Title: The effects of taurine on repeat sprint cycling after low or high cadence exhaustive exercise in females

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Abstract

This study investigated the effects of taurine on repeated sprint exercise, performed after fixed incremental ramp exercise to exhaustion at isokinetic high (90 r/min) or low (50 r/min) cadences. In a double-blind, repeated measures design, nine females completed an incremental ramp test to volitional exhaustion, followed by 2-min active recovery and 6 x 10-s sprints on a cycle ergometer, in one of four conditions: high cadence (90 r/min) + taurine (50 mg/kg body mass); high cadence + placebo (3 mg/kg body mass maltodextrin); low cadence (50 r/min) + taurine; low cadence + placebo. Heart rate (HR) and blood lactate concentration B[La] were measured before and after the ramp test and after the sprints. Taurine lowered HR *vs.* placebo prior to the ramp test ($P = 0.004$; $d = 2.1$). There was an effect of condition on ramp performance ($P < 0.001$), with higher end-test power ($d = 3.7$) in taurine conditions. During repeated sprints, there was a condition × time interaction ($P = 0.002$), with higher peak sprint power in the placebo conditions compared to taurine (sprint 2-6; $P < 0.05$). B[La] was higher in taurine compared to placebo post-ramp ($P = 0.004$; $d = 4.7$). Taurine lowered pre-exercise HR and improved incremental end-test power output, with subsequent detrimental effects on sprint performance, independent of cadence. Short endurance performance can be acutely enhanced after taurine ingestion but this effect might not be maintained across longer periods of exercise or induce the need for longer recovery periods.

Key words: ergogenic aids; supplementation; cycling; amino acids.

Introduction

Oral taurine supplementation can enhance exercise performance (Balshaw et al. 2013; Milioni et al. 2016; Warnock et al. 2017; Zhang et al. 2004). However, the understanding of taurine's ergogenic potential has been clouded by its frequent co-ingestion with caffeine in research (Forbes et al. 2007; Gwacham and Wagner 2012). This is problematic because isolated caffeine has ergogenic effects (Burke 2008). There are few studies that have investigated isolated taurine ingestion or administered it in doses that appear necessary to affect performance. For example, Warnock et al. (2017) reported improvements in repeat sprint ability following oral consumption of taurine at a dose of 50 mg/kg body mass (3.5-4.5 g). Similar doses have been shown to reduce muscle damage and oxidative stress after eccentric exercise (Da Silva et al. 2013) and possibly improve anaerobic capacity (Milioni et al. 2016). However, studies using smaller doses of taurine have been less consistent, with reported improvements in 3 km running performance after 1 g of taurine (Balshaw et al. 2013) but similar doses (1-1.6 g) of taurine having no effect on prolonged endurance (Rutherford et al. 2010) or 4 km time trial performance (Ward et al. 2016). Chronic supplementation with taurine over longer periods (6 g/day for seven days) has also been reported to increase $\dot{V}O_{2max}$ (Zhang et al. 2004).

Taurine is the most abundant free amino acid in mammalian tissue, accounting for 50-60 % of the free amino acid pool (Huxtable 1992), and is available to facilitate a variety of biological processes that can support exercise performance. Plasma taurine levels range from 29 to 49 μM, which are increased (10 to 60 mM) in skeletal muscle (Ishikura et al. 2013). Taurine is transported into skeletal and cardiac muscle through a taurine transporter (TauT) and assists with sarcoplasmic reticulum Ca^{2+} handling, particularly in type II muscle fibres (Hamilton et al. 2006). Improvements in muscle performance have been attributed to taurine-facilitated Ca^{2+} handling of both cardiac and skeletal myocytes (Huxtable 1992). Furthermore, prevention of taurine uptake in TauT knockout mice significantly reduced time to exhaustion (Ito et al. 2014). These observations are consistent with the reports of reduced skeletal muscle function following *in vitro* taurine depletion (Hamilton et al. 2006). Other suggested physiological roles of taurine include enhanced mitochondrial buffering (Hansen et al. 2006) or activation of extra-synaptic gammaaminobutyric acid (GABA) receptor isoforms in the thalamus (Jia et al., 2008), both of which could affect exercise performance.

There is a ~ 25% depletion in type II, yet not type I, fibre muscle taurine concentration after exhaustive exercise in rats (Matsuzaki et al. 2002) and ~ 85% increases in taurine urinary excretion have been observed after marathon running in humans, indicating release of taurine from the exercising muscle (Cuisinier et al. 2001). However, taurine supplementation (5 g/day for 1 week) does not alter its content in the resting skeletal muscle among healthy individuals, whose taurine muscle content is presumably maximised (Henriksson 1991). This is despite causing a 13-fold increase (60 μM to 750–1,000 μM) in plasma concentration 90 min after consumption (Galloway et al. 2008). Increases in plasma taurine levels have been suggested to supress muscle taurine release during periods of intracellular hypo-osmolality (Ishikura et al. 2013), thereby maintaining intramuscular taurine content and availability for other biological processes.

The evidence suggests that when plasma taurine concentration is sufficiently increased, then both physiological and performance benefits can be conferred. It is also clear that depletion of taurine is biased toward type II fibres. One method of targeting type II muscle fibres has been to perform cycling exercise at fixed power outputs, while restricting cadence, inducing low velocity-high force muscle contraction (Lepers et al. 2001). Low cadence cycling at a fixed power output should preferentially recruit higher threshold motor units compared to high cadence conditions (Ahlquist et al. 1992). Based on the above collective reasoning, low cadence exercise that is assumed to

preferentially recruit type II fibres, should lead to a higher depletion of muscle taurine, creating greater dependency on available plasma taurine to facilitate ongoing muscle contraction.

The aims of this study were to investigate the effects of ergogenic doses of taurine (50 mg/kg) *vs.* placebo on repeated sprint exercise, performed after fixed incremental ramp exercise to exhaustion at controlled high (90 r/min) or low (50 r/min) cadences in females. It was hypothesized that i) the high cadence + taurine condition would produce the greatest overall performance in both the ramp exercise and subsequent repeated sprint protocol by providing high taurine availability, without the assumed heavy depletion during prior ramp exercise and ii) both taurine conditions would enhance overall performance on the ramp and repeated sprint protocol compared to any placebo conditions by offsetting the potential for taurine depletion during prior exercise.

Methods

Design

The participants reported to the laboratory on five occasions at the same time of day (1000 hours). On visit 1, a familiarisation session was provided. On visits 2-5, a randomised, double-blind, counterbalanced, cross-over design was followed, whereby the participants completed an incremental ramp test to volitional exhaustion, followed by 2-min active recovery and 6 x 10-s sprints on a cycle ergometer in isokinetic mode in one of four conditions: high cadence (90 r/min) + taurine (50 mg/kg body mass); high cadence + placebo (3 mg/kg body mass maltodextrin); low cadence (50 r/min) + taurine (50 mg/kg body mass); low cadence + placebo (3 mg/kg body mass maltodextrin). The testing was conducted over a two week period, with each participant visiting the laboratory twice per week, separated by 48-h.

Participants

Nine female university lacrosse players (age 22 ± 5 years, stature 1.63 ± 0.4 m, body mass 65.0 ± 9.2 kg) took part in this study. Informed consent was obtained from all individual participants included in the study. *A-priori* sample sizes were calculated using G*Power (Version 3.0.10). Given the typical effect sizes (Cohen's d = 0.5-1.0; Warnock et al. 2017) reported using repeat-sprint protocols with taurine, a sample size of seven was deemed sufficient to identify differences between groups with a statistical power of 0.80. We recruited nine participants to account for experimental mortality, raising the statistical power to 0.90. The participants were asked to arrive at the laboratory 7-10 days after the start of the menstrual cycle, having not completed any exercise in the 48-h before testing, and having abstained from alcoholic and caffeine consumption in the 24-h prior. The participants were instructed to stay hydrated and consume a well-balanced meal no less than 2-h before testing, which was recorded and replicated across each day. The participants consumed an additional 200 ml of fluid 1-h prior to exercise during each visit. Institutional ethical approval was granted for this study, which was conducted in accordance with the 1964 Helsinki declaration.

Supplementation

All of the supplements were prepared in a powder form and measured using an analytical balance (Precisa 125A, Precisa Gravimetrics AG, Zurich, Switzerland) for subsequent ingestion in gelatine capsules. The capsules contained one of the following: taurine (50 mg /kg body mass) + maltodextrin (3 g/kg body mass) or placebo (3 mg/kg body mass maltodextrin). Participants' body mass was taken prior to each trial to measure the correct dose and the supplements were balanced such that an equal number of capsules were ingested between conditions. The dosages of taurine followed the recommendations of recent studies (Da Silva et al. 2013; Warnock et al. 2017) and were all sourced from the same company (My Protein, Manchester, UK). After ingestion, the participants rested in a seated position for 1.5-h in a quiet room and were observed by the investigators. The 1.5-h timing was chosen as this accounted for the peak plasma availability of taurine after oral administration (Ghandforoush-Sattari et al. 2010).

Familiarisation
The participants were fitted to an electronically-braked cycle ergometer (Lode Excalibur Sport, Lode B.V. Medical Technology, Groningen, The Netherlands), where saddle and handlebar position was recorded for all subsequent trials. The test protocol was explained to the participants, in detail, and was demonstrated by one of the research team. The participants were also shown the rating of perceived exertion (RPE) scale (Borg 6-20) and provided with instructions of how to interpret their score. The participants then completed the early stages of the incremental test and three maximal sprints on the bike. The typical error (TE) of the repeated sprints in our laboratory is 20 W.

Experimental Protocol
Resting heart rate (HR) (Polar FT1, Polar Electro Oy, Kempele, Finland) and blood lactate concentration B[La] were measured 5-min prior to the beginning of each test (1.5 h after taking the supplements). Heart rate was measured continuously for 5 min, with the final 1-min reported. A lancet was used to extract a capillary blood sample from the index finger to measure B[La], which was measured using a calibrated analyser (Biosen C Line, EKF diagnostic GmbH, Barleben, Germany). For all B[La] measurements, two samples were taken and the mean was calculated. A 5-min steady state warm up of 100 W was performed, after which another 3-min was provided to prepare for the test protocol. The participants then completed a ramp test, which started at 50 W and increased by 35 W/min on the ergometer. The tests were conducted on an ergometer fixed in the isokinetic mode, such that cadence was controlled to either 50 or 90 r/min (low or high cadence, respectively). The ramp test was continued to volitional fatigue and terminated when the increasing power output could not be sustained. In the last 10-s of the test, RPE and HR were recorded and a second B[La] was taken 1-min after completion. The participants continued to cycle at the same cadence for a period of 3-min after the test but the power output was reduced to 50 W. This was a period of active recovery that transitioned into the repeated sprint protocol, which comprised a series of six 10-s sprints at a resistance of 0.075 × body mass, with each sprint interspersed by 10-s recovery. A 5-min recovery period was then completed at 50 W. A final measurement of HR and RPE was recorded in the 10-s after the sprints and B[La] was measured 1-min after. Inter-sprint fatigue index (percentage change in mean power output between the six sprints) was calculated based on the equation of Fitzsimons et al. 1993:

$$\text{Inter-sprint fatigue \%} = 100 - [(\text{Total power output} / \text{Ideal power output}) \times 100] \quad \text{[Eq. 1]}$$

Where:
Total power output = sum of peak power values from all sprints
Ideal power output = the total number of sprints × highest peak power.

Statistical Analysis
A three-way repeated measures analysis of variance (RM-ANOVA) was conducted, with cadence (low or high), condition (taurine or placebo) and time as the independent variables. Time effects were considered at three levels (resting, post-ramp and post-sprints) for measurements of HR, RPE and B[La] and six levels (sprints 1-6) for: peak power and mean power. A two-way RM-ANOVA was conducted (cadence × condition) for ramp test end power and inter-sprint fatigue index. Greenhouse-Geisser corrections were used when the assumption of sphericity was violated. Significant interactions between the independent variables were followed-up using Bonferroni tests to identify pairwise differences. Statistical significance was accepted at $P < 0.05$ and all analyses were performed on IBM SPSS Statistics (Version 21, IBM Corp., Armonk, NY, USA). Effect sizes (Cohen's *d*) were also calculated for all pairwise differences. Effect sizes were defined as: trivial = 0.2; small = 0.21–0.6; moderate = 0.61–1.2; large = 1.21–1.99; very large > 2.0.

Results

There were no trial order effects found for end-power on the ramp test (P = 0.799) or for sprint performance (P = 0.573).

There was no condition × cadence interaction effect for ramp test end power ($F_{(1,8)}$ = 0.001, P = 0.972); however, there was an effect of condition ($F_{(1,8)}$ = 30.238, P < 0.001), with higher end-test power (d = 3.7) in the taurine (197 ± 11 W or 4.0-min) conditions compared to placebo (185 ± 10 W or 3.5-min). In the subsequent repeated sprint tests, there was no condition × cadence × time interaction ($F_{(5,40)}$ = 1.264, P = 0.298), nor was there an interaction between condition and cadence ($F_{(1,8)}$ = 0.571, P = 0.472) but there was a condition × time interaction ($F_{(5,40)}$ = 4.742, P = 0.002). *Post-hoc* tests revealed higher peak sprint power in the placebo conditions compared to the taurine condition during sprint 2 (P = 0.049; d = 2.5), sprint 3 (P = 0.008; d = 4.5), sprint 4 (P = 0.06; d = 3.9), sprint 5 (P = 0.03; d = 5.1), and sprint 6 (P = 0.008; d = 4.2) (Figure 1). Consistent with these results, there was no interaction between condition and cadence for inter-sprint fatigue index ($F_{(1,8)}$ = 0.589, P = 0.465) but there was a main effect for condition ($F_{(5,40)}$ = 21.037, P = 0.002). *Post-hoc* tests revealed greater (P < 0.001; d = 1.41) fatigue index in the taurine (28.2 ± 6.4 %) compared to placebo conditions (19.1 ± 6.9 %).

*********INSERT FIGURE 1 HERE*********

There was no interaction between condition, cadence and time for B[La] ($F_{(2,16)}$ = 0.009, P = 0.991); however, there was a two-way interaction between condition and time ($F_{(2,16)}$ = 4.110, P = 0.036). *Post-hoc* tests revealed higher B[La] in the taurine condition compared to placebo post-ramp test (P = 0.004; d = 4.7) (Figure 2). There was no interaction between condition, cadence and time for HR ($F_{(2,16)}$ = 1.578, P = 0.237) but there was a two-way interaction between condition and time ($F_{(2,16)}$ = 11.327, P = 0.005). *Post-hoc* tests revealed lower HR in the taurine condition *vs.* placebo prior to the ramp test (P = 0.004; d = 2.1) (Figure 2). There were only main effects of time on RPE ($F_{(1,8)}$ = 164.7, P < 0.001) and no interactions between condition or cadences (Figure 2).

*********INSERT FIGURE 2 HERE*********

Discussion

In partial agreement with our first hypothesis, the main findings of this study were that oral ingestion of taurine (50 mg/kg body mass) acutely ameliorated incremental ramp exercise performance. There was no difference in ramp exercise or sprint performance between the two cadence conditions and no interaction with taurine supplementation. In other words, only taurine ingestion was responsible for an increase in ramp exercise. However, the effects of taurine supplementation on initial ramp exercise were such that subsequent repeated sprint performance was impaired. The extended work achieved in both of the taurine conditions (i.e. low or high cadence) induced a higher glycolytic response, as indicated by the increase in end-ramp exercise B[La] (Figure 2). Therefore, it is likely that the participants experienced a greater degree of peripheral fatigue, leading to poorer sprint performance. It is unlikely that the blood plasma was devoid of taurine, owing to the brevity of the ramp test; however, the 3-min period of active recovery between the two exercise bouts, which was intentionally insufficient to permit full recovery among all groups, was most detrimental to both taurine conditions. Therefore, acute taurine supplementation did not provide a sustained ergogenic effect, despite facilitating initial time to fatigue on the ramp.

Based on the findings of others (Ahlquist et al. 1992), we fixed cadence at 50 r/min in an attempt to control muscle fibre recruitment patterns, such that higher threshold motor units would be preferentially selected; at least in earlier exercise stages. Similar to the suggestions of others (Sidossis et al. 1992), there was no difference in power output on the ramp test between the low or high cadence conditions, reflected by the similarity in end test power (Figure 1). Likewise, the effects of taurine supplementation did not depend on the cadence condition (i.e. no interaction). As such, controlling cadence in the ramp exercise had no independent effect during this study. Given that we did not measure muscle fibre recruitment patterns, it is not possible to directly comment on the interaction between taurine and muscle fibre types; however, these findings reject the hypothesis that the effects of taurine ingestion on repeated sprint performance would be less effective after performing low cadence (high force) pre-fatiguing exercise. These findings could be related to the possible incorrect assumption that the slower muscle shortening velocities and greater muscle tension induced by low cadence conditions leads to greater recruitment of fast twitch fibres (Lepers et al. 2001). Alternatively, it is feasible that type II fibres were recruited equally between conditions as fatigue ensued. This would be consistent with the known progressive increase in EMG activity levels during repeated muscle contractions to fatigue (Pincivero et al. 2006). If this was the case, then the muscle fibres used would include a mixture of type I, IIa and IIx fibres, permitting an equal effect of taurine across conditions.

That taurine supplementation improved ramp exercise performance is consistent with the reported improvements in 3 km time-trial performance (Balshaw et al. 2013), increased power output during repeated Wingate sprints (Warnock et al. 2017) and improvements in $\dot{V}O_{2max}$ (Zhang et al. 2004). Collectively, this evidence indicates an ergogenic effect of taurine on activities with a predominant oxidative metabolic requirement. However, Rutherford et al. (2010) provided 1.6 g of oral taurine and found no differences in cycling time to exhaustion at 5 kJ/kg of external work, performed immediately after 90 min at 66.5% $\dot{V}O_{2max}$. The reasons for the discrepancies between studies could be related to the selected doses. For example, Zhang et al. (2004) provided 6 g/day of taurine, while Warnock et al. (2017) provided 50 mg/kg (~ 3.5-4.5 g), which is higher than the 1.6 g provided by Rutherford et al. (2010). However, Balshaw et al. (2013) provided only 1 g of taurine. Therefore, a more plausible explanation could be the choice of exercise performance. The prolonged exercise period (90 min) used by Rutherford et al. (2010), which commenced 60 min after taurine ingestion, is longer than the 1-h half-life of plasma taurine (Ghandforoush-Sattari et al. 2010). This means that plasma taurine is likely to have significantly reduced by the start of the time to exhaustion conducted by Rutherford and colleagues. Based on the apparent inability of taurine supplementation to change muscle taurine content after 7 days (Galloway et al. 2008), its ergogenic effects most likely depend upon high plasma taurine levels, which would have been less available in their study. Whilst we did not measure plasma taurine concentration in the current study, which was a limitation, the period of time between ingestion and performance is more likely to have permitted an ergogenic effect.

Heart rate was reduced in the taurine group at the start of the test (Figure 2). This has been reported elsewhere after single doses (Warnock et al. 2017) and 4 weeks of taurine supplementation (Ahmadian et al. 2017). Indeed, these studies have also reported reductions in blood pressure and rate pressure product, indicating the potential cardio-protective effects of taurine at higher doses. To the best of our knowledge, there has been no dose-response study of taurine on cardiovascular function but this would be necessary to verify this effect and elucidate the associated mechanisms. Depending on the consistency of these findings, it is possible that that confers a health benefit, as well as an ergogenic one.

The mechanisms that explain the role of taurine are numerous but often relate to its effect on Ca^{2+} storage in the sarcoplasmic reticulum and intracellular handling, via increased Ca^{2+}-activated ATPase

pump activity in both skeletal and cardiac myocytes (Huxtable 1992). These changes permit increases in both skeletal and cardiac contractility, Indeed, deficiency reduces left ventricular contractility in feline hearts (Novotny et al. 1991) and taurine-containing energy drinks increase stroke volume in humans (Baum and Weiss, 2001). In addition, an anti-oxidative role has been ascribed to taurine based on *in vitro* investigations, which demonstrate taurine's pH buffering capacity in the mitochondrial matrix (Hansen et al. 2006). In this instance, taurine facilitates the function of rate-limiting oxidative enzymes (i.e. isocitrate dehydrogenase) and reduces the production of reactive oxygen species, thus stabilising the mitochondrial matrix and improving the efficiency of ATP turnover for energy-demanding processes in the cell (Hansen et al. 2006). The combination of these mechanisms would partly explain the effects of taurine on endurance activities, as well as short-term force production.

The decrement in repeated sprint performance observed after the exhaustive bout of exercise in the taurine condition is more difficult to explain. There are two possible explanations. Firstly, participants that were supplemented with taurine exercised for longer and, therefore, may have experienced a greater degree of peripheral fatigue, thus reducing subsequent sprint performance. Secondly, taurine release by contracting muscles into the plasma, via an osmoregulatory process, may have facilitated a subsequent decline in performance. Plasma taurine concentration ranges from 29 to 49 uM in humans (Cuisinier et al. 2001) and is ~ 500-fold greater in skeletal muscle (Galloway et al. 2008), thus creating an osmotic gradient. Supplementation with 1.66 g of taurine induces a 13-fold increase in plasma taurine concentration 2-h post-ingestion (Galloway et al. 2008). During and immediately after exhaustive exercise, muscle osmolality would increase as a result of increased intracellular lactate production (Lang et al. 1998) and phosphocreatine breakdown (Sejersted et al. 2000), leading to cell swelling. In order to sustain osmolality, the muscle actively expels inorganic ions or organic molecules, including taurine. We postulate that supplementation with taurine would decrease this osmotic gradient, thereby reducing transport of solutes out of the muscle cell. Indeed, supplementation with taurine is known to suppress taurine release from the skeletal muscle (Ishikura et al. 2013). The consequence would be increased osmotic stress and potential cellular damage, which could explain the diminished performance in the repeated sprint test. At this stage, this reasoning is speculative and remains to be explored *in vivo*.

Oral ingestion of taurine 1.5-h before exercise improved exhaustive incremental end-test power output, independent of a low (50 r/min) or high (90 r/min) cadence condition. It is possible that these effects are peripheral and relate to role of taurine on skeletal muscle contractile function. Subsequent sprint performance was impaired in the taurine group. The reasons for this are unclear; however, it would appear that taurine ingestion facilitates initial performance, leading to greater fatigue or osmotic imbalance, thus extending the required recovery period or increasing intracellular damage. In combination with the results of others, athletes competing in short, yet aerobically-biased cycling or running events could benefit from acute taurine ingestion. However, because of the decrements in subsequent sprint performance, the recovery time required between exercise bouts and cellular responses must be investigated further.

Compliance with Ethical Standards

Conflict of Interest: The authors declare that they have no conflict of interest.

Ethical approval: All procedures were performed in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from all individual participants included in the study

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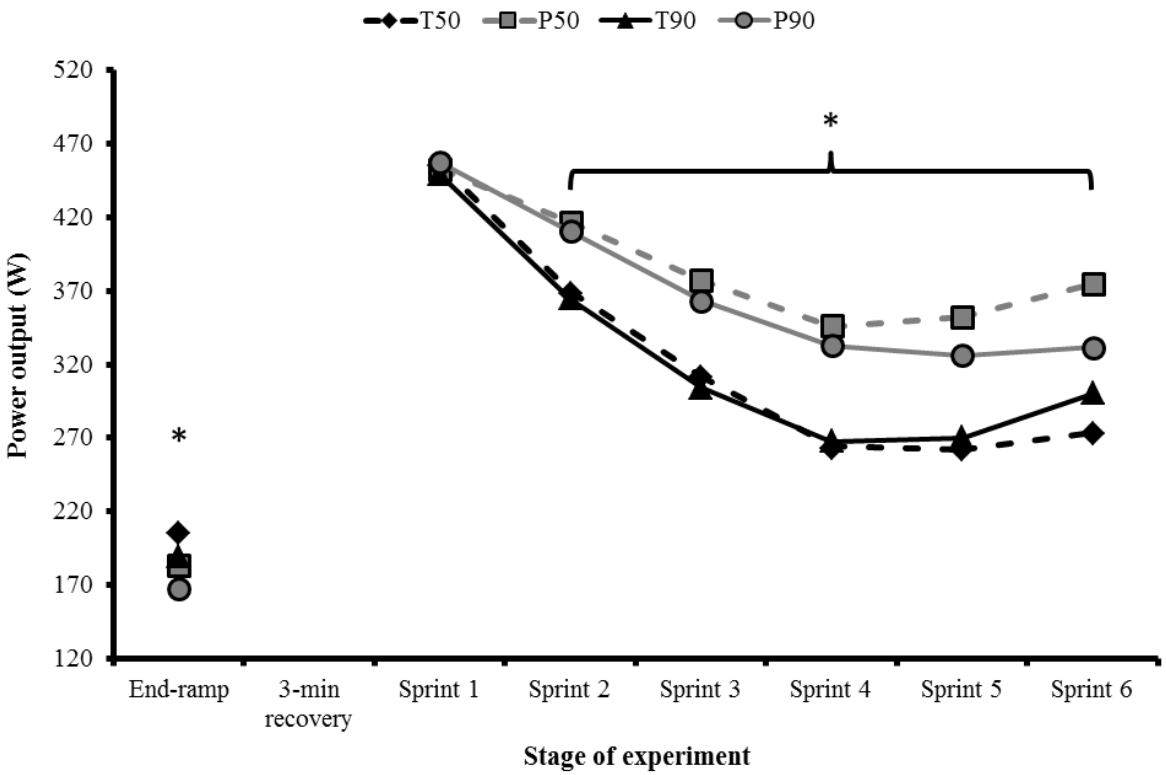

Figure 1. The effects of high (90) or low (50) cadence and taurine (T) or placebo (P) on peak power output at the end of incremental ramp exercise and during 6 repeated 6 sprints (n = 9). * = sig. difference between taurine *vs.* placebo groups after the ramp test and during sprints 2-6).

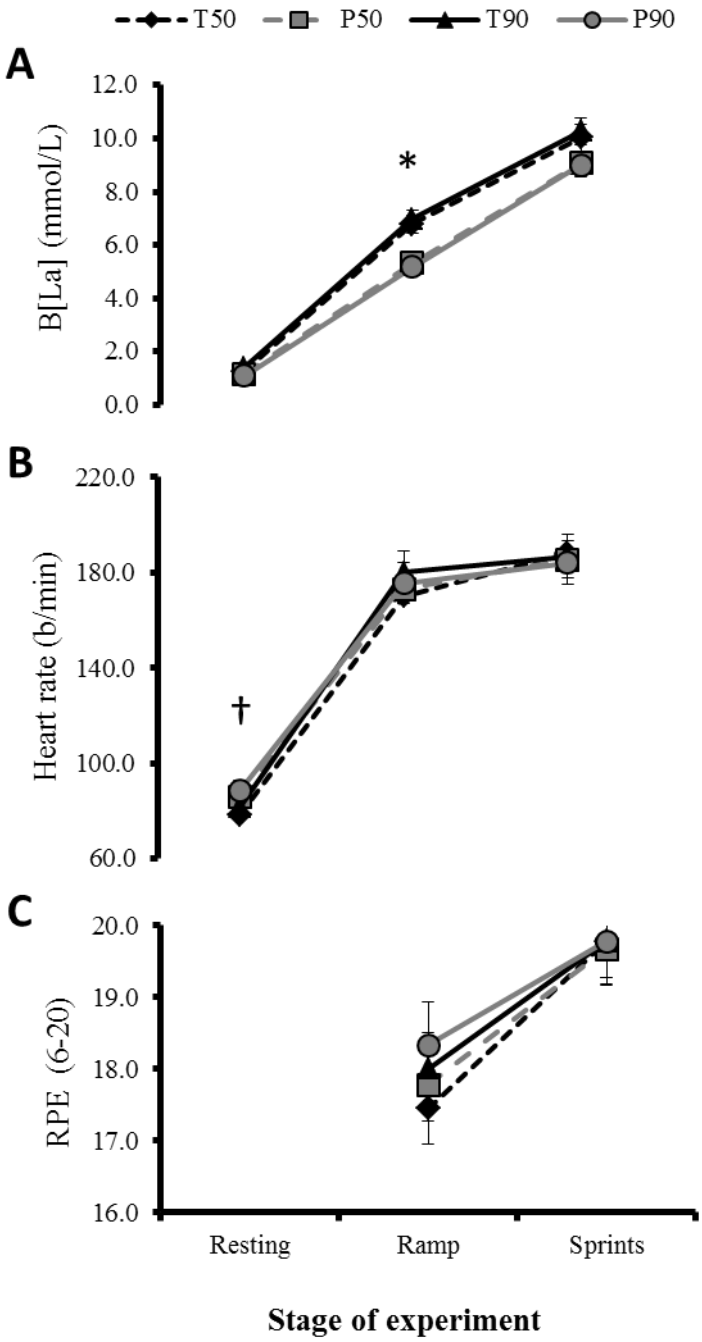

Figure 2. The effects of high (90) or low (50) cadence and taurine (T) or placebo (P) on blood lactate concentration (B[La]), heart rate (HR) and rating of perceived exertion (RPE) during rest, incremental ramp exercise and repeated sprints (n = 9). * = sig. higher taurine *vs.* placebo B[La] after ramp test. † = sig. higher placebo *vs.* placebo HR before ramp test.