The Effect of Acute Taurine Ingestion on Endurance Performance and Metabolism in Well-Trained Cyclists

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This study examined whether acute taurine (T) ingestion before prolonged cycling would improve time-trial (TT) performance and alter whole-body fuel utilization compared with a control (CON) trial and a placebo (PL) trial in which participants were told they received taurine but did not. Eleven endurance-trained male cyclists (27.2 ± 1.5 yr, 74.3 ± 2.3 kg, 59.9 ± 2.3 ml · kg⁻¹ · min⁻¹; M ± SEM) completed 3 trials in a randomized, crossover, blinded design in which they consumed a noncaloric sweetened beverage with either 1.66 g of T or nothing added (CON, PL) 1 hr before exercise. Participants then cycled at 66.5% ± 1.9% VO₂max for 90 min followed immediately by a TT (doing 5 kJ of work/kg body mass as fast as possible). Data on fluid administration, expired gas, heart rate, and ratings of perceived exertion were collected at 15-min intervals during the 90-min cycling ride, but there were no differences recorded between trials. There was no difference in TT performance between any of the 3 trials (1,500 ± 87 s). Average carbohydrate (T 2.73 ± 0.21, CON 2.88 ± 0.19, PL 2.89 ± 0.20 g/min) and fat (T 0.45 ± 0.05, CON 0.39 ± 0.04, PL 0.39 ± 0.05 g/min) oxidation rates were unaffected by T supplementation. T ingestion resulted in a 16% increase (5 g, ~84 kJ; p < .05) in total fat oxidation over the 90-min exercise period compared with CON and PL. The acute ingestion of 1.66 g of T before exercise did not enhance TT performance but did result in a small but significant increase in fat oxidation during submaximal cycling in endurance-trained cyclists.

Keywords: ergogenic aid, respiratory measurements, cycling performance, time trial

Taurine, or 2-aminoethanesulfonic acid, is a conditionally essential sulfur-containing amino acid. It is the most abundant free amino acid in heart, brain, leukocytes, and skeletal muscle (for reviews see Hayes & Sturman, 1981; Huxtable, 1992). Given taurine’s ubiquitous presence, it has been suggested to be involved in a wide range of metabolic processes such as cell volume regulation (Guizouarn, Motais, Garcia-Romeu, & Borgese, 2000), Ca²⁺-dependent excitation-contraction processes for optimal force development (Galler, Hutzler, & Haller, 1990), antioxidant defense from stress responses (Zembron-Lacny, Szyszka, & Szygula, 2007; Zhang et al., 2004), and modulation of nerve excitation potential (Davison & Kaczmarek, 1971).

Taurine is currently claimed as a functional ingredient (~1,000–2,000 mg taurine per serving) in more than 10 commercialized “energy” drinks, with many manufacturers claiming it has numerous ergogenic effects. However, rigorous scientific evidence to support these claims is lacking; most previous studies examined the metabolic, cognitive, and performance effects of taurine supplements in combination with many additional ingredients (e.g., caffeine) or did not use appropriate isocaloric or isocarbohydrate placebo control beverages (Alford, Cox, & Wescott, 2001; Barthel et al., 2001; Baum & Weiss, 2001; Forbes, Candow, Little, Magnus, & Chilibeck, 2007; Geiss, Jester, Falke, Hamm, & Waag, 1994; Jester, Grigereit, Bernhardt, Heil, & Banzer, 1997; Seidl, Peryl, Nicham, & Hauser, 2000). Therefore, a definitive role for taurine to potentially improve exercise performance or alter metabolism in humans remains to be clarified.

Our laboratory recently examined plasma taurine kinetics after a single acute dose of taurine, as well as the chronic effects of 7 days of taurine supplementation on skeletal-muscle taurine content and substrate metabolism during 2 hr of submaximal cycling (Galloway, Talanian, Shoveller, Heigenhauser, & Spriet, 2008). The acute taurine supplementation caused a 13-fold increase in plasma taurine lasting ~2.5 hr, but 7 days of taurine supplementation did not alter skeletal-muscle taurine content or substrate utilization during submaximal exercise. However, this study and many previous studies did not examine the effects of an acute dose of exclusively taurine taken immediately before exercise on subsequent metabolic and performance outcomes.

Therefore, we undertook the current investigation to examine the effects of an acute dose of taurine on endurance time-trial performance and whole-body metabolism in well-trained cyclists. We hypothesized that 1.66 g of taurine, ingested in a noncaloric sweetened bever-
age before exercise, would not alter whole-body fuel utilization or improve endurance time-trial performance compared with control ingestion of just the sweetened beverage. We also hypothesized that a placebo trial, in which participants were told they received taurine in their drink but in reality did not, would increase time-trial performance without altering whole-body fuel utilization.

Methods

Participants

Eleven endurance-trained male athletes (cyclists and triathletes) volunteered to participate in the study. They were engaged in regular endurance training (>9 cycling hr/week) before, and during, the testing period and none were using any conflicting medications. Their mean (± SE) age, weight, and maximal oxygen uptake (VO2max) were 27.2 ± 1.5 years, 74.3 ± 2.3 kg, and 59.9 ± 2.3 ml·kg⁻¹·min⁻¹, respectively. All participants were informed of the experimental protocol and associated risks of the study, both orally and in writing, before written informed consent was obtained. The University of Guelph Human Research Ethics Board approved the study.

Preexperimental Protocol

Participants initially underwent a continuous incremental cycle test to exhaustion to determine maximal pulmonary oxygen uptake (VO2max; Vmax 229°C, SensorMedics, Yorba Linda, CA) on a cycle ergometer (Lode Instruments, Groningen, The Netherlands). After the VO2max test, participants visited the laboratory on five more occasions: twice for practice trials and three visits for the experimental protocol. Visits to the laboratory were separated by at least 1 wk, and, during all trials, participants used their own bike seats and pedals for consistency between trials. Two practice trials were used to familiarize the participants with the exercise protocols and time trial for improved reliability and to confirm the ~65% VO2max power output during steady-state cycling. The mean (± SE) relative exercise intensity for all the trials was 66.5% ± 1.9% VO2max, which corresponded to an absolute power output of 195.9 ± 7.1 W.

During the first practice trial, participants cycled for 30 min at ~65% VO2max, to confirm or adjust the required power output. They then immediately began the first practice time trial, which took approximately 25 min and required them to complete 5 kJ of work/kg body mass (BM). The second practice trial replicated the experimental protocol in full and consisted of a 90-min steady-state ride at ~65% VO2max and the subsequent time trial. Expired pulmonary gases were sampled during six time intervals: from 12 to 15, 27 to 30, 42 to 45, 57 to 60, 72 to 75, and 87 to 90 min. Heart rate (HR) and rating of perceived exertion (RPE) were taken immediately after gas sampling at 15, 30, 45, 60, 75, and 90 min. HR was recorded using a Polar heart-rate monitor, and RPE was recorded using the modified Borg scale (Borg, 1973). Immediately after gas sampling, and throughout the 90-min ride at 15, 30, 45, 60, 75, and 90 min, a commercial carbohydrate (CHO) sports drink (Gatorade: 6.3% CHO, 18 mmol/L sodium) was consumed within ~5 min to ensure proper fluid (5 ml/kg, 371 ± 12 ml) and carbohydrate (23.4 ± 0.7 g CHO) replacement throughout the trial. The total fluid and CHO consumption during the 90-min steady-state-cycling session were matched for all three trials and amounted to 2.23 ± 0.69 L and 140.3 ± 4.3 g, respectively.

Whole-body rates of CHO and fat oxidation (g/min) were calculated during the steady-state cycling from the rates of CO2 production (VCO2) and O2 consumption (VO2) using the nonprotein respiratory-exchange ratio (RER) values according to the following equations (Perronnet & Massicotte, 1991):

CHO oxidation (g/min) = [4.585 × VCO2 (L/min)] – [3.226 × VO2 (L/min)]

Fat oxidation (g/min) = [1.695 × VO2 (L/min)] – [1.701 × VCO2 (L/min)]

During the initial 90-min steady-state-cycling segment, the power output was controlled by the manually set, electronically braked cycle ergometer. During the time-trial segment the ergometer was switched from manual to linear mode, in which the participant controlled power output by varying his pedal cadence. The linear mode was set to elicit a power output of ~70% VO2max at 90 rpm. Participants volitionally worked at a higher power output by increasing their pedal cadence. During all time trials, the participants were aware of their rpm, power output (W), and total work completed (a running percentage of completed kJ) but were blinded to the elapsed time. The participants competed for monetary prizes awarded to those with the lowest average total performance time in all three time trials, which encouraged maximal effort in all three experimental trials.

Experimental Protocol

The three experimental trials (taurine, control, and placebo) outlined in the preexperimental section were conducted in a randomized, counterbalanced, crossover, blinded design. Each participant reported to the laboratory between 8 and 9 a.m., after an overnight fast of ~12 hr. Laboratory temperatures were 22.3 ± 0.3, 22.5 ± 0.4, and 22.4 ± 0.4 °C for taurine, control and placebo respectively. Participants were instructed to eat as they would normally 24 hr before a cycling competition and to maintain a 24-hr dietary recall record before the practice trial. From this information, identical diets were then prepared, measured, and delivered to each participant for the 24 hr before each experimental trial. This resulted in a relative dietary macronutrient breakdown of 62% CHO, 24% fat, and 14% protein, which was exactly replicated on subsequent trials. During each trial, 1 hr before the commencement of cycling, participants consumed 500 ml of a Crystal Light (Kraft Foods) drink with either 1.66 g...
of taurine (Taurine Free-Form Amino Acid, NOW Foods, Bloomingdale, IL) or nothing added (control, placebo). Crystal Light is a low-calorie (5 kcal/224 ml) artificial-sweetener-based (aspartame) drink, which was prepared to mask any potential flavor that taurine might have provided. The taurine and control trials were administered in a double-blind fashion. The full description of the cycling portion of the trial is outlined in the preceding section. To examine a potential placebo effect in a third trial (placebo), the participants were told they had received taurine in their drink, but in reality they had not, and their drinks were identical to control. In the taurine trial, participants actually received two individual 1-g taurine capsules, which were dissolved directly into the Crystal Light. Subsequent analysis of the taurine content in the capsules by the University of Guelph Laboratory Services revealed the presence of only 0.83 g of taurine per capsule. All drinks were prepared ~12 hr in advance by the same independent individual who was not present at the experimental trials. Posttrial questionnaires showed that the Crystal Light flavoring did serve to mask the taurine and participants were adequately blinded. Participants were asked to maintain their normal training patterns but to refrain from intense exercise and caffeine and alcohol consumption 48 hr before each trial.

Statistics

All data are presented as $M \pm SE$. Dependent variables were analyzed using two-way repeated-measures ANOVA (treatment vs. time). A Student Newman-Keuls post hoc test was used to test for significance when a significant $F$ ratio was obtained. Average performance time, time-trial power outputs, and total CHO and fat oxidation over the entire 90 min of cycling were analyzed via a one-way repeated-measure ANOVA. Average power output between trials at a given time point was analyzed using a paired $t$ test. Statistical significance was accepted at $p < .05$.

Results

Performance Time Trial

There was no difference in the average time to complete 5 kJ of work/kg BM as quickly as possible between any of the three time trials (taurine 1,550 ± 101, control 1,472 ± 78, placebo 1,478 ± 81 s; Figure 1). The coefficients of variation (CV) between all 3 time trials were 4.1% ± 1.6% and 2.4% ± 0.6% between the nontaurine control and placebo trials, respectively. However, there was no clear pattern in performance times; 2 participants performed slightly faster on taurine, 5 participants performed fastest during the control trial, and 4 participants performed their best during the placebo trial. The average power output throughout the entire time trial for taurine supplementation (247.7 ± 7.4 W) was not significantly different from the control (258.4 ± 7.1 W) or placebo (255.3 ± 7.4 W) trial.

Cardiorespiratory Responses

During the 90-min pre-time-trial ride, average HR (taurine 148 ± 3, control 148 ± 3, placebo 148 ± 3 beats/min) and RPE (taurine 12.2 ± 0.5, control 11.7 ± 0.6, placebo 11.9 ± 0.5) were not different between any of the treatments (Table 1). Time-trial values for both HR (taurine 178 ± 3, control 180 ± 3, placebo 180 ± 2 beats/min) and RPE (taurine 17.8 ± 0.3, control 17.2 ± 0.4, placebo 17.4

Figure 1 — Effect of taurine, control, or placebo ingestion on time to complete a 5-kJ/kg body mass time trial after 90 min of steady-state cycling at ~65% $V_{O_2max}$. Values are $M \pm SEM$, $N = 11$. No significant difference between trials was observed.
± 0.4) were not different between trials but significantly greater than the 90-min values in all trials.

**CHO and Fat Oxidation**

Exercise VO$_2$ and VCO$_2$ were not significantly affected by taurine and increased ($p < .05$) over time in all three trials (Table 1). Because there were no trial differences in RER, both average CHO (taurine 2.73 ± 0.21, control 2.88 ± 0.19, placebo 2.89 ± 0.20 g/min) and fat (taurine 0.45 ± 0.05, control 0.39 ± 0.04, placebo 0.39 ± 0.05 g/min) oxidation rates were unaffected by taurine supplementation (Table 1). However, when examining total fat and CHO oxidation over 90 min of steady-state cycling, there was significantly ($p = .038$; main trial effect) more fat oxidized during the taurine trial than with both control and placebo (Figure 2).

The nearly identical fat and CHO oxidation rates of the control and placebo trials were collapsed and compared with taurine. A small (~5 g fat, ~20 kcal, ~84 kJ) but significant 15.7% ± 8.1% average increase in total fat oxidation for taurine was found over the 90-min exercise period compared with the collapsed data (average of control and placebo). Individually, 7 participants showed an increase in fat oxidation with taurine supplementation compared with the collapsed data of control and placebo trials, while 3 participants showed no change and 1 participant had a small decrease.

Fat oxidation contributed 27% in the taurine trial and 23% in the collapsed data (average of control and placebo) throughout the 90-min exercise period. When examining the time course of increased fat oxidation in the taurine trial during the 90-min exercise period, the initial 30 min of exercise had the greatest relative increase in fat oxidation compared with the collapsed control and placebo data (+25.9% ± 9.2%; Table 1). However, this relative increase in fat oxidation steadily declined throughout the rest of the 90-min exercise period (30–60 min +12.0% ± 7.4% taurine vs. collapsed, 60–90 min +3.6% ± 1.7% taurine vs. collapsed; Table 1).

**Discussion**

This study examined the whole-body metabolic and performance effects of acutely consuming solely taurine before a steady-state cycling bout and subsequent cycling time-trial performance (time to complete 5 kJ of work/kg BM; ~25 min). This is the first study to report that 1.66 g of taurine taken 1 hr before 90 min of cycling at ~65 VO$_{2\max}$ resulted in a small, but significant, 16% increase in total whole-body fat oxidation in endurance-trained men. However, this did not result in any change in HR or RPE or any difference in final time-trial performance between taurine versus control or from the verbal suggestion that taurine had been ingested in a placebo trial.

**Taurine and Cycling Performance**

Taurine is currently incorporated as a functional ingredient in numerous “energy” drinks, with many companies claiming ergogenic effects. The amount of taurine in most commercialized products ranges from 1,000 to 2,000 mg...
### Table 1  Respiratory Responses During Steady-State Cycling and a Time Trial (TT) After Ingestion of Taurine (T), Control (CON), or Placebo (PL), $M \pm SD, N = 11$

<table>
<thead>
<tr>
<th>Measure</th>
<th>Trial</th>
<th>13–15 min</th>
<th>27–30 min</th>
<th>43–45 min</th>
<th>57–60 min</th>
<th>73–75 min</th>
<th>87–90 min</th>
<th>100 min</th>
<th>110 min</th>
<th>End of TT</th>
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<tbody>
<tr>
<td>HR</td>
<td>T</td>
<td>143 ± 2</td>
<td>148 ± 3</td>
<td>148 ± 3*</td>
<td>150 ± 3*</td>
<td>150 ± 3*</td>
<td>151 ± 3*</td>
<td>171 ± 2†</td>
<td>176 ± 3†</td>
<td>187 ± 3†‡</td>
</tr>
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<td>142 ± 2</td>
<td>147 ± 3</td>
<td>148 ± 3*</td>
<td>151 ± 3*</td>
<td>150 ± 3*</td>
<td>150 ± 3*</td>
<td>174 ± 3†</td>
<td>177 ± 3†</td>
<td>188 ± 3†‡</td>
</tr>
<tr>
<td></td>
<td>PL</td>
<td>143 ± 3</td>
<td>146 ± 3</td>
<td>148 ± 3*</td>
<td>148 ± 3*</td>
<td>150 ± 3*</td>
<td>150 ± 3*</td>
<td>175 ± 2†</td>
<td>176 ± 3†</td>
<td>188 ± 2†‡</td>
</tr>
<tr>
<td></td>
<td>T</td>
<td>11.0 ± 0.6</td>
<td>11.9 ± 0.4*</td>
<td>12.2 ± 0.4*</td>
<td>12.3 ± 0.4*</td>
<td>12.7 ± 0.4*</td>
<td>13.2 ± 0.6*</td>
<td>16.5 ± 0.5†‡</td>
<td>18.0 ± 0.4†‡</td>
<td>19.0 ± 0.0†‡</td>
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<td>RPE</td>
<td>CON</td>
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<td>11.6 ± 0.4*</td>
<td>12.0 ± 0.4*</td>
<td>12.2 ± 0.4*</td>
<td>12.4 ± 0.4*</td>
<td>15.7 ± 0.6†‡</td>
<td>17.3 ± 0.5†‡</td>
<td>18.6 ± 0.2†‡</td>
</tr>
<tr>
<td></td>
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<td>10.5 ± 0.6</td>
<td>11.2 ± 0.6*</td>
<td>11.5 ± 0.5*</td>
<td>12.2 ± 0.4*</td>
<td>12.9 ± 0.6*</td>
<td>13.1 ± 0.6*</td>
<td>16.1 ± 0.5†‡</td>
<td>17.2 ± 0.4†‡</td>
<td>18.9 ± 0.1†‡</td>
</tr>
<tr>
<td>VO₂</td>
<td>T</td>
<td>2.85 ± 0.12</td>
<td>2.93 ± 0.13*</td>
<td>2.94 ± 0.12*</td>
<td>2.94 ± 0.14*</td>
<td>2.95 ± 0.40*</td>
<td>2.98 ± 0.41*</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>2.87 ± 0.10</td>
<td>2.90 ± 0.11*</td>
<td>2.93 ± 0.12*</td>
<td>2.93 ± 0.10*</td>
<td>2.97 ± 0.12*</td>
<td>2.97 ± 0.11*</td>
<td>ND</td>
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<tr>
<td></td>
<td>PL</td>
<td>2.87 ± 0.11</td>
<td>2.91 ± 0.11*</td>
<td>2.97 ± 0.11*</td>
<td>2.96 ± 0.11*</td>
<td>2.98 ± 0.11*</td>
<td>2.97 ± 0.12*</td>
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<tr>
<td>VCO₂</td>
<td>T</td>
<td>2.60 ± 0.12</td>
<td>2.65 ± 0.13</td>
<td>2.67 ± 0.12*</td>
<td>2.66 ± 0.13*</td>
<td>2.67 ± 0.12*</td>
<td>2.69 ± 0.12*</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>CON</td>
<td>2.67 ± 0.11</td>
<td>2.67 ± 0.11</td>
<td>2.69 ± 0.12*</td>
<td>2.68 ± 0.11*</td>
<td>2.70 ± 0.12*</td>
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<td>ND</td>
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<td>ND</td>
</tr>
<tr>
<td></td>
<td>PL</td>
<td>2.61 ± 0.11</td>
<td>2.63 ± 0.12</td>
<td>2.67 ± 0.12*</td>
<td>2.66 ± 0.11*</td>
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<tr>
<td>RER</td>
<td>T</td>
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<td>0.90 ± 0.01</td>
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<tr>
<td></td>
<td>CON</td>
<td>0.93 ± 0.01</td>
<td>0.92 ± 0.01</td>
<td>0.92 ± 0.01</td>
<td>0.91 ± 0.01</td>
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<tr>
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<td>0.93 ± 0.01</td>
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<tr>
<td>CHO</td>
<td>T</td>
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<td>2.72 ± 0.23</td>
<td>2.75 ± 0.22</td>
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<tr>
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<td>3.00 ± 0.20</td>
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<td>2.84 ± 0.18</td>
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<tr>
<td></td>
<td>PL</td>
<td>2.95 ± 0.21</td>
<td>2.93 ± 0.22</td>
<td>2.90 ± 0.22</td>
<td>2.89 ± 0.19</td>
<td>2.87 ± 0.20</td>
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<tr>
<td>FAT</td>
<td>T</td>
<td>0.40 ± 0.06</td>
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<td>0.45 ± 0.06*</td>
<td>0.46 ± 0.05*</td>
<td>0.45 ± 0.05*</td>
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<tr>
<td></td>
<td>CON</td>
<td>0.31 ± 0.04</td>
<td>0.36 ± 0.05*</td>
<td>0.39 ± 0.04*</td>
<td>0.41 ± 0.04*</td>
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<tr>
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<td>0.34 ± 0.06</td>
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</tr>
</tbody>
</table>

**Note.** HR = heart rate, beats/min; RPE = rating of perceived exertion; VO₂ = oxygen uptake, L/min; ND = no determination; VCO₂ = carbon dioxide production, L/min; CHO = carbohydrate oxidation, g/min; FAT = fat oxidation, g/min.

*Significantly different than the 13- to 15-min measurement ($p < .05$). †Significantly different than all of the 90-min steady-state measurements ($p < .05$). ‡Significantly different than previous time point ($p < .05$).
per drink. Therefore the primary aim of the current study was to examine the performance effects of ingesting 1.66 g of taurine 1 hr before 90 min of submaximal steady-state cycling followed by an ~25-min time trial. We chose this dosing protocol because plasma taurine kinetics have been shown to peak at 90–120 min (Galloway et al., 2008), coinciding with the commencement and duration of the time trial. In support of our first performance hypothesis, we found no effect of taurine ingestion on endurance time-trial performance compared with a control trial.

Several previous studies found positive physical and cognitive performance effects of energy drinks that contain taurine, including increased upper body muscle endurance (Forbes et al., 2007), anaerobic performance (Alford et al., 2001), endurance-time-to-exhaustion performance (Alford et al., 2001; Geiss et al., 1994), \( \text{VO}_{2\text{max}} \) (Jester et al., 1997), and several different cognitive-based measurements (Barthel et al., 2001; Seidl et al., 2000). However, all those studies examined the effects of taurine supplements in combination with many additional ingredients (e.g., CHO, caffeine) or did not use an appropriate isocaloric or iso-CHO placebo control beverage. Therefore, it is impossible to definitively ascertain whether any of the reported performance effects were caused exclusively by the taurine, per se, or by a combination of ingredients or the fact that some of the placebo beverages were not matched in calories to the taurine drink. In summary, the current investigation is the first to examine exclusively the acute effects of taurine on subsequent time-trial performance (not time to exhaustion), and no ergogenic effects were found. These results may not be surprising because our recent work demonstrated that chronic supplementation of taurine for 1 week did not result in an increase in skeletal-muscle taurine content (Galloway et al., 2008). This implies that any potential ergogenic effect of taurine could only occur through interaction with receptors on the muscle membrane or by affecting other organs (i.e., liver, adipose tissue) during exercise, and not within the muscle itself.

Several rodent studies using prolonged (>2 weeks) taurine supplementation found increases in endurance performance (Imagawa et al., 2009; Miyazaki et al., 2004; Yatabe, Miyakawa, Miyazaki, Matsuzaki, & Ochiai, 2003) or isometric skeletal-muscle twitch force (Goodman et al., 2009). However, there appears to be a species difference in the skeletal-muscle uptake of taurine during prolonged supplementation. Rodents can increase the muscle content of taurine by ~40% over a 2-week period (Goodman et al., 2009; Yatabe et al., 2003), whereas human muscle taurine content appears to be tightly regulated, because 7 days of supplementation did not result in any muscle taurine or metabolism changes in humans (Galloway et al., 2008).

**Practice Trials, Time-Trial Pacing, and Placebo Effect**

To ensure optimal conditions to titrate out any potential performance differences between treatments, double-blinded experimental trials need to be conducted in which participants receive no time, distance, or wattage (pace) feedback (Jeukendrup & Currell, 2005). Accordingly, whenever a time trial or competition is longer than ~60 s, whether pacing information is provided or not, athletes undertake some sort of pacing strategy to optimize their chance of success (Foster, Schrager, Snyder, & Thompson, 1994). Previous investigations have shown that serial time trials without distance or time feedback result in progressively improved performances, being nearly optimal by the third blinded time trial (Mauger, Jones, & Williams, 2009). It appears that prior experience of an unknown distance results in the creation of an internal “relative” distance monitor that helps establish an optimal pacing strategy. Thus, in the current study, two practice time trials were implemented to ensure that participants had learned the best possible pacing strategy, or the optimal management of power output, before the three experimental trials. The general pattern of wattage output was similar between trials and to previous studies reporting wattage outputs throughout cycling time trials (Chambers, Bridge, & Jones, 2009; Foster et al., 1994). Over the initial 50–60% of the time trial, participants demonstrated a small and steady decline in power output that was recovered over the last 25% of the time trial with large increases in power output in all trials. This acceleration during the late stages of more prolonged (>20 min) races that feature minimal air resistance is common (Foster et al., 1994).

Contrary to our second hypothesis, we found no performance enhancement from the verbal suggestion that taurine was in the preexercise drink (placebo effect trial). A placebo effect is a favorable or positive outcome arising purely from the belief that one has received a beneficial treatment (Clark, Hopkins, Hawley, & Burke, 2000). In the sport nutrition performance research field there appear to be only two published studies that examined the phenomenon of the placebo effect in direct relation to performance outcomes (Beedie, Stuart, Coleman, & Foad, 2006; Clark et al., 2000). Those studies both found a small, but worthwhile, 4.3% and 3.1% improvement over 40-km and 10-km cycling time trials, respectively (Beedie et al., 2006; Clark et al., 2000), just from the belief that participants had consumed an ergogenic aid when in fact they had not. Paton and Hopkins (2006) calculated that the smallest practically beneficial improvement in power output for an elite cyclist is 0.5 multiplied by the typical variation in individual performance, which is ~0.6%. This suggests that in those former studies (Beedie et al., 2006; Clark et al., 2000), the performance improvement from a perceived placebo effect alone is greater than the minimum improvement necessary for a beneficial performance outcome in elite athletes. Thus, it appears to be warranted that future performance-based studies that feature a crossover design consider adding a placebo-effect treatment arm. These designs would add clarity to some of the underlying mechanisms explaining a performance effect and ensure that a proposed performance-enhancing agent is at least as, or more, ergogenic than any placebo effect.
Increase in Total Whole-Body Fat Oxidation

To our knowledge, this is the first study to report a small (~84 kJ) but significant ~16% increase in total fat oxidation after the acute supplementation of taurine before steady-state exercise. Conversely, two previous reports found no effect of taurine on whole-body fuel metabolism measured via indirect calorimetry during exercise (Galloway et al. 2008; Jester et al., 1997). However, one of those studies had participants consume 1.6 g of taurine 2–4 hr before exercise with their last meal, which resulted in their missing the plasma taurine peak (from 1.5 to 2 hr) during 2 hr of cycling at ~60% VO2max (Galloway et al., 2008). Furthermore, in that study participants were examined postprandially and were only recreationally active, and it has been shown that skeletal and plasma amino acid responses to exercise are different between trained and untrained participants (Graham, Turcotte, Kiens, & Richter, 1995). Jester et al.’s (1997) study had participants consume a drink with CHO, taurine, and caffeine 20 min before 25 min of exercise featuring an incremental protocol to exhaustion. In that study, average RER throughout the entire protocol showed a trend for increased fat oxidation compared with an iso-CHO and -caffeine control drink (RER 0.99 ± 0.08 vs. 1.04 ± 0.08).

Our current data cannot elucidate the mechanism responsible for the increase in total whole-body fat oxidation. Nevertheless, several in vitro cell-culture studies have shown that cAMP production can be directly stimulated by taurine, through adenyl cyclase activation (Chen, Li, & Kong, 1996; Chen, Zhang, Xie, & Li, 2004; Mal’chikova & Elizarova, 1981; Mal’chikova, Speranskaia, & Elizarova, 1979; Raizada & Krishna Murti, 1973) or perhaps through an increased secretion of catecholamines (Takekura, Tanaka, Watanabe, Yoshikawa, & Ono, 1986). Activation of the adenyl cyclase/cAMP cascade, either directly or mediated via augmented catecholamines, is a primary mechanism responsible for increased lipolysis and fat oxidation during moderate-intensity exercise, either at the site of the adipocyte or via hormone-sensitive lipase activation in skeletal muscle (for a review, see Watt & Sprriet, 2004). These measures were not made in the current study because no blood or muscle samples were taken so as not to hinder the primary focus of examining endurance performance after the acute consumption of taurine. A previous study did find that a drink with CHO, taurine, and caffeine resulted in a significant ~45% increase in both plasma epinephrine and norepinephrine content compared with a drink containing identical amounts of CHO and caffeine (Jester et al., 1997). Furthermore, another study showed an ~45% nonsignificant increase in plasma free fatty acids after 45 min and at exhaustion during submaximal cycling exercise with a drink containing CHO, taurine, and caffeine compared with a drink containing the same amounts of just CHO and caffeine (Geiss et al., 1994). Certainly future studies examining the acute effects of administering solely taurine before submaximal steady-state exercise are warranted to corroborate the current study’s findings of increased fat oxidation and to illuminate the potential responsible mechanisms. The potential for taurine to affect lipolysis in adipose tissue and in skeletal muscle would be consistent with taurine only affecting muscle through receptors on the muscle membrane, given that our recent work has demonstrated that skeletal muscle does not take up extra taurine after supplementation for 1 week (Galloway et al., 2008).

In conclusion, the acute supplementation of 1.66 g of taurine 1 hr before 90 min of submaximal cycling followed by an ~25-min time trial was not ergogenic. Telling participants they were receiving taurine in their preexercise drink when they did not also was not ergogenic. Taurine also had no effect on the normal physiological responses (HR, RPE) to exercise compared with control and placebo trials. Acute taurine ingestion did produce a significant 16% increase in total whole-body fat oxidation during 90 min of submaximal exercise before the time trial. Future studies are warranted to further confirm this acute increase in fat oxidation, to determine the possible mechanisms of this shift in relative fuel utilization, and to ascertain whether this small increase in fat oxidation could lead to any performance effects during more prolonged performance situations.

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