Amino Acids (2018) 50:189–198
https://doi.org/10.1007/s00726-017-2505-3

ORIGINAL ARTICLE

Taurine supplementation can increase lipolysis and affect the contribution of energy systems during front crawl maximal effort

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Received: 7 February 2017 / Accepted: 13 October 2017 / Published online: 29 October 2017

Abstract Taurine can affect the energy system metabolism, specifically the lipid metabolism, since an increase in lipid oxidation may promote carbohydrate savings. We hypothesized that taurine supplementation associated with high-intensity exercise could increase levels of lipolysis, benefiting swimmer performance. Nine male competitive swimmers performed two 400-m front crawl maximal efforts with a 1-week washout, and the athletes received 6 g of taurine (TAU) or placebo (PLA) supplementation 120 min before performing the effort. Oxygen consumption and the contribution of the energy systems were analyzed post effort using a Quark CPET gas analyzer. Blood samples were collected before, and 5 min post the effort for taurine and glycerol analysis. Immediately before and 3, 5, and 7 min post the effort, blood samples from the earlobe were collected to determine lactate levels. An increase of 159% was observed in taurine plasma levels 120 min post ingestion. Glycerol levels were higher in both groups post effort; however, the TAU condition promoted an 8% higher increase than the PLA. No changes were observed in swimmer performance or lactate levels; however, the percentage change in lactate levels ($\Delta[La^-]$) was different (TAU: 9.36 ± 2.78 mmol L^{-1}; PLA: 11.52 ± 2.19 mmol L^{-1}, $p = 0.04$). Acute taurine supplementation 120 min before performing a maximal effort did not improve swimmer performance; however, it increased glycerol plasma levels and reduced both the $\Delta[La^-]$ and lactic anaerobic system contribution.

Keywords Taurine · Swim · Lipolysis · Performance

Introduction

To maximize athletes' performance, researchers are seeking nutrients that could enhance physical training (Jeukendrup et al. 2005). Taurine is a free nitrogen compound used as a

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nutritional supplement due to its capacity to regulate calcium ion transport into the sarcoplasmic reticulum (Schaffer et al. 2010). In addition, this ergogenic resource is also able to increase calcium levels, promote the greater power of muscle contraction (Bakker and Berg 2002), and regulate insulin sensibility (Carneiro et al. 2009). Considering this hormone plays an important role for athletes due to the regulation of the carbohydrate metabolism, taurine supplementation could increase glucose availability and energy production, which in turn would stimulate glycogen resynthesis and protein synthesis, benefiting performance.

Zhang et al. (2004) evaluated the intake of taurine (6 g) during seven days in cyclists and observed an increase in the maximum rate of oxygen consumption (VO_2max) and time to exhaustion. Balshaw et al. (2013) analyzed the effect of acute taurine intake (1 g) 2 h before a 3-km running test and verified an enhancement in performance of 1.7%, which reinforces the use of taurine as an ergogenic aid. Furthermore, other studies reported beneficial effects of taurine on lipid metabolism (Murakami 2015; Tsuboyama-Kasaoka et al. 2006), as it may stimulate the gene expression related to lipid metabolism, mitochondrial biogenesis, and respiratory function (Tsuboyama-Kasaoka et al. 2006).

According to Rutherford et al. (2010), acute taurine supplementation before training in cyclists resulted in a higher body fat oxidation, which suggests that this ergogenic aid can improve energy substrates post exercise. The aforementioned taurine actions can affect performance and metabolic system contribution, since an increase in lipid oxidation can result in carbohydrate savings, minimizing the utilization of glycogen stores and favoring post-training recovery.

Moreover, availability of the substrate can influence physical performance by increasing lipolysis during exercise and reducing muscle glycogen utilization, thereby promoting an immediate mechanism to save glycogen storage (Carter et al. 2001). Therefore, the lipolysis index determined by the quantification of glycerol blood levels is an important method used to evaluate lipid oxidation in athletes during exercise.

The specific demand determination of each sport is crucial for developing optimized training models, nutritional strategies, and ergogenic aids to maximize athletic performance (Artioli et al. 2012). Several studies have analyzed the fast component of excess post-exercise oxygen consumption (EPOC) to determine the alactic anaerobic metabolism, and the delta peak plasma lactate multiplied by 3 and by the athletes' body mass to determine the lactic anaerobic metabolism (LAM) (Artioli et al. 2012; Zagatto et al. 2011; Tomlin and Wenger 2001).

Considering the previously described actions of taurine and knowing its peak blood concentration can be reached 120 min after consumption (Ghandforoush-Sattari et al. 2010), we verified the effects of acute taurine supplementation 120 min before performing a 400-m front crawl maximal effort on performance, lipid metabolism, and energy system contribution. Thus, we hypothesized that taurine supplementation associated with high-intensity exercise could promote increased levels of lipolysis and benefit the physical performance of swimmers.

Methods

Subjects

Ten male competitive swimmers with a minimum of 3 years' experience at regional or national competition level volunteered to participate in the study; however, one athlete was withdrawn before completion of the study as he did not perform all the measurements. The analysis was based on the remaining nine swimmers (age 19.78 ± 3.38 years; body mass 76.29 ± 8.62 kg; height 180.11 ± 8.23 cm; VO_2peak 60.42 ± 7.70 mL kg^{-1} min^{-1}). The present study was approved by the Human Subject Committee of the School of Physical Education and Sport of Ribeirão Preto–University of São Paulo, approval number CAAE 34881914.5.0000 and conducted in accordance with the Declaration of Helsinki. All participants and/or their parents were informed about the experimental procedures and risks, and both provided written informed consent authorizing the athletes' participation in the study. Participants were only recruited if they were not currently taking chronic or daily doses of nutritional supplements or anti-inflammatory medication and were instructed to avoid drinking caffeine, alcohol, and energy drinks for at least 12 h before each measurement.

The sample size was calculated based on the results of Brisola et al. (2015), using the data of anaerobic contribution (MAOD) in the exercise between placebo and supplementation (Sodium bicarbonate) groups. Analysis with G*Power software version 3.0.10 (Heinrich-Heine University, Düsseldorf, Germany) gave the study sample size required for the analysis as 6 individuals, to provide a statistical power of 80% with an alpha of 0.05.

Study design

A double-blind, crossover with 1-week washout study design was conducted. The participants performed two 400-m front crawl maximal efforts with an interval of 1 week between efforts, and at each time point, the athletes received taurine (TAU) or placebo (PLA) supplementation 120 min before the swimming effort. The TAU supplementation consisted of 6 g of taurine obtained from Ajinomoto CO (taurine, 99% pure, Ajinomoto CO., INC., Limeira, SP, Brazil), and starch was used as the placebo. All capsules were produced by the University Pharmacy

of the University Hospital (HCFMRP USP). Before the trials, a warm-up was performed consisting of approximately 1000 m front crawl stroke of low to moderate intensity, determined subjectively by the swimmers. Performance (time and velocity) was analyzed by the motion-analysis software Kinovea™ (version 0.8.15, available for download at http://www.kinovea.org). The tests were performed in a 25-m swimming pool with a water temperature of 25 ± 1 °C. Before the effort, the baseline oxygen consumption at rest was collected and, immediately after the effort, oxygen consumption was collected for 5 min. The participants were requested to avoid taurine food sources such as fish, seafood, and energy drinks in the 24 h preceding the study protocol.

Dietary intake

Athletes were instructed to complete a three-day food record to verify their energy intake. Diet Win professional 2012® software was used to examine kcal and macronutrients. The data were analyzed considering the dietary reference intakes (DRIs) for healthy adults (IOM 2005). The energy requirement (ER) was calculated using the DRI formula, considering a high physical activity coefficient (IOM 2005).

Data collection

During the trials, expired gases were collected breath-by-breath immediately after each 400 m effort and analyzed by a Quark CPET gas analyzer (Cosmed®, Rome, Italy). The gas analyzers were calibrated immediately before and verified after each test using a gravimetrically certified determined gas mixture, while the ventilometer was calibrated pre-exercise and verified post-exercise using a 3 L syringe in accordance with the manufacturer's instructions. Following removal of outliers to exclude discrepant breaths, breath-by-breath data were interpolated to give 1 s values (OriginPro 8.0, OriginLab Corporation, Microcal, Massachusetts, USA) to enhance the underlying response characteristics (Zagatto et al. 2011). Blood samples were collected from an antecubital vein using vacutainer tubes (Vacutainer Becton–Dickinson Company, Plymouth, UK) 120 min after the supplementation (before the effort) and 5 min after the effort, to analyze taurine and glycerol from the plasma. Immediately before the maximal effort of 400 m, blood samples from the earlobe were collected to determine lactate concentration at rest ($[La^-]_{rest}$). In addition, blood samples from the earlobe were collected to determine peak blood lactate concentration ($[La^-]_{peak}$) at three, five, and seven minutes after the end of the effort. The $[La^-]$ was assessed using a blood lactate analyzer YSI-2300 (Yellow Springs Instruments®, Ohio, USA).

Taurine assay

Plasma taurine was quantified by high-performance liquid chromatography (Shimadzu, model LC 10AD) through the method of Deyl et al. (1986). Taurine 99% (Sigma-Aldrich, St. Louis, MO, USA) was used as a standard.

Markers of lipolysis

The quantification of plasma glycerol was performed through the enzymatic method (glycerol phosphate oxygenase) using the Glycerol Assay Kit sigma® in a mass spectrometer.

Oxygen consumption analysis

After the 400 m effort, the subjects were instructed to breathe immediately into a face mask (Hans Rudolph, Kansas City, MO, USA) connected to a breath-by-breath gas analyses system (Quark PFT, Cosmed®, Rome, Italy) (Bonne et al. 2014). The VO_{2Peak} values were calculated using a 30 s backward extrapolation technique described elsewhere (Bonne et al. 2014; Latt et al. 2010). The VO_2 values were transformed into $\log VO_2$ and plotted against time. Through a linear regression, the y-intercept was considered as VO_{2Peak} (after the maximal 400 m effort).

Estimation of the contribution of the energy systems

The rectangular energetic demand (100% of the energy spent in the maximal effort of 400 m) was calculated through the product of VO_{2Peak} calculated at the end of the 400 m and the effort time (VO_{2Peak} × effort time) (Montpetit et al. 1981), as proposed by Jurimae et al. (2007). The VO_2 baseline was also retrieved from the rectangular energetic demand integral. The anaerobic contribution was calculated through the sum of anaerobic alactic (Ana_{ALA}) and anaerobic lactic (Ana_{LAC}) (Bertuzzi et al. 2010; Kalva-Filho et al. 2015; Zagatto et al. 2011). Ana_{ALA} was assumed as the fast component of excess post-oxygen consumption as used by others (Di Prampero and Margaria 1968; Margaria et al. 1933). The Ana_{LAC} was determined by net lactate accumulation (i.e., the difference between $[La^-]$ peak and rest values; $\Delta[La^-]$), considering a metabolic equivalent of 3 mL O_2^{-1} kg^{-1} for each unit of lactate elevated with maximal effort (Zamparo et al. 2011). The aerobic contribution was determined by the difference

between rectangular energetic demand and anaerobic contribution (Ana_{ALA} and Ana_{LAC}). These procedures were similar to those performed by others (Bertuzzi et al. 2010; Zagatto et al. 2011), including in swimming (Kalva-Filho et al. 2015). The contribution of the energy systems is presented in percentage (%) and kJ, considering 20.9 kJ L^{-1} O_2 (Zamparo et al. 2011).

Statistical analysis

Data normality was tested and confirmed by the Shapiro–Wilk test, permitting the use of parametric statistics. Data are presented as the mean and standard deviation (SD). To compare the placebo and taurine condition, the Student *t* test for paired samples was used. In all cases, a significance of 5% was adopted. The program utilized to perform the graphics was Origin.

Table 1 Requirements and intake of total energy, carbohydrate, protein and lipid of swimmers

	Requirements[a]	Intake
Energy (kcal)	3.684	3.448 ± 344
Carbohydrate (% TCI)	45–65	49.87 ± 6
Protein (% TCI)	10–35	20.32 ± 2
Lipid (%TCI)	20–35	28.41 ± 5

% TCI percentage of total calorie intake

[a]According to dietary reference intake

Results

The food intake and energy expenditure quantifications (Table 1) showed that the energy intake of the swimmers was similar to their energy requirements for carbohydrates, proteins, and lipids.

Regarding the taurine plasma levels 120 min after ingestion, we observed an increase of 159% when compared with the placebo baseline levels ($p = 0.001$). Individual taurine plasma levels PRE and POST maximal effort of 400 m for TAU and PLA conditions can be observed in Fig. 1.

Furthermore, taurine plasma levels after the maximal effort of 400 m in the placebo condition were significantly higher in comparison with the pre situation (mean values of taurine plasma levels in TAU condition PRE: 1192.08 ± 278.89 µmol L^{-1}; POST 1299.09 ± 330.91 µmol L^{-1}, $p = 0.19$; PLA condition PRE: 484.82 ± 89.36 µmol L^{-1}; POST 601.29 ± 81.31 µmol L^{-1}, $p = 0.01$). Even if the taurine supplementation promoted higher plasma taurine levels before the exercise protocol in the TAU PRE condition, these levels remained higher post-exercise (689.8 µmol L^{-1} higher than PLA POST), and no significant changes were observed ($p = 0.07$) when comparing the delta of variation of taurine plasma levels between pre and post for either condition (TAU and PLA).

The glycerol levels presented an increase in both groups post effort (TAU condition PRE 0.76 ± 0.15; POST 1.34 ± 0.21 µM, $p = 0.01$; PLA condition PRE 0.73 ± 0.09; POST 1.13 ± 0.18 µM, $p = 0.01$). Individual glycerol levels

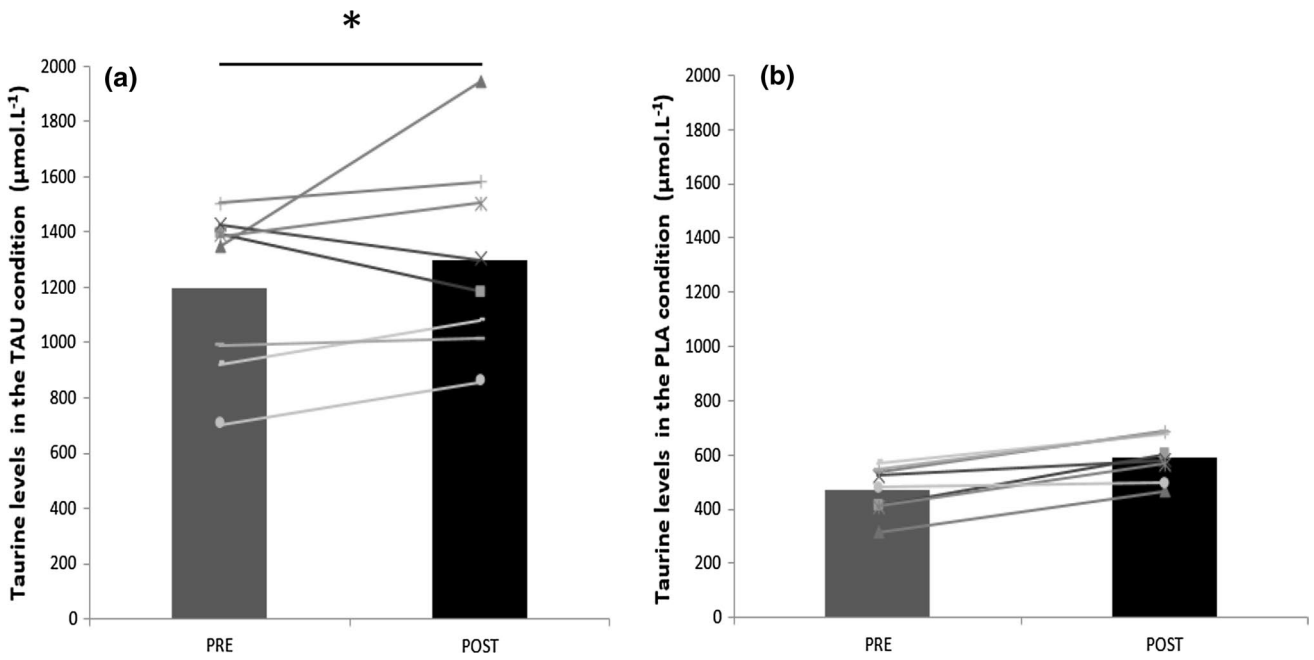

Fig. 1 Taurine levels (µmol.L^{-1}) for TAU (**a**) and PLA (**b**) conditions between PRE and POST maximal effort of 400m. *Significant higher in relation to PLA condition ($p = 0.011$)

Table 2 Individual glycerol levels PRE and POST maximum effort for both conditions ($n = 9$)

Athlete	TAU			PLA		
	PRE (μM)	POST (μM)	Δ (%)	PRE (μM)	POST (μM)	Δ (%)
1	0.9	1.32	31.8	0.68	0.86	20.9
2	0.78	1.33	41.4	0.7	1.27	44.9
3	0.87	1.34	35.1	0.6	0.94	36.2
4	0.75	1.47	49.0	0.6	0.96	37.5
5	0.87	1.23	29.3	0.78	1.24	37.1
6	0.77	1.74	55.7	0.8	1.25	36.0
7	0.52	1.03	49.5	0.83	1.37	39.4
8	0.9	1.47	38.8	0.8	1.04	23.1
9	0.51	1.12	54.5	0.8	1.2	33.3
Mean ± SD	0.75 ± 0.15	1.32 ± 0.21	43.24 ± 9.4[a]	0.73 ± 0.09	1.13 ± 0.17	35.34 ± 7.9

TAU taurine condition, *PLA* placebo condition, *PRE* before maximum effort, *POST* after maximum effort, *Δ* delta of variation between PRE and POST in percent

[a]Different from the placebo condition

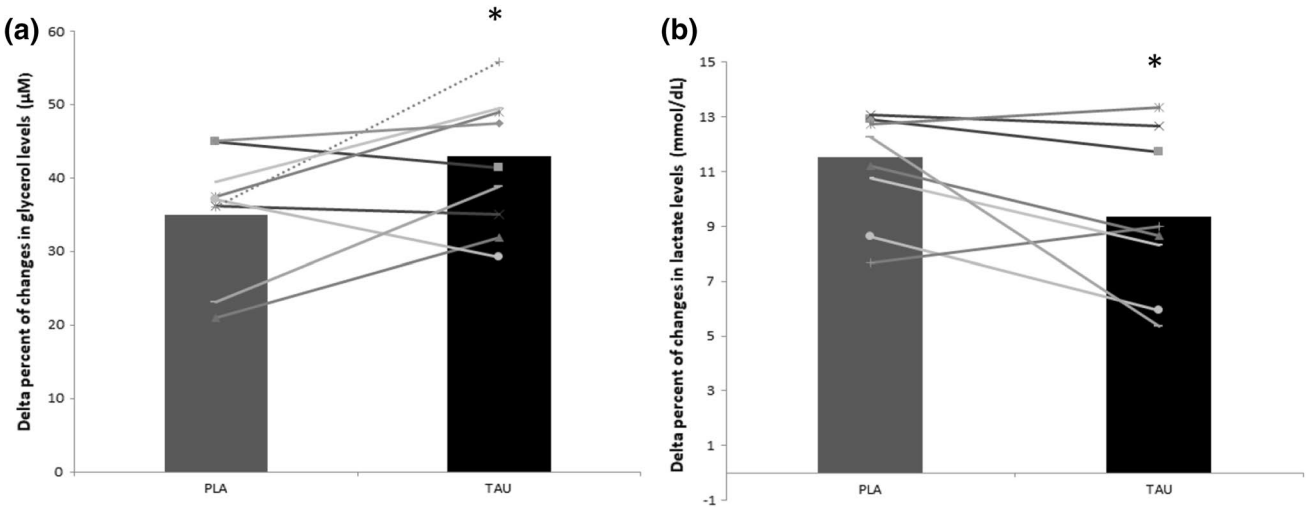

Fig. 2 Percentage of change in glycerol (**a**) and lactate (**b**) levels between PRE and POST maximal effort of 400 m for TAU and PLA conditions. *Significant increase in relation to PLA condition ($p < 0.05$)

PRE and POST maximum effort for both conditions can be observed in Table 2. However, the delta of variation of the glycerol levels was 8% higher for the TAU condition in comparison with the PLA condition ($p = 0.04$) (Fig. 2). Figure 2a shows the glycerol percentage change between PRE and POST 400 m maximal effort for both conditions.

Swimmer performance in the maximal effort of 400 m was not significantly different between the conditions (TAU condition: 282.74 ± 18.09 s and 1.42 ± 0.09 m s^{-1}; PLA condition 280.71 ± 17.41 s and 1.43 ± 0.08 m s^{-1}). Table 3 presents rest and peak (POST maximal effort of 400 m) lactate levels for both conditions.

Additionally, the $[La^-]_{rest}$ (TAU condition: 2.01 ± 0.38 mmol L^{-1}; PLA condition: 1.81 ± 0.42 mmol L^{-1}) and $[La^-]_{peak}$ (TAU condition: 11.37 ± 2.84 mmol L^{-1}; PLA condition: 13.33 ± 2.28 mmol L^{-1}) were not significantly different between the conditions; however, the Δ$[La^-]$ (TAU condition: 9.36 ± 2.78 mmol L^{-1}; PLA condition: 11.52 ± 2.19 mmol L^{-1}) was significantly different ($p = 0.04$). Figure 2b shows the percentage change in lactate levels between PRE and POST maximal effort of 400 m for both conditions. Consequently, the contribution of the lactic anaerobic metabolism for the TAU condition was significantly lower in comparison with the PLA

Table 3 Individual lactate levels PRE and POST maximum effort for both conditions ($n = 9$)

Athlete	TAU			PLA		
	Rest (mmol L^{-1})	Peak (mmol L^{-1})	Δ (%)	Rest (mmol L^{-1})	Peak (mmol L^{-1})	Δ (%)
1	2.20	10.86	8.66	1.20	12.42	11.22
2	2.23	13.95	11.72	2.26	15.17	12.91
3	1.65	14.31	12.66	1.71	14.79	13.08
4	1.92	15.27	13.35	1.92	14.66	12.74
5	1.83	7.79	5.96	1.52	10.16	8.63
6	2.87	11.85	8.98	2.38	10.04	7.66
7	1.90	10.22	8.31	1.43	12.21	10.78
8	1.61	6.96	5.35	1.63	13.91	12.27
9	1.88	11.12	9.24	2.27	16.62	14.35
Mean ± SD	2.01 ± 0.38	11.37 ± 2.84	9.36 ± 2.78	1.81 ± 0.42	13.33 ± 2.28	11.52 ± 2.19

TAU taurine condition, *PLA* placebo condition, *Rest* before maximum effort, *Peak* after maximum effort, *Δ* delta of variation between resting and peak in percent. No statistical changes were observed

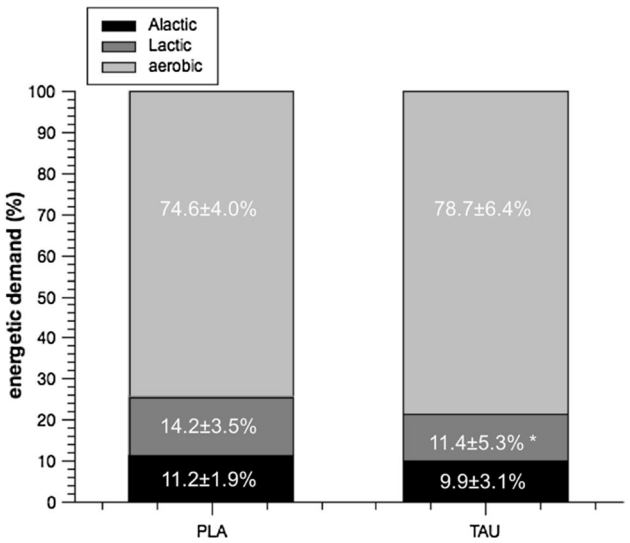

Fig. 3 Contribution of the energy systems on the taurine and placebo conditions. *Significant lower in relation to PLA condition ($p = 0.04$)

condition ($p = 0.04$). Figure 3 shows the contribution of the energy systems to the maximum effort of 400 m.

Discussion

The main purpose of this study was to verify the effects of acute taurine supplementation 120 min before performing a 400-m front crawl maximal effort on performance, lipid metabolism, and energy system contribution. Although our results showed that the ingestion of 6 g of taurine increased lipolysis (i.e., free glycerol levels) and reduced the contribution of the lactic anaerobic metabolism for maximal efforts of 400 m, athlete performance was not affected. Inappropriate food intake can directly affect the metabolism and performance (Kerksick et al. 2008); however, our data verified that all swimmers reached the energy intake requirements according to the dietary reference intake (ACSM 2016), which means that nutritional status did not influence their performance.

As previously described by other investigations (Ghandforoush-Sattari et al. 2010; Zhang et al. 2004), the dose of 6 g of taurine was sufficient to increase its plasma level. Interestingly, only the PLA condition presented a significant increase in the absolute values of plasma taurine when comparing pre and post maximal effort of 400 m. However, the delta of variation of the taurine plasma levels was not different in either condition (i.e., PLA vs. TAU), which suggests that taurine plasma levels immediately post-exercise were affected by training. In this way, the increase in taurine levels observed in the placebo condition could be associated with taurine release from muscles (Ward et al. 1999; Cuisinier et al. 2001).

Differently from these findings, Galloway et al. (2008) did not show differences in taurine plasma levels when comparing pre and post exercise for the placebo condition. Nevertheless, these studies used efforts with low intensity, while we used a maximal effort of 400 m. Thus, our findings suggest that high-intensity exercise may affect blood taurine levels. According to Ward et al. (1999) changes in the plasma amino acid content can be observed only immediately after the cessation of exercise and return to appropriate reference ranges 10 h after completion of the exercise. Furthermore, the researchers suggested that the speed performed, referred to as the intensity, rather than the duration of the exercise, is strongly correlated with elevated taurine levels and possibly demonstrates its release from muscle fibers (Ward et al. 1999).

However, the sources of taurine that can increase plasma content post-exercise are still unclear, and some studies suggest that higher taurine levels may reflect muscle damage and muscle fatigue, as well as changes in electrophysical properties of the muscle membrane, or an adaptation to changes in blood osmolarity such as the co-release with water to maintain plasma volume and calcium homeostasis (Ward et al. 1999, 2016; Spriet and Whitfield 2015).

Herein, taurine supplementation increased the glycerol plasma levels after the effort, indicating an enhancement in lipid oxidation. It is well described in the literature that lipid may be used as fuel during exercise, specifically triacylglycerol (TAG). In fact, when the exercise is initiated, the lipolysis rate and fatty acid release from the adipose tissue are increased due to β-adrenergic stimulation (Jeukendrup 2002). The breakdown of TAG results in the release of free fatty acids and glycerol (Saponaro et al. 2015). According to Pitsiladis et al. (1999), free glycerol cannot be re-esterified to adipose tissue due to the absence of glycerol kinase enzyme. Therefore, blood glycerol levels can be considered as an indirect measure of lipolysis in the human body (Jeukendrup et al. 1998), as it remains in circulation for a longer period than free fatty acids before being used in the glycolytic pathway or the liver gluconeogenesis (Maughan et al. 2010).

Since taurine can increase lipolysis, free fatty acid transport across the mitochondrial membranes, and possibly metabolism in the β-oxidation pathway (Kim et al. 2016; Rutherford et al. 2010), it may increase the aerobic energetic demand during exercise. However, manipulation of the skeletal muscle mitochondrial volume, which determines the overall capacity to oxidize fat, might also be considered an overarching control point (VanLoon et al. 2001).

Rutherford et al. (2010) demonstrated that acute supplementation of 1.66 g of taurine with a low-calorie drink 1 h before a prolonged cycling exercise increased by 16% in athletes' body fat oxidation when compared with the placebo group. The authors suggested that taurine improved fat oxidation during the effort through activation of adenylate cyclase and cyclic adenosine monophosphate (AMPc).

Tsuboyama-Kasaoka et al. (2006) supplemented rats with 5% taurine and observed an increase in the gene expression of the co-activator factor PGC-1α and of the peroxisome proliferator-activated receptor (PPAR) in their white adipose tissue. While PGC-1α and PPAR can increase energy expenditure and mitochondrial biogenesis, PGC-1α can also increase lipid substrates (Murakami 2015). In addition, taurine can increase the gene expression of lipoprotein lipase, acyl-CoA oxidase, acyl-CoA synthase, and acyl-CoA dehydrogenase, which are related to regulation of the oxidation of lipid substrates (Murakami 2015). In addition, higher taurine availability can modulate the respiratory chain and improve fatty acid metabolism by activating complex I generating adenosine triphosphate (ATP) (Schaffer et al. 2016). Thus, improvement in the electron transport chain can decrease lactate accumulation.

The improvements in lactate levels and performance can be explained by an interaction between taurine and the muscle membrane, by enhanced mitochondrial buffering, or by improvement in force production mediated through the increased calcium release to the contractile filament (Hansen et al. 2006; Dutka et al. 2014). Furthermore, as previously mentioned, higher taurine levels may reflect changes in muscle membrane or blood osmolarity and calcium homeostasis (Ward et al. 1999, 2016; Spriet and Whitfield 2015). According to Nakada et al. (1991), taurine can improve lactic acid buffering in brain cells and intramuscularly in rats. However, in human exercise performance studies, post-exercise plasma lactate remained largely unaltered by taurine supplementation (Lee et al. 2003; Rutherford et al. 2010), even in the presence of an improvement in performance (Balshaw et al. 2013).

Since taurine can increase lipid oxidation (Murakami 2015; Tsuboyama-Kasaoka et al. 2006) and the peak concentration of plasma taurine can be reached between 1.5 to 6 h post administration (Ghandforoush-Sattari et al. 2010), in the current investigation, the maximal effort of 400 m was performed within the period of peak taurine (i.e., 120 min post intake) and this higher availability of taurine in the blood during exercise promoted an additional effect on lipid metabolism. Regarding the relationship between taurine supplementation and performance improvement, one of the proposed theories indicated that this ergogenic resource can decrease blood lactate accumulation during exercise (Imagawa et al. 2009), which would influence the amount of energy provided by the glycolytic pathway during high-intensity exercise and, thereby, anaerobic capacity (Milioni et al. 2016).

Our data are partially consistent with the theory above, since the $\Delta[La^-]$ of the TAU condition was significantly lower compared to the PLA condition. However, we did not observe significant differences in the 400 m performance between the two conditions. The reduction in the lactic anaerobic system contribution during the maximal effort of 400 m for the TAU condition may be explained by the higher availability of glycerol, which possibly reduced glycolysis activation and increased the aerobic contribution. Interestingly, the aerobic contribution in the TAU condition was approximately 12% higher (not statistically significant) compared to the PLA condition. Thus, we consider that the participation of the oxidative metabolism in the ATP resynthesis for the TAU condition was higher than the PLA condition, which may be considered as an indicator of metabolic efficiency improvement.

This possible higher participation of the oxidative metabolism in the ATP resynthesis for the TAU condition would

allow a higher swimming economy (i.e., the response of the oxygen consumption to the same submaximal intensity) compared to the PLA condition. However, high-level swimming performance can be achieved with either low economy and high maximal oxygen consumption or high economy and lower maximal oxygen consumption (Unnithan et al. 2009; Termin and Pendergast 2001). Therefore, the higher participation of the oxidative metabolism in the ATP resynthesis for the TAU condition (i.e., an indicator of metabolic efficiency that would probably improve the swimming economy) does not necessarily imply in the improvement of maximal swimming performance (i.e., the maximal effort of 400 m). In accordance with our data, Rutherford et al. (2010) verified that an acute dose of taurine increased an indicator of metabolic efficiency improvement (i.e., total fat oxidation) without significant improvement in the maximal performance of well-trained cyclists. Future studies are necessary to verify whether this supplementation strategy may be more effective for high-intensity exercises with duration longer than 5 min. Furthermore, the VO_2max did not change between taurine and placebo condition (TAU: 61.56 ± 3.30 mL kg^{-1} min^{-1}; PLA: 59.28 ± 1.66 mL kg^{-1} min^{-1}; $p = 0.50$) and the energetic cost was not different between the conditions (TAU: 19.84 ± 1.24 L; PLA: 18.73 ± 0.85 L: $p = 0.35$).

An interesting study developed by Dutka et al. (2014) used mechanically skinned segments of human vastus lateralis muscle fibers to verify the effects of taurine on sarcoplasmic reticulum (SR) calcium (Ca^{2+}) accumulation and contractile apparatus properties in type I and II fibers. The authors observed that myoplasmic exposure to taurine during approximately 10 min led to a substantial increase in the Ca^{2+} accumulation rate by the SR in fibers type I and II, which may improve both contractile function and metabolic efficiency. The relationship between taurine and Ca^{2+} sensitivity of myofibrillar proteins is not limited to skeletal muscle cells. In an elegant investigation, Ramila et al. (2015) verified that taurine transporter knockout (TauTKO) mice hearts exhibited elevated protein phosphatase 1 activity as well as reduced calcium calmodulin-dependent protein kinase II autophosphorylation and phospholamban phosphorylation, which were responsible for the reduction in the SR Ca^{2+} ATPase activity. In addition, TauTKO mice hearts displayed a rightward shift in the Ca^{2+} dependence of the myofibrillar Ca^{2+} ATPase, a property connected to the upregulation of troponin I phosphorylation. Taken together, these data reinforced the observation that TauTKO mice hearts exhibited both systolic and diastolic impairs, which were related to SR Ca^{2+} ATPase activity reduction.

While Dutka et al. (2014) evaluated the direct effects of taurine in skeletal muscle samples, we used oral taurine supplementation. In fact, to guarantee that the 400-m front crawl maximal effort was performed with the peak levels of plasma taurine, our protocol was based on the results of Ghandforoush-Sattari et al. (2010). Therefore, it is important to point out that the availability of taurine for the muscle cells depends on the digestion process and nutrient metabolism, which may have influenced on the lack of performance improvement between TAU and PLA conditions. Recently, Milioni et al. (2016) also investigated whether the use of 6 g of taurine would improve the performance in high-intensity running and positively affect the anaerobic capacity measured by the method proposed by Bertuzzi et al. (2010). The authors did not verify significant improvements in running performance and showed an unclear effect on anaerobic capacity (Milioni et al. 2016). However, the improvements in performance can be explained by an interaction between taurine and the muscle membrane, by enhanced mitochondrial buffering, or by an improvement in force production mediated through the increased calcium release to the contractile filament (Hansen et al. 2006; Dutka et al. 2014).

Furthermore, according to Nakada et al. (1991) taurine can improve lactic acid buffering in brain cells and intramuscularly in rats. However, in human exercise performance studies, post-exercise plasma lactate remained largely unaltered by taurine supplementation (Lee et al. 2003; Rutherford et al. 2010), even in the presence of an observed improvement in performance (Balshaw et al. 2013). Despite the methodological limitations of the studies involving athletes and nutrition (i.e., reduced sample size, competition schedule, difficulty to perform invasive assessments), evaluation of the nutritional efficacy of ergogenic resources on performance and metabolic participation in sports is fundamental.

In conclusion, acute supplementation of 6 g of taurine 120 min before performing a 400-m front crawl maximal effort did not improve athlete performance; however, it increased glycerol plasma levels and reduced both the $\Delta[La^-]$ and lactic anaerobic system contribution. Therefore, this taurine dose seems to be an interesting supplementation strategy for high-intensity efforts as the increase in glycerol plasma levels may save muscle glycogen stores, increasing the recoverability of athletes for subsequent efforts.

Compliance with ethical standards

Ethical approval All procedures performed in studies involving human participants were 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.

Conflict of interest The authors declare no competing financial interests.

References

American College of Sports Medicine (2016) American Dietetic Association, and Dietitians of Canada. Nutrition and athletic performance. Med Sci Sports Exerc 32:2130–2145

Artioli GG, Bertuzzi RC, Roschel H, Mendes SH, Lancha AH Jr, Franchini E (2012) Determining the contribution of the energy systems during exercise. J Vis Exp 61:e3413. doi:10.3791/3413

Bakker AJ, Berg HM (2002) Effect of taurine on sarcoplasmic reticulum function and force in skinned fast-twitch skeletal muscle fibres of the rat. J Physiol 538(Pt 1):185–194. doi:10.1113/jphysiol.2001.012872

Balshaw TG, Bampouras TM, Barry TJ, Sparks SA (2013) The effect of acute taurine ingestion on 3-km running performance in trained middle-distance runners. Amino Acids 44(2):555–561. doi:10.1007/s00726-012-1372-1

Bertuzzi RC, Franchini E, Ugrinowitsch C, Kokubun E, Lima-Silva AE, Pires FO, Nakamura FY, Kiss MA (2010) Predicting MAOD using only a supramaximal exhaustive test. Int J Sports Med 31(7):477–481. doi:10.1055/s-0030-1253375

Bonne TC, Lundby C, Jørgensen S, Johansen L, Mrgan M, Bech SR, Sander M, Papoti M, Nordsborg NB (2014) "Live High-Train High" increases hemoglobin mass in Olympic swimmers. Eur J Appl Physiol 114(7):1439–1449. doi:10.1007/s00421-014-2863-4

Brisola GM, Miyagi WE, da Silva HS, Zagatto AM (2015) Sodium bicarbonate supplementation improved MAOD but is not correlated with 200- and 400-m running performances: a double-blind, crossover, and placebo-controlled study. Appl Physiol Nutr Metab 40(9):931–937. doi:10.1139/apnm-2015-0036

Carneiro EM, Latorraca MQ, Araujo E, Beltrá M, Oliveras MJ, Navarro M, Berná G, Bedoya FJ, Velloso LA, Soria B, Martín F (2009) Taurine supplementation modulates glucose homeostasis and islet function. J Nutr Biochem 20(7):503–511. doi:10.1016/j.jnutbio.2008.05.008

Carter SL, Rennie C, Tarnopolsky MA (2001) Substrate utilization during endurance exercise in men and women after endurance training. Am J Physiol Endocrinol Metab 280(6):E898–E907

Cuisinier C, Ward RJ, Francaux M, Sturbois X, de Witte P (2001) Changes in plasma and urinary taurine and amino acids in runners immediately and 24 h after a marathon. Amino Acids 20(1):13–23

Deyl Z, Hyanek J, Horakova M (1986) Profiling of amino acids in body fluids and tissues by means of liquid chromatography. J Chromatogr 379:177–250

Di Prampero PE, Margaria R (1968) Relationship between O_2 consumption, high energy phosphates and the kinetics of the O_2 debt in exercise. Pflugers Arch 304(1):11–19

Dutka TL, Lamboley CR, Murphy RM, Lamb GD (2014) Acute effects of taurine on sarcoplasmic reticulum Ca^{2+} accumulation and contractility in human type I and type II skeletal muscle fibres. J Appl Physiol 117:797–905. doi:10.1152/japplphysiol.00494.2014

Galloway SD, Talanian JL, Shoveller AK, Heigenhauser GJ, Spriet LL (2008) Seven days of oral taurine supplementation does not increase muscle taurine content or alter substrate metabolism during prolonged exercise in humans. J Appl Physiol 105(2):643–651. doi:10.1152/japplphysiol.90525.2008

Ghandforoush-Sattari M, Mashayekhi S, Krishna CV, Thompson JP, Routledge PA (2010) Pharmacokinetics of oral taurine in healthy volunteers. J Amino Acids 2010:346237. doi:10.4061/2010/346237

Hansen SH, Andersen ML, Birkedal H, Cornett C, Wibrand F (2006) The important role of taurine in oxidative metabolism. Adv Exp Med Biol 583:129–135. doi:10.1007/978-0-387-33504-9_13

Imagawa TF, Hirano I, Utsuki K, Horie M, Naka A, Matsumoto K, Imagawa S (2009) Caffeine and taurine enhance endurance performance. Int J Sports Med 30(7):485–488. doi:10.1055/s-0028-1104574

Institute of Medicine (2005) Dietary reference intakes: applications in dietary assessment. Washington DC

Jeukendrup AE (2002) Regulation of fat metabolism in skeletal muscle. Ann N Y Acad Sci 967:217–235. doi:10.1111/j.1749-6632.2002.tb04278.x

Jeukendrup AE, Saris WH, Wagenmakers AJ (1998) Fat metabolism during exercise: a review–part II: regulation of metabolism and the effects of training. Int J Sports Med 19(5):293–302. doi:10.1055/s-2007-971921

Jeukendrup AE, Jentjens RLPG, Moseley L (2005) Nutritional considerations in triathlon. Sports Med 35(2):163–181. doi:10.2165/00007256-200535020-00005

Jurimae J, Haljaste K, Cicchella A, Lätt E, Purge P, Leppik A, Jürimäe T (2007) Analysis of swimming performance from physical, physiological, and biomechanical parameters in young swimmers. Pediatr Exerc Sci 19(1):70–81. doi:10.1123/pes.19.1.70

Kalva-Filho CA, Zagatto AM, Araújo MI, Santiago PR, da Silva AS, Gobatto CA, Papoti M (2015) Relationship between aerobic and anaerobic parameters from 3-minute all-out tethered swimming and 400-m maximal front crawl effort. J Strength Cond Res 29(1):238–245. doi:10.1519/JSC.0000000000000592

Kerksick C, Harvey T, Stout J, Campbell B, Wilborn C, Kreider R, Kalman D, Ziegenfuss T, Lopez H, Landis J, Ivy JL, Antonio J (2008) International Society of Sports Nutrition position stand: nutrient timing. J Int Soc Sports Nutr 5:17. doi:10.1186/1550-2783-5-17

Kim J, Park J, Lim K (2016) Nutrition supplements to stimulate lipolysis: a review in relation to endurance exercise capacity. J Nutr Sci Vitaminol 62(3):141–161. doi:10.3177/jnsv.62.141

Latt E, Jürimäe J, Mäestu J, Purge P, Rämson R, Haljaste K, Keskinen KL, Rodriguez FA, Jürimäe T (2010) Physiological, biomechanical and anthropometrical predictors of sprint swimming performance in adolescent swimmers. J Sports Sci Med 9:398–404

Lee HM, Paik IY, Park TS (2003) Effects of dietary supplementation of taurine, carnitine, or glutamine on endurance performance and fatigue parameters in athletes. Korean J Nutr 36:711–719

Margaria R, Edwards H, Dill DB (1933) The possible mechanisms of contracting and paying the oxygen debt and the role of lactic acid in muscular contraction. Am J Physiol 106:689–715

Maughan RJ, Fallah J, Coyle EF (2010) The effects of fasting on metabolism and performance. Br J Sports Med 44(7):490–494. doi:10.1136/bjsm.2010.072181

Milioni F, Malta Ede S, Rocha LG, Mesquita CA, de Freitas EC, Zagatto AM (2016) Acute administration of high doses of taurine does not substantially improve high-intensity running performance and the effect on maximal accumulated oxygen deficit is unclear. Appl Physiol Nutr Metab 41(5):498–503. doi:10.1139/apnm-2015-0435

Montpetit RR, Léger LA, Lavoie JM, Cazorla G (1981) $\dot{V}O_2$ peak during free swimming using the backward extrapolation of the O_2 recovery curve. Eur J Appl Physiol Occup Physiol 47(4):385–391

Murakami S (2015) Role of taurine in the pathogenesis of obesity. Mol Nutr Food Res 59(7):1353–1363. doi:10.1002/mnfr.201500067

Nakada T, Hida K, Kwee IL (1991) pH-lactate dissociation during anoxic insult: taurine effect. NeuroReport 2(6):325–328

Pitsiladis YP, Smith I, Maughan RJ (1999) Increased fat availability enhances the capacity of trained individuals to perform prolonged exercise. Med Sci Sports Exerc 31(11):1570–1579. doi:10.1097/00005768-199911000-00012

Ramila KC, Jong CJ, Pastukh V, Ito T, Azuma J, Schaffer SW (2015) Role of protein phosphorylation in excitation-contraction coupling in taurine deficient hearts. Am J Physiol Heart Circ Physiol 308:H232–H239. doi:10.1152/ajpheart.00497.2014

Rutherford JA, Spriet LL, Stellingwerff T (2010) The effect of acute taurine ingestion on endurance performance and metabolism in well-trained cyclists. Int J Sport Nutr Exerc Metab 20(4):322–329

Saponaro C, Gaggini M, Carli F, Gastaldelli A (2015) The subtle balance between lipolysis and lipogenesis: a critical point in metabolic homeostasis. Nutrients 7(11):9453–9474. doi:10.3390/nu7115475

Schaffer SW, Jong CJ, Ramila KC, Azuma J (2010) Physiological roles of taurine in heart and muscle. J Biomed Sci 17(Suppl 1):S2. doi:10.1186/1423-0127-17-S1-S2

Schaffer SW, Shimada-Takaura K, Jong CJ, Ito T, Takahashi K (2016) Impaired energy metabolism of the taurine deficient heart. Amino Acids 48(2):549–558. doi:10.1007/s00726-015-2110-2

Spriet LL, Whitfield J (2015) Taurine and skeletal muscle function. Curr Opin Clin Nutr Metab Care 18(1):96–101. doi:10.1097/MCO.0000000000000135

Termin B, Pendergast DR (2001) Training using the stroke frequency-velocity relationship to combine biomechanical and metabolic paradigms. J Swim Res 14:9–17

Tomlin DL, Wenger HA (2001) The relationship between aerobic fitness and recovery from high intensity intermittent exercise. Sports Med 31(1):1–11. doi:10.2165/00007256-200131010-00001

Tsuboyama-Kasaoka N, Shozawa C, Sano K, Kamei Y, Kasaoka S, Hosokawa Y, Ezaki O (2006) Taurine (2-aminoethanesulfonic acid) deficiency creates a vicious circle promoting obesity. Endocrinol 147(7):3276–3284. doi:10.1210/en.2005-1007

Unnithan V, Holohan J, Fernhall B, Wylegala J, Rowland T, Pendergast DR (2009) Aerobic cost in elite female adolescent swimmers. Int J Sports Med 30(3):194–199. doi:10.1055/s-0028-1104583

VanLoon LJ, Greenhaff PL, Constantin-Teodosiu D, Saris WH, Wagenmakers AJ (2001) The effects of increasing exercise intensity on muscle fuel utilization in humans. J Physiol 536:295–304. doi:10.1111/j.1469-7793.2001.00295.x

Ward R, Francaux M, Cuisinier C, Sturbois X, De Witte P (1999) Changes in plasma taurine levels after different endurance events. Amino Acids 16:71–77

Ward R, Bridge CA, McNaughton LR, Sparks SA (2016) The effect of acute taurine ingestion on 4-km time trial performance in trained cyclists. Amino Acids 48(11):2581–2587. doi:10.1007/s00726-016-2282-4

Zagatto A, Redkva P, Loures J, Kalva Filho C, Franco V, Kaminagakura E, Papoti M (2011) Anaerobic contribution during maximal anaerobic running test: correlation with maximal accumulated oxygen deficit. Scand J Med Sci Sports 21(6):e222–e230. doi:10.1111/j.1600-0838.2010.01258.x

Zamparo P, Capelli C, Pendergast D (2011) Energetics of swimming: a historical perspective. Eur J Appl Physiol 111(3):367–378. doi:10.1007/s00421-010-1433-7

Zhang M, Izumi I, Kagamimori S, Sokejima S, Yamagami T, Liu Z, Qi B (2004) Role of taurine supplementation to prevent exercise-induced oxidative stress in healthy young men. Amino Acids 26(2):203–207. doi:10.1007/s00726-003-0002-3