Taurine supplementation improves economy of movement in the cycle test independently of the detrimental effects of ethanol

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ABSTRACT: Taurine (TA) ingestion has been touted as blunting the deleterious effects of ethanol (ET) ingestion on motor performance. This study investigated the effects of ingestion of 0.6 mL·kg^{-1}of ET, 6 grams of TA, and ethanol in combination with taurine (ET+TA) on economy of movement (EM) and heart rate (HR). Nine volunteers, five female (22 ± 3 years) and four male (26 ± 5 years), participated in a study that used a counterbalanced experimental design. EM and HR were measured for 6 min while the subjects were pedalling at a fixed load 10% below the anaerobic threshold. The blood alcohol concentration (BAC) was similar between ET and ET+TA treatments at 30 min after ingestion and after exercise (12.3 mmol·L^{-1} vs. 13.7 mmol·L^{-1}, and 9.7 mmol · L^{-1} vs 10.9 mmol·L^{-1}, respectively). EM was significantly different among treatments, with lower mL·W^{-1} following ingestion of TA (-7.1%, $p<0.001$) than placebo and ET+TA (-2.45%, $p=0.001$) compared to ET. HR (bpm) was significantly ($p<0.05$) higher for ET (137 ± 14 bpm) than the other three treatments (placebo = 129 ± 14 bpm; TA = 127 ± 11 bpm; TA+ET = 133 ± 12 and ET = 137 ± 14 bpm). Taurine improved EM when compared to placebo or ET, and reduced HR when compared to ET. The combination of ET+TA also enhanced EM compared to placebo, and reduced HR in comparison to ET alone. Therefore, these findings indicate that taurine improves EM and counteracts ethanol-induced increases in HR during submaximal exercise.

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INTRODUCTION

Ethyl alcohol (ethanol) is the most consumed psychoactive drug worldwide, constituting a public health problem [1]. Among sportspeople, from college to elite athletes, high consumption of ethanol is not uncommon, mainly when considering team sports [2, 3]. Beyond affecting personal health, ethanol can also diminish physical performance, depending on the dose consumed and on the exercise type. Acute ethanol intake can negatively influence several neuromotor and metabolic mechanisms [4-6]. Ethanol consumption leads to loss of muscle strength due to the inhibition of sarcolemmal calcium channel actions and can also affect thermoregulation and hydration. In addition, ethanol inhibits the gluconeogenesis precursors associated with reduced muscle glycogen storage. Ethanol also negatively affects neurological functions and, therefore, the motor unit recruitment patterns [7-9]. The acute effects of ethanol may also influence cardiovascular system functions, inducing, for instance, arrhythmia and atrial fibrillation, which will consequently be related to EM distortions [10].

In turn, administration of the amino acid taurine (2-aminoethane sulfonic acid) can positively modulate endurance performance [11]. It has been suggested that taurine may alter calcium transport and

osmoregulation in muscle, resulting in enhanced mitochondrial buffering and improved contractile filament sensitivity and force production [12, 13]. These effects can improve motor and cardiovascular functions and, therefore, endurance performance [14].

The ingestion of energy drinks containing taurine in combination with *alcoholic* beverages has been used to reduce the depressant effects of ethanol [15], although some research did not report these results [16]. Oxygen consumption during exercise has been evaluated after ingestion of mixed energy drinks and alcohol [17], but the short time of the intake prior to the exercise (<1 hour) is not sufficient to analyze the acute effect of the influence of taurine, since the peak plasma concentration of this amino acid occurs at approximately two hours after intake [18]. Despite the use of taurine in the treatment of the deleterious effects of chronic ethanol abuse on the liver [19], the acute physiological effects of the ingestion of these two substances together are not clear. EM is multifaceted, with metabolic, cardiopulmonary, biomechanical and neuromuscular components. Taurine may positively modulate several of these components (muscle efficiency, mitochondrial metabolism, calcium homeostasis, antagonistic effects to ethanol, among others). Thus exogenous administration of taurine may improve EM and reduce cardiovascular stress during submaximal exercise [20-22].

Accordingly, this study aimed to determine the acute effects of taurine and ethanol intake on EM. The secondary aim was to assess the effects of these substances on the heart rate (HR) responses to mild to moderate exercise.

MATERIALS AND METHODS

Subjects

A total of nine volunteers, five of whom were females, with the following features participated in this study: aged 24 ± 4 years, height 1.67 ± 0.11 m, body mass 62.6 ± 13.6 kg and fat mass 8.5 ± 3.6 kg; the volunteers were apparently healthy and *regularly* practiced *physical activity;* they were non-smokers and non-athletes and they had a $\dot{V}O_{2\ max}$ of 42.0 ± 5.8 mL·kg^{-1}·min^{-1}. The study was performed in a repeated measures design to control the biological variability between subjects. Thirty-six observations, four replicate measures of each subject, were performed. The statistical power was 0.75 and the sample size for a significance level of 0.5 was 29 [23]. Individuals with liver disorders (as determined by the activity of the enzymes aspartate aminotransferase, alanine aminotransferase, direct and indirect bilirubin, alkaline phosphatase, gamma-glutamyl transferase and lactate dehydrogenase) were excluded from the study after analysis of the first blood samples. Individuals taking medica-

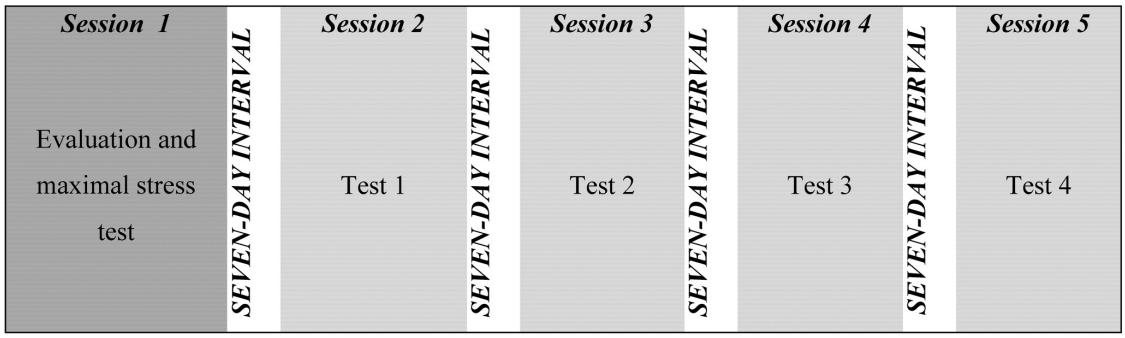

FIG 1. Schematic diagram of the study design

tions and alcohol users with a weekly intake greater than fifteen or less than two servings were also excluded.

The participants were asked to avoid physical activities with more than 5 metabolic equivalents (METs) and foods/drinks containing caffeine and taurine 48 hours before the tests. Volunteers read and signed an informed consent form. All experimental procedures were approved by the local ethics committee on human research (03899312.5.0000.5257).

Experimental design

First, the volunteers were submitted to a stress test to determine their anaerobic threshold and maximal oxygen uptake. After sample characterization, the experimental interventions were performed as shown in Figure 1.

Six grams of microcrystalline cellulose were diluted in 0.150 L of an orange-flavoured drink containing 0% carbohydrate (Clight juice powder, 21 $g \cdot L^{-1}$) and used as the placebo solution. Similarly, six grams of powdered taurine (99.3%) (Galena, Campinas, Brazil) were combined with orange-flavoured drink for the experimental solution. Ethanol (*Orloff* – 38% alcohol content) was administered in doses of 0.6 $mL \cdot kg^{-1}$, combined with the ingestion of an orange-flavoured drink in a proportion of 2:1 (juice:vodka).

Taurine (TA) and placebo solutions were ingested 120 minutes before the exercise, while the alcoholic beverage (ET) was ingested thirty minutes before the exercise. As the peak plasma level of TA [18] occurs at about 120 minutes and ethanol at 30 minutes after intake [24], in the taurine + ethanol treatment (TA+ET), ethanol was ingested 90 minutes after the administration of taurine, and the exercise was performed thirty minutes after the ingestion of ethanol. Taurine administration followed a simple double-blind *procedure*.

Ergometric protocol

Metabolism was analyzed using open-circuit indirect calorimetry (Vista Mini-CPX, Vacumed, Ventura, CA, USA). The minute ventilation ($\dot{V}_M$) was measured using a flow sensor with dynamic resistance. The fraction of expired oxygen was measured using a cold fuel cell system, while the fraction of carbon dioxide was measured using an infrared sensor, according to the manufacturer's specifications. The subjects used a silicone mask (V-Mask, Hans Rudolph Inc., Kansas City, MO, USA) attached to a *turbine* with *bidirectional* gas *flow* ("MIR" Turbine, Vacumed, Ventura, CA, USA). A cycle ergometer with an electromagnetic brake (Imbrasport, Porto Alegre, RS, Brazil) was used to apply the load.

Initially, a graded maximal and continuous exercise protocol (GXT) was used to determine maximal power output (W_{max}), maximal oxygen uptake ($\dot{V}O_{2\ max}$) and anaerobic threshold. The anaerobic threshold (AnT) was determined by pulmonary ventilation responses, using the simplified V-slope and ventilatory equivalent methods [25-27]. Maximal oxygen uptake ($\dot{V}O_{2\ max}$) was determined by three of the following test criteria: a) VO_2 plateau ≤ 150 $mL \cdot min^{-1}$; b) respiratory exchange ratio (RER) ≥ 1.10; c) estimated maximal heart rate ≥ 90%; d) rating of perceived exertion chart ≥ 19 (6-20); e) blood lactate ≥ 8 $mmol \cdot L^{-1}$ [28]. The test started with the subject seated on a cycle ergometer for six minutes, followed by four minutes of *pedalling without* any load. The next step was the *graded* phase (ramp) and was performed according to procedures described by Nogueira and Pompeu [29]. The perceived exertion was determined using the Borg scale (*6-20 point scale)* at the end of each minute [30].

A constant-load protocol (SWT) was adopted in the EM tests. The resting and warm-up procedures used in the graded exercise test (GXT) were used in this protocol. An intensity 10% lower than the load of the anaerobic threshold was subsequently determined and maintained for 10 minutes.

In both tests, a cadence of 1 Hz was used and controlled through an audio-visual metronome (Wittner Junior Plast 826, Isny/Allgäu, Germany). HR was continuously measured with a monitor (Vantage NV, Polar ElectroOy, Kempele, Finland). $\dot{V}_M$, oxygen consumption per minute ($\dot{V}O_2$) and carbon dioxide production per minute ($\dot{V}CO_2$) were sampled online in breath-by-breath mode, recording one breath every eleven in the maximal stress test, and the means were obtained at a 30-second interval in the SWT. The collected signals were integrated through Vista Turbo Fit 5.1 software (Ventura, CA, USA).

The EM was analyzed using the $\dot{V}O_2$ $(ml) \cdot L^{-1}$ values obtained during six minutes of exercise, disregarding both the initial and the final two minutes. The same time interval was used to calculate the mean HR.

TABLE 1. Plasma ethanol concentrations.

Treatment	Plasma ethanol concentration ($mmol \cdot L^{-1}$)				
	Pre-ingestion	30 min after the ingestion	*P* value	Post-exercise	*P* value
ET	0	12.3 ± 4.3**	0.001	9.7 ± 2.0 **	0.001
TA+ET	0	13.7 ± 2.9**	0.001	10.9 ± 1.4**	0.001

Values are expressed as means and SDs. ** Significant difference compared to the baseline for each treatment.

Blood collection and biochemical analysis

To assess the plasma ethanol concentrations, three samples (20 mL) were collected in each treatment (ET and ET+TA). The first sample was collected at the beginning of the experiment. The second sample was collected thirty minutes after the ingestion of ethanol and the third immediately after the exercise. The venous blood was obtained from the *median cubital vein* of the forearm and was collected with and without sodium fluoride (BD Vacutainer Fluoride/EDTA) to obtain serum or plasma. Plasma ethanol was analyzed with an automated chemiluminescence apparatus (Siemens Dimension Series). Liver enzymes were measured in the serum by dry chemistry using the VITROS Chemical System (Ortho-Clinical Diagnostics – Johnson & Johnson).

Controls and calibrations

The metabolic analyzer was calibrated before each test with a certified gas mixture containing 16% oxygen and 4.1% carbon dioxide and balanced with nitrogen (Linde, Br.). For the control, the fractions of oxygen and carbon dioxide were measured in the mixture used for calibration at the end of each test.

The ethanol assays were calibrated by measuring three different concentrations (0, 21.7 and 65.1 mmol·L^{-1}) of commercial aqueous ethanol solutions (Siemens Dimension, Ref: DC37A) in triplicate. The assay's quality was assessed using commercial control serum (21.7 mmol·L^{-1} ethanol – Siemens Dimension, Ref: #545) in quintuplicate (coefficient of variation [CV] = 0.18%). For liver enzymes, the assay was calibrated for each batch of reagents using a commercial calibrator kit specific for hepatography (VITROS Calibrator *Kit* III). The quality control was performed before the tests using control sera with normal and abnormal levels for liver parameters.

Data analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, USA), SigmaPlot (Systat Software Inc., Chicago IL, USA) and Microsoft Excel for Windows (Microsoft, Redmond, WA, USA). Descriptive statistics with means ± standard deviations (SDs) were used. Data normality and sphericity were tested in all analyses using the Shapiro-Wilk and Mauchly's Shapiro-Wilk tests, respectively. When the null hypothesis was rejected in the normality or sphericity tests, non-parametric statistics were then used by applying the Friedman test for rank-ordered samples and the Wilcoxon test for post hoc multiple comparisons between treatments. In the dependent variables $\dot{V}O_2$ and HR, non-parametric statistics was used. *Two-way ANOVA* with repeated measures and a post hoc Bonferroni test were used in the comparisons between plasma ethanol concentrations (treatment *vs* time-point). The Greenhouse-Geisser correction were applied in ANOVA with repeated measures. The level of significance was set at $p \leq 0.05$.

RESULTS

Plasma ethanol concentrations from the ET and TA+ET treatments are shown in Table 1. According to the two-way ANOVA (treatment vs time-point), a main effect was detected for the time-point variable ($F = 137.19$; $p = 0.001$). Significant increases in the plasma ethanol concentration were observed in both treatments. However, there were no significant differences between treatments at the specific time-points (pre-ingestion, 30 after ingestion and after exercise). The blood alcohol concentration was similar between genders, both in ET ($F = 1.509$; $p = 0.307$) and ET+TA ($F = 0.049$; $p = 0.837$).

Figure 2 presents EM among treatments. The Wilcoxon signed ranks test showed that EM was similar (p=0.662) between ET (18.9 ± 1.45 mL·W^{-1}) and Placebo (18.7 ± 1.79 mL·W^{-1}),

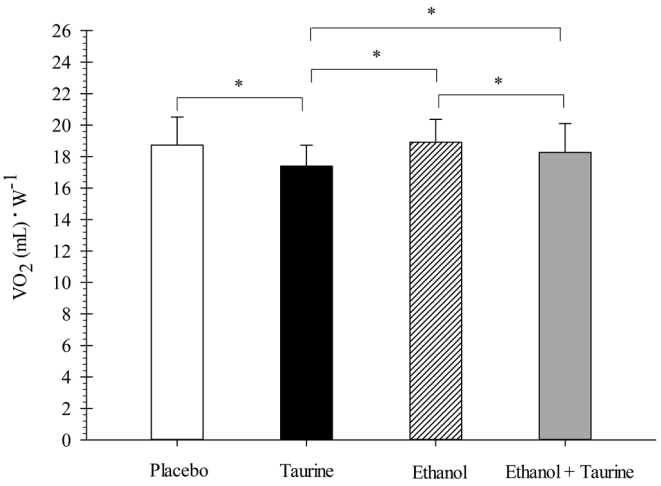

FIG 2. Economy of movement. Values are expressed as means and standard deviations.
* Significant difference ($p \leq 0.05$)

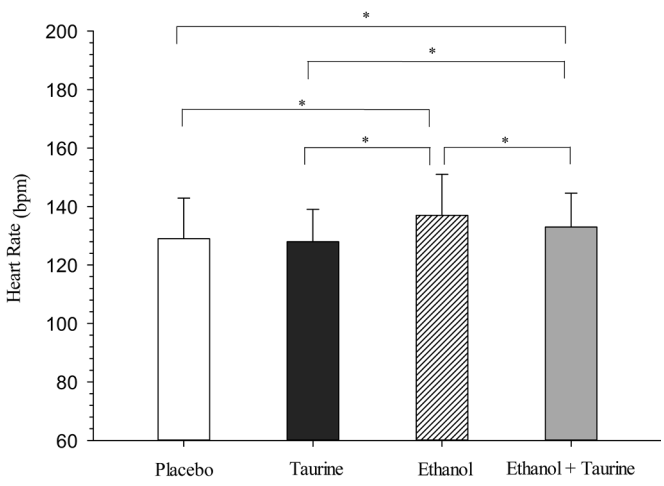

FIG 3. Heart rate during exercise. Values are expressed as means and standard deviations.
* Significant difference ($p \leq 0.05$).

and enhanced (-7.1% vs. Placebo, $p<0.001$) with TA (17.4 ± 1.32 mL · W^{-1}) and TA+ET (-2.45% vs. Placebo, 17.39 ± 1.33 mL·W^{-1}, $p=0.003$). HR was significantly higher in the ET treatment compared to the other treatments ($p = 0.001$). HR was significantly lower when ethanol was ingested together with taurine ($p = 0.001$) compared to ET alone (Figure 3).

DISCUSSION

This study addressed the acute effects of the ingestion of taurine, ethanol and the combination of these substances on motor performance and HR during submaximal exercise. The main findings were a positive effect of taurine intake on EM and submaximal HR. ET ingestion raised exercise HR as compared to placebo, but did not alter EM.

Acute ethanol intake may impair physical performance [24]. Suter and Schutz [6] showed that ingestion of 0.5 g of ethanol per kilogram of lean body mass promoted a 4% decrease in power output in a 60-min time trial at a work intensity corresponding to 80% of $\dot{V}O_{2\,max}$. Conversely, acute ethanol intake (1.48–1.59 g · kg^{-1}) did not affect the isokinetic and isometric endurance performances in 20 men and women [31]. EM analysis can contribute to elucidating the contradictory results available in the literature. The present study revealed that the ingestion of 0.6 mL·kg^{-1} of ethanol during submaximal exercise below the anaerobic threshold did not affect the EM (Figure 2). However, it should be highlighted that the relationship between physical performance and acute ethanol intake can also be dose-dependent [32] and that the dose of ethanol differed among most of the studies on this subject [24, 31].

In contrast, taurine ingestion has been used to improve endurance performance [22]. However, the acute effects of this amino acid are not well established, and the evidence on taurine's efficacy is mixed. Most studies are confounded because they have examined taurine ingestion concurrent with other active ingredients (caffeine, glucuronolactone) in commercially available energy drinks [33-35]. Some evidence supports an ergogenic effect of taurine in isolation, [20] while others refute it [36]. Balshaw and Bampouras [20] observed an increase in 3 km run time trial performance in athletes who were supplemented with 1 g of taurine two hours before exercise. These authors stated that the mechanism behind taurine's efficacy was "unknown", but found that VO_2 was similar between conditions, despite a faster running speed after taurine ingestion, suggestive of an increase in EM. In contrast, Milioni and Malta [36] found no benefit of 6 g of prior taurine ingestion for either time to exhaustion or maximal accumulated oxygen deficit during a supramaximal (110% of $\dot{V}O_{2\,max}$) run. The present data support the findings of Balshaw and Bampouras [20], finding a taurine-induced improvement in EM. Thus it appears that any beneficial effect of taurine on exercise performance might be limited to submaximal exercise conditions.

The combined administration of ethanol and taurine is popularly used as a recreational drink to increase sensations of *pleasure* and to *reduce* the intensity of some *depressant effects* of *ethanol* on the nervous system [15]. In addition, studies have shown that the $\dot{V}O_{2\,max}$ does not significantly change with the ingestion of ethanol when it is or is not combined with energy drink intake [17]. Taurine is associated with ethanol metabolism by accelerating the activity of the liver enzyme aldehyde dehydrogenase, which might constitute one mechanism responsible for its antagonistic effect to ethanol, in addition to the physiological mechanisms. Watanabe and Hobara [37] detected an increase in acetaldehyde metabolism in mouse livers when ethanol was administered together with taurine. Thus, the current study analyzed EM and HR and found that taurine blunted the ethanol-induced rise in HR during exercise.

Ethanol and taurine may impact cardiovascular function during exercise. Tachycardia, peripheral vasodilatation and intravascular volume depletion are the main acute symptoms of ethanol intake [10]. In the present study, HR increased during exercise in the ET treatment (Figure 3); this is in agreement with other studies in the literature [17, 38]. In contrast, taurine may increase stroke volume and end-diastolic volume and may reduce HR [14, 39]. The present study found that the acute effect of taurine attenuated the *alcohol-induced HR* increase, but TA alone did not change HR from the placebo condition. The lack of difference in exercise HR between taurine and placebo is consistent with previous research [20].

The mechanism by which taurine enhanced EM is unknown, but may be attributable to taurine's documented neural effects. The present study demonstrates that EM was enhanced when taurine was ingested together with ethanol (Figure 2). Thus, while ethanol has toxic effects, taurine possibly inhibits the influx of free [Ca^{2+}], thus reducing neuronal excitotoxicity and preserving mitochondrial activity [40, 41]. These physiological effects are important for controlling cell osmolarity and *mitochondria*l [Ca^{2+}] homeostasis [40, 42], thus resulting in better contractile sensitivity in response to [Ca^{2+}] and development of muscle tension; we speculate that those mechanisms are important for normal muscle contraction and EM [43]. In addition, individuals who had received previous treatments with taurine exhibited lower excitotoxicity of glutamate receptors [40, 42]. Studies have demonstrated that concentrations of the amino acid taurine increased the most in brain regions following ethanol injection [44]. Accordingly, because of its diverse physiological effects, it has been suggested that taurine is part of a physiological adaptation to oppose the toxic effects of ethanol.

The main limitations of this study were: 1) lack of a strict standardized food intake programme; 2) the small amount of ethanol administered in comparison to other studies; 3) the results of non-athletes cannot be totally extrapolated for athletes; and 4) although we mixed vodka with an orange-flavoured drink to mask the taste of ethanol, we were not able to insert the double-blind design for ethanol, because of the characteristic taste and specific physiological changes of this substance. New studies are warranted to cover this topic.

In conclusion, taurine intake reduces the deleterious effects caused by acute ethanol intake, attenuating ethanol-induced increases in

exercise HR. In addition, taurine can promote an increase in submaximal endurance performance by improving EM.

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Disclosure statement

No potential conflict of interest was reported by the authors.

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