inference of the intervention was determined using the method of Hopkins (2002). To perform this analysis the p-value produced from the paired T-test on overall 3KTT performance was inserted into magnitude-based inference software (Hopkins, 2007).

Results

Differences in overall 3KTT performance between conditions (TA: 646.6 ± 52.8s and PL: 658.5 ± 58.2s) reached statistical significance ($p = 0.013$) (Fig. 1). Seven of the eight participants performed better in the TA trial which equated to a 1.7% improved performance over the PL trial (range -0.34 to 4.24%) which was 0.5% above the coefficient of variation for the 3KTT and greater than the defined minimal worthwhile change for related populations. The 95% confidence intervals for mean differences in overall performance time between TA and PL were -3.4 to -20.4s. Differences between conditions for 500m splits throughout the 3KTT (Fig. 2) did not reach statistical significance but demonstrated a tendency towards an effect ($p = 0.081$, $f = 4.140$). Relative oxygen uptake (Fig. 3), HR (Fig. 4) and RPE (Fig. 5) did not differ between conditions ($p = 0.803$, $f = 0.070$; $p = 0.364$, $f = 0.940$; and $p = 0.760$, $f = 0.100$, respectively). Blood lactate concentrations (Post 3KTT- TA: 10.48 ± 3.25, PL 9.39 ± 2.81mmol^{-1}) did not differ statistically between conditions ($p = 0.302$, $f = 0.214$). Magnitude-based inference results assessing the probability that acute TA ingestion is beneficial/trivial/harmful to overall 3KTT performance were 99.3/0.1/0.6% respectively.

Discussion

Overall 3KTT performance

The current study provides novel data for the literature regarding the effect of TA ingestion on human exercise performance. No study investigating the effect of TA ingestion alone on running performance has used a self-paced time-trial to assess performance or investigated the effect of TA on endurance trained participants (Lee et al., 2003; Zhang et al., 2004; Geiss et al., 1994; Alford et al., 2001). Overall, 3KTT performance was 11.9s quicker in the TA condition, with seven of eight participants demonstrating improvement in exercise performance. Similar research investigating time trial performance with TA administration in trained cyclists did not demonstrate a significant difference between conditions. The differences in overall time trial results between the current study and Rutherford and colleagues (2010) are likely due to methodological differences. Factors such as TA ingestion timing and exercise protocol may have contributed to whether or not TA enhanced endurance performance and to what extent. Rutherford and colleagues utilised a 1-hour ingestion period followed by a further 1.5-hour submaximal intensity ride before their cycling time trial. A 1-hour ingestion period has been shown to be insufficient for maximising plasma TA concentration, with 2-hours previously defined as optimal for achieving acute peak plasma TA concentrations (Galloway et al., 2008). It is physiologically plausible to suggest that a 2.5-hour period between ingestion and the start of the cycling time trial as well as conducting a submaximal intensity cycle in the intervening time may have dampened any influence the ingested TA may have had on time trial performance. Previously it has been demonstrated that both force production and neuromuscular activation

are decreased following 1–hour of submaximal cycling compared to pre-exercise measures suggesting decreased central drive (Lepers et al., 2002). Additionally, plasma TA levels have shown to decrease drastically at rest after peak levels have been reached (Galloway et al., 2008). The drop in plasma TA levels is between one third and one half of peak levels at the 1-hour period following peak levels following an acute single does of TA. How this rapid decline in peak plasma levels may influence exercise performance is uncertain. The current study involved an immediate maximal time trial performance following the designated TA ingestion period whereas Rutherford and colleagues assessed differences in substrate oxidation between conditions in the intervening 2.5-hours between ingestion and the start of the cycling time trial. Such a design may lack ecological validity for endurance performance assessment.

Similarly to the current study, research assessing endurance capacity rather than simulated time-trials (as in the current study) has also reported a positive effect of TA administration (Lee et al., 2003; Zhang et al., 2004). Zhang and colleagues (2004) utilised an incremental intensity cycle test, whereas Lee and colleagues (2003) employed a time to exhaustion run at 75% of $VO_{2\,max}$. The magnitude of the improvement in the TA condition in these studies was considerably greater than the current study perhaps due to the longer exercise duration. Additionally, the TA administration protocols used in these studies differed considerably from the current study. Supplementation of TA consisted of 6 g or 4 g per day for 7 (Zhang et al., 2004) or 14 days (Lee et al., 2003), respectively. These studies do not indicate how close to the start of the endurance assessment the final TA ingestion of the supplementation period occurred. Recent studies have indicated human TA supplementation over a similar time period does not increase muscle TA content and acute ingestion must be carefully timed to achieve peak levels (Galloway et al., 2008). Therefore, it is difficult to interpret if TA supplementation may have benefited exercise capacity in these two studies or whether other dietary factors may have influenced any response.

Research examining TA ingestion within energy drinks has shown to improve exercise performance. Exercise time to fatigue during incremental intensity cycling following a submaximal intensity ride (Geiss et al., 1994), submaximal endurance exercise time defined by HR (Alford et al., 2001) and the number of submaximal intensity bench press repetitions performed before volitional fatigue (Forbes et al., 2007) have all been reported to significantly benefit from the ingestion of energy drinks containing TA. These findings add to the growing prevalence of energy drinks being utilised in the sport and exercise science domain as a means of improving exercise performance. However, these data must be interpreted with extreme caution. Extrapolating findings from such studies in an attempt to examine the influence of TA on endurance performance is problematic due to the additive ingredients contained within such energy drinks. Furthermore, such studies have used ingestion periods of 0.5-1 hour (Alford et al., 2001; Forbes et al., 2007; Geiss et al., 1994) that would be too short to obtain peak TA plasma levels, assuming TA uptake is not augmented or diminished when consumed with the other ingredients within such drinks.

Physiological variables and RPE

The physiological and subjective measures performed within the current study provide support for existing TA ingestion research. However, the current study investigated these variables in a simulated endurance performance assessment more similar to real-world competitive endurance events than previous TA ingestion studies. The finding from this study that HR was not significantly different between the TA and PL conditions

was consistent with previous research (Galloway et al., 2008; Rutherford et al., 2010; Zhang et al., 2004). However, TA has previously been shown to have positive clinical applications for cardiac tissue through the modulation of intracellular Ca^{2+} (Azuma et al., 1992; Satoh and Sperelakis, 1998). Acute TA ingestion has also been demonstrated to decrease the HR of endurance trained athletes after 45-minutes of submaximal intensity cycling compared to an equivalent drink without TA during exercise (Geiss *et al*., 1994). In this study TA was co-ingested with caffeine and glucuronolacton, as a result it cannot be concluded whether it was TA alone or a combination of the ingredients that decreased HR after 45-minutes of cycling (Geiss et al., 1994). Based on these findings the duration of the endurance assessment within the current study may have been too short to demonstrate TA's influence on HR or co-ingestion with caffeine and glucuronolacton may have been required to elicit such an effect (Geiss et al., 1994). Therefore, the ingestion of 1000 mg of TA alone does not appear to exert any effect on sympathetic nervous system function in trained endurance athletes during maximal intensity exercise as previously detailed during prolonged submaximal activities (Geiss et al., 1994).

The findings of the current study are congruent with previous research as TA ingestion was not shown to influence oxygen uptake (Rutherford et al., 2010; Galloway et al., 2008), blood lactate concentrations (Galloway et al., 2008; Lee et al., 2003) or RPE (Lee et al., 2003; Rutherford et al., 2010). In contrast maximal oxygen uptake has been demonstrated to improve following TA supplementation in sedentary individuals in an incremental cycle test to exhaustion (Zhang et al., 2004). However, the fact that oxygen uptake did not differ between conditions despite the 3KTT being completed in significantly less time in the TA condition could be interpreted as a favourable effect of TA ingestion on central factors or muscular coordination. Any potential effect of TA on exercise metabolism during simulated time-trial performance would appear to act through interaction with the muscle membrane (Rutherford et al., 2010). This particularly relevant given that acute TA ingestion only increases plasma content not muscle content (Galloway et al., 2008). Unless prior TA ingestion attenuates TA losses from the muscle during maximal endurance exercise. However, it cannot be confirmed at this time that a 1000mg dose of TA is sufficient to produce a concentration gradient to prevent such losses from the muscle, only that it represents a substantially larger content than reported maximal daily intakes in normal diets. Such studies are yet to be conducted, although it has been demonstrated that TA content in the muscle decreases following longer duration exercise in endurance athletes(Cuisinier et al., 2001, 2002; Graham et al., 1995; Ward et al., 1999), therefore further investigation seems warranted. The primary focus of the present study was to ascertain if TA ingestion benefited maximal endurance performance when not preceded by prolonged submaximal intensity activity. The role of TA in neuronal function has previously been identified, with TA exerting both inhibitory and excitatory influence within the brain (El Idrissi and Trenkner, 2004). In vivo animal model research also suggests the potential for extracellular TA to modulate calcium ion content, thereby stabilising membranes and the neurotransmitter generation (Richard et al. 1995). Although no research currently exists investigating the effect of TA ingestion on the human nervous system in terms of muscle coordination or force production TA ingestion has shown the potential to positively influence neural function in populations suffering dysfunction (Birdsall, 1998). Therefore the use of alternative measures such as electromyography with synchronised kinematic data to quantify knee joint angle and force production during running may be warranted to allow assessment on whether TA influences neuromuscular function or muscular force production in an *in vivo* human model as has previously been detailed in an *in vitro* animal model (Bakker and Berg, 2002). However if alterations in muscle force

production were responsible for improved performance with TA ingestion in the current study they may have been too minimal to be detected by measures such as HR or oxygen uptake. However, enhanced time trial performance due to improved muscular coordination may suggest enhanced running efficiency which would not have increased HR or oxygen uptake. Studies observing enhanced endurance capacity following TA supplementation have not demonstrated alterations in perceived exertion (Lee et al., 2003) or have not measured RPE values (Zhang et al., 2004). Therefore minimal research exists clarifying any potential effect TA may have on perceptions of effort.

Conclusion

In conclusion, this study provides novel data for the literature as it was demonstrated that the ingestion of a 1000 mg dose of TA 2-hours prior to a 3KTT significantly enhanced endurance running performance. Furthermore magnitude based-inference results denoting practical significance state that TA is likely to be beneficial and unlikely to be harmful to the 3KTT performance of trained middle distance runners. The performance improvement in the TA condition also exceeded the minimum worthwhile change in performance for both UK national and elite level performance in competitive 3km races. However, the current study indicates that the ingestion of 1000 mg of TA does not affect HR, oxygen uptake, blood lactate concentrations or RPE of trained endurance athletes during maximal middle distance running. Therefore the mechanism responsible for the beneficial effect of TA ingestion on 3KTT performance in the current study remains to be elucidated. Future research should employ more invasive procedures in an attempt to identify a potential mechanism.

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Figure captions

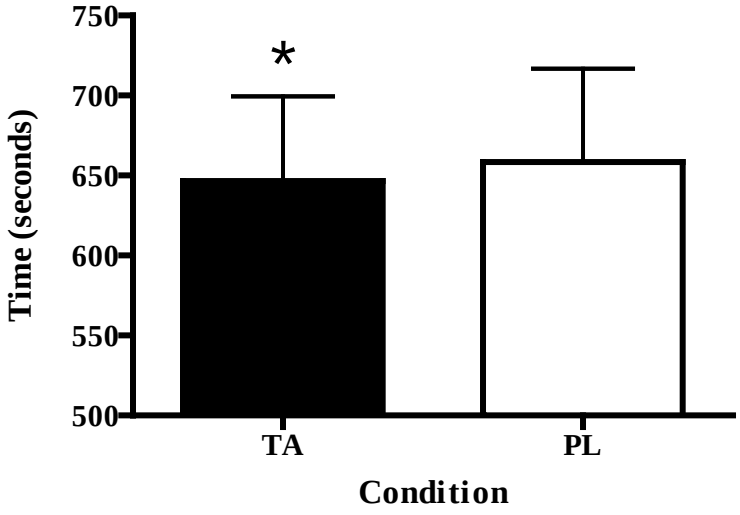
Fig. 1 Overall 3KTT performance time in the TA and PL conditions. * denotes significant difference between conditions

Fig. 2 Speed at 500m splits during the 3KTT in the TA and PL conditions.

Fig. 3 Relative oxygen uptake at 500m splits during the 3KTT in the TA and PL conditions.

Fig. 4 Heart rate at 500m splits during the 3KTT in the TA and PL conditions.

Fig. 5 RPE at 500m splits during the 3KTT in the TA and PL conditions.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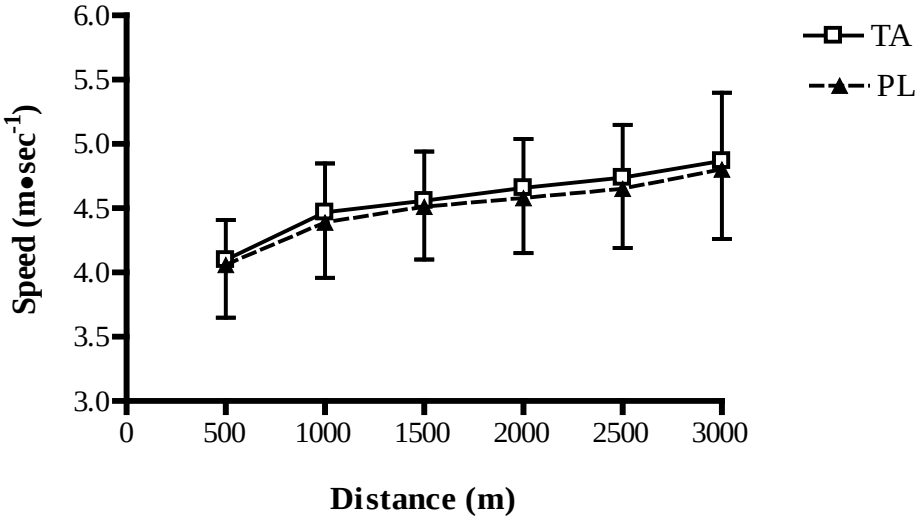
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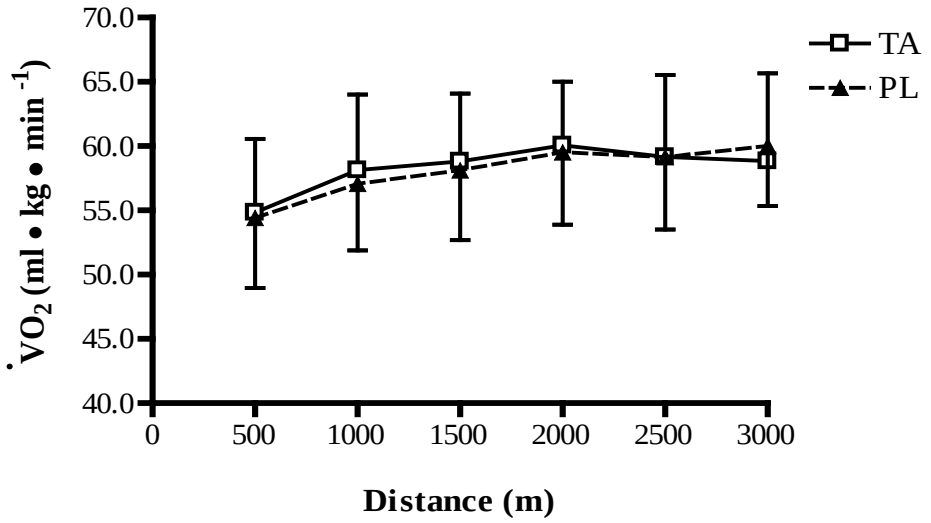
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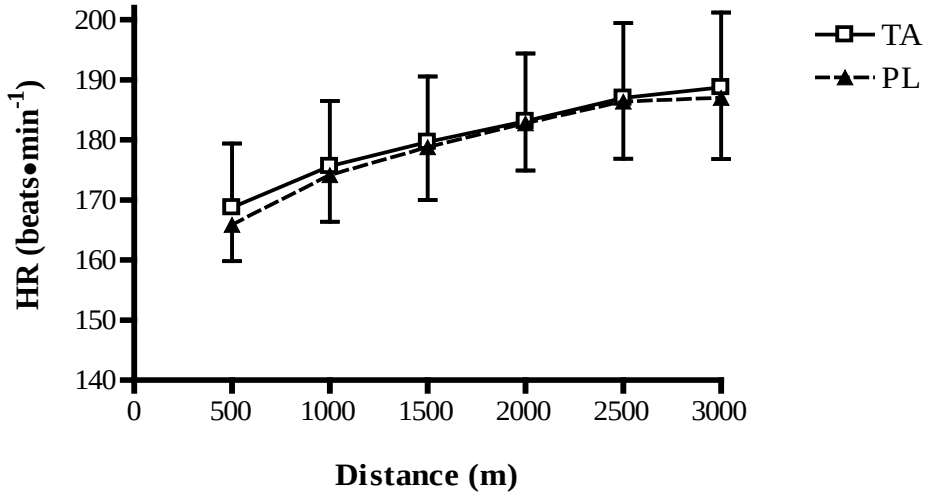
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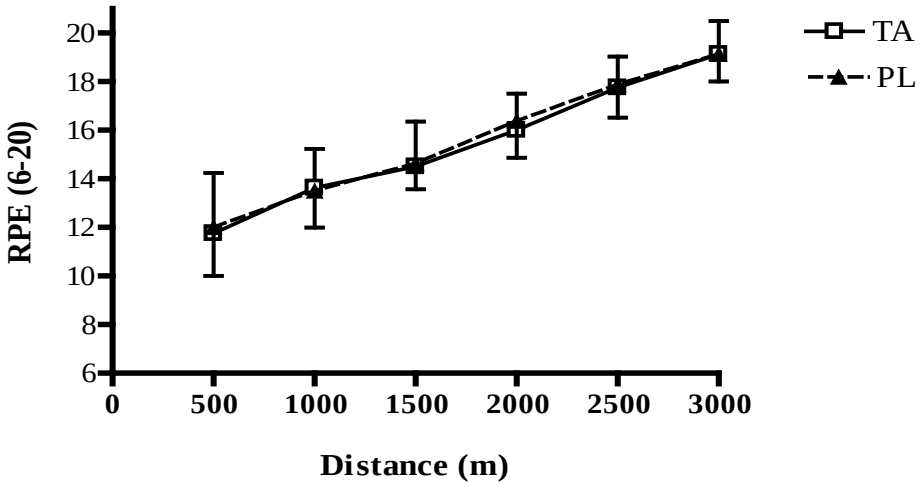
TA
PL


RPE (6-20)
20
18
16
14
12
10
8
6
0
500
1000
1500
2000
2500
3000
Distance (m)
TA
PL