The Effect of Acute Taurine Ingestion on Human Maximal Voluntary Muscle Contraction

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ABSTRACT

LIM, Z. X., A. SINGH, Z. Z. X. LEOW, P. G. ARTHUR, and P. A. FOURNIER. The Effect of Acute Taurine Ingestion on Human Maximal Voluntary Muscle Contraction. *Med. Sci. Sports Exerc.*, Vol. 50, No. 2, pp. 344–352, 2018. **Purpose**: This study aimed to examine the effect of taurine ingestion on maximal voluntary muscle torque and power in trained male athletes with different caffeine habits. **Methods**: Fourteen male athletes 21.8 ± 2.5 yr old were separated into caffeine and noncaffeine consumers to control for the effect of caffeine withdrawal on muscle function. On separate occasions, participants performed four isokinetic or three maximal isometric knee extensions with and without taurine (40 $mg \cdot kg^{-1}$ body mass) after a double-blind, counterbalanced design. Muscle contractile performances were compared between the first sets as well as between the sets where these variables scored best. **Results**: In response to isokinetic contraction, taurine treatment in the noncaffeine consumers resulted in a significant fall in first (−16.1%; $P = 0.013$) and best peak torque (−5.0%; $P = 0.016$) as well as in first (−17.7%; $P = 0.015$) and best power output (−8.0%; $P = 0.008$). In the caffeine consumers deprived of caffeine, taurine intake improved best power (5.2%; $P = 0.045$). With respect to the isometric variables, there was a significant decrease in the first (−5.1%; $P = 0.002$) and best peak torque (−4.3%; $P = 0.032$) in the noncaffeine group, but no effect in the group of caffeine consumers deprived of caffeine. Taurine ingestion increased blood taurine levels but had no effect on plasma amino acid levels. **Conclusions**: Taurine ingestion is detrimental to maximal voluntary muscle power and both maximal isokinetic and isometric peak torque in noncaffeine consumers, whereas taurine ingestion in caffeine-deprived caffeine consumers improves maximal voluntary muscle power but has no effect on other aspects of contractile performance. **Key Words:** ERGOGENIC AIDS, CAFFEINE WITHDRAWAL, MUSCLE POWER, TRYPTOPHAN BRANCHED-CHAIN AMINO ACID RATIOS

Over the past decade, a number of studies have reported that taurine can affect muscle function. For instance, taurine deficiency disrupts muscle excitation–contraction coupling (1) and is associated with several skeletal muscle disorders such as myotonia congenital, Duchenne muscular dystrophy, and related myopathies (2). Also, the addition of taurine to isolated muscle fibers from mice and humans augments sarcoplasmic reticulum Ca^{2+} accumulation (3,4) and release (3), and increases the magnitude of depolarization-induced force (3), thus raising the issue of whether taurine ingestion may be beneficial to exercise performance.

Several studies have examined the effect of taurine ingestion on endurance exercise performance. Compared with a drink of similar composition that contained no taurine, taurine-containing drinks have been reported in a study by Geiss and colleagues (5) to increase time to exhaustion and reduce heart rate in an endurance performance trial. However, the presence of other ingredients in their taurine containing drink, such as caffeine, sugar, and glucuronolactone, does not exclude the possibility that taurine in combination with one or more of these ingredients exerts a synergic effect. This limitation is not shared, however, by other studies that have investigated the acute and chronic effects of taurine alone on endurance performance. One of these studies showed that taurine supplementation (6 $g \cdot d^{-1}$) for 7 d significantly increased peak rate of oxygen consumption ($\dot{V}O_{2peak}$), cycling time to exhaustion, and maximal workload by 6.9%, 2.7%, and 3.8%, respectively (6). Another study showed that 2 wk of taurine supplementation (4 $g \cdot d^{-1}$) improved treadmill running time to exhaustion by 6.9 min at an intensity of 75% $\dot{V}O_{2peak}$ (7). Balshaw and colleagues (8) also reported that the acute ingestion of 1 g of taurine improved time to complete a 3-km run by an average of 1.7% in trained middle distance runners. In contrast, the acute ingestion of taurine has been reported not to enhance cycling time-trial performance in endurance-trained cyclists (9,10), as well as time to exhaustion during high-intensity running in recreationally trained males (11).

The only studies that have examined the effect of taurine on muscle power have focused on repeated sprint performance and shown that the acute ingestion of energy drinks containing taurine and other ingredients such as caffeine does not

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improve repeated sprint performance in female and male athletes (12,13). To the best of our knowledge, the direct effect of acute taurine ingestion on muscle maximal strength and power still remains to be examined in humans. This is surprising given the evidence that taurine increases the depolarization-induced force generated by isolated muscle fiber preparations (3). For this reason, the primary aim of this study was to test the hypothesis that taurine ingestion increases muscle power and both peak isometric and isokinetic torque in trained competitive athletes. Given that both taurine and caffeine stimulate Ca^{2+} release channels in skeletal muscles (3,14) and that taurine can oppose some of the caffeine withdrawal symptoms (15), the recruitment of noncaffeine consumers together with 24-h caffeine-deprived habitual caffeine consumers to test our hypothesis might affect our findings. This is because caffeine withdrawal reversal may occur where the benefits of taurine could be mainly one of restoring performance from caffeine withdrawal rather than improving performance per se, thus leading to different levels of taurine-mediated benefits between noncaffeine consumers and caffeine-deprived habitual caffeine consumers. Because of this potential confounding factor, our secondary aim was to examine the extent to which the effect of taurine on muscle power differs between noncaffeine consumers and caffeine-deprived caffeine consumers.

METHODS

Participants

Fourteen healthy trained male athletes with ages from 20 to 28 yr, with no injury, and involved in competitive sports relying heavily on power training (Australian rules football, football, rowing, sprints) were recruited for this study (Table 1). To address the possibility that caffeine withdrawal symptoms (16) before testing might interfere with our findings, participants were recruited so that half were noncaffeine consumers as defined by the absence of coffee, energy drink, or tea consumption (some caffeine ingestion, however, occurred due to the ingestion of low-caffeine–containing food) and the other half were habitual caffeine consumers ingesting more than 100 mg of caffeine a day as determined using a validated caffeine consumption questionnaire (17). These two groups were matched for age, body mass index (BMI), and aerobic fitness (Table 1). Although written informed consent was obtained from all participants, they were deceived about the real purpose of the study to minimize the possibility of a placebo effect. To this end, participants were informed that the research aimed at determining the effect of different concentrations of taurine on maximal voluntary contraction. At the end of the study, all participants were debriefed and informed about the true purpose of the study. This research project was approved by the Research Ethics Committee of The University of Western Australia.

Experimental Design

Familiarization session. Participants were required to visit our laboratory for a familiarization session. During this session, all participants were familiarized with the exercise protocol on the Biodex isokinetic dynamometer (System 3 Pro; Shirley, NY). In addition, anthropometric data and $\dot{V}O_{2peak}$ were determined. $\dot{V}O_{2peak}$ was performed on a cycle ergometer (Cyclemax 6.3; Human Movement, The University of Western Australia, Crawley, Australia) by means of a graded exercise test, which commenced at 100 W, with increment of 50 W every 3 min, until volitional exhaustion. During the test, expired respiratory gasses were collected and analyzed for O_2 and CO_2 content every 15 s.

Experimental design: overview. To examine the effect of taurine on maximal voluntary muscle power, maximal voluntary isometric contraction, and peak rate of torque development, participants attended separate testing sessions during which they ingested either taurine or a placebo. Both treatments were administered 1 to 2 wk apart after a randomized, counterbalanced, double-blind design for both the caffeine-deprived habitual caffeine consumers and noncaffeine consumer groups. One hour after placebo or taurine ingestion, both isometric and isokinetic assessments of the quadriceps muscles were performed, with the order of these assessments being identical between testing sessions.

Testing session. Before each testing session, participants were instructed to avoid alcohol, caffeine, supplementations,

TABLE 1. Descriptive characteristics before testing in caffeine-deprived caffeine consumers, noncaffeine consumers, and all participants.

	Noncaffeine Group		Caffeine Group		
	PLA	TAU	PLA	TAU	All Participants
Age, yr	21.1 ± 1.9		22.4 ± 3.1		21.8 ± 2.5
Body mass, kg	80.2 ± 8.8		77.5 ± 9.7		78.8 ± 9.0
Height, cm	182.3 ± 7.9		181.9 ± 7.0		182.1 ± 7.2
BMI, $kg \cdot cm^{-2}$	24.8 ± 1.6		23.4 ± 2.5		23.8 ± 2.5
Sum of 6 skinfolds, mm	47.1 ± 14.6		38.9 ± 4.3		43.0 ± 11.2
Sum of 8 skinfolds, mm	60.2 ± 18.8		51.5 ± 6.2		55.8 ± 14.2
$\dot{V}O_2$ peak, $L \cdot min^{-1} \cdot kg^{-1}$	53.5 ± 6.2		57.2 ± 7.1		55.3 ± 6.7
Caffeine intake, $mg \cdot d^{-1}$	26.7 ± 32.1		327.4 ± 146.6[a]		177.1 ± 186.4
Sleep quality (out of 5)	3.9 ± 0.7	4.0 ± 0.6	3.7 ± 0.5	3.9 ± 0.7	—
Sleep duration, h	7.1 ± 0.8	7.4 ± 1.3	7.1 ± 0.7	7.5 ± 1.2	—
Energy, kJ	8738 ± 2357	9847 ± 2593	10,676 ± 1490	11,696 ± 3246	—
kJ from protein, %	23.8 ± 5.0	22.5 ± 3.1	24.4 ± 11.5	25.4 ± 7.7	—
kJ from fat, %	31.9 ± 11.3	30.8 ± 10.2	35.6 ± 6.1	35.7 ± 8.2	—
kJ from carbohydrate, %	41.9 ± 10.9	43.8 ± 9.7	37.1 ± 11.8	36.5 ± 11.8	—

Values are expressed mean ± SD.
[a]A significant difference to the noncaffeine group ($P < 0.001$).

APPLIED SCIENCES

and medications for 24 h and to minimize their physical activity level. They were also instructed to arrive in the laboratory early in the morning (between 6:00 and 7.30 AM) after an overnight fast without taking any food or nutritional supplements on the day of testing, but with water intake being allowed *ad libitum*. Participants were also asked to record their food intake in a diary the day before testing and to replicate the same diet before subsequent sessions (Table 1). Also, they were required to keep record of their sleep duration and to rate the quality of their sleep out of five (1 being poor and 5 being excellent) on the night before testing (Table 1). Finally, to ensure that participants kept their physical activity level to a minimum before testing, they recorded their physical activity level for 24 h before testing using a physical activity diary. All testing sessions were carried out in the morning at the same time and on the same days of the week. Finally, participants were advised to wear the same sport attire for all testing sessions.

On each testing day, participants attended the laboratory, emptied their bladder, and rested on a chair for 15 min, after which heart rate was recorded using a heart rate monitor (Polar FS1; Polar Electro, Inc, Lake Success, NY). After this, each participant ingested either cellulose-filled placebo capsules (10 $mg \cdot kg^{-1}$ body mass; PLA) or capsules providing 40 $mg \cdot kg^{-1}$ body mass of taurine with 10 $mg \cdot kg^{-1}$ body mass of cellulose (TAU) and drank 250 mL of water. This dose of taurine was chosen because it amounts to ~3 g of taurine for a 75-kg participant, which is considered middle range relative to past-study taurine doses (1–6 $g \cdot d^{-1}$ of taurine [2]). Participants then rested for 1 h before testing because this has been reported to be the time required for plasma taurine to reach peak concentration after ingestion (18). The reactions of each participant to taurine ingestion were also monitored using the questionnaire of Ferreira and colleagues (19), which was administered before and 1 h after taurine or placebo administration. After the completion of the last testing session, participants were asked if they noticed any differences in taste or appearance between capsules ingested between sessions by rating the difference on a scale from 0 (no difference) to 5 (very noticeable difference).

Isokinetic and isometric muscle contraction protocol. Before assessing muscle contractile performance, participants sat on a Biodex dynamometer (System 3 Pro), with the axis of rotation of the dynamometer aligned to the lateral epicondyle of the right femur. The leg was secured to the lever arm of the dynamometer 3–5 cm above the lateral malleoli using a noncompliant strap. To minimize any extraneous movement, noncompliant chest, pelvic and thigh straps were used to secure participants to the dynamometer's seat. Dynamometer settings were recorded during the familiarization and replicated during subsequent trials. The participants were then subjected to both an isokinetic and isometric contraction protocol, with their arms crossed on their chests while performing these protocols.

Before isokinetic testing, hip joint was fixed at 95° and the range of motion for the knee joint was set from 0° to 90°. Then, participants performed four maximal knee extensions at an angular velocity settings of 60°·s upon hearing a signal emitted by the ergometer, with 30 s of passive recovery between each extension. Peak torque, maximum rate of torque development, and power for both treatments were compared either between the first contractions or between the contractions where these variables scored at their highest.

Before assessing muscle isometric performance, the knee and hip joints of the right leg were fixed at 70° and 95°, respectively. All participants were instructed to perform a maximal voluntary contraction protocol consisting of three 4-s maximal isometric knee extensions during which torque was recorded. Each effort was separated by 2 min of passive recovery. The highest torque achieved in response to the isometric contractions was taken as peak torque. The rate of torque development was measured over the first 200 ms of contraction. Reaction time was measured as the time taken from the start of signal emitted by the ergometer to the activation of the leg extension. Peak torque, rate of torque development, and reaction time for both treatments were compared between the first isometric contractions and between the isometric contractions where these variables scored at their highest.

Analyses of plasma taurine and amino acids levels. Before and after taurine or placebo ingestion, blood from fingertips was collected into blood collection tubes (K_2-EDTA Microtainer Tubes with BD Microgard Closure; Beckton Dickinson Oy, Vantaa, Finland) and part of it was centrifuged at 2550*g* for 20 min to separate the taurine-rich platelets from plasma (20). Then, the plasma was immediately frozen in liquid nitrogen and stored at −80°C until analyzed. Before amino acid analyses, plasma samples were deproteinized with 5-sulfosalicylic acid combined with Norvaline as the internal standard (21). Then, the levels of taurine and free amino acids in plasma were measured by high-pressure liquid chromatography using for precolumn derivatization *o*-phthaldialdehyde (for the primary amino acids) and fluorenylmethyl chloroformate (for the secondary amino acids [22,23]). The tryptophan/branched-chain amino acid (BCAA; sum of isoleucine, leucine, and valine) ratios and tryptophan/large neutral amino acid (LNAA; sum of BCAA, phenylalanine, tyrosine) ratios were tabulated before analysis.

Caffeine withdrawal symptoms measurements. The severity of caffeine withdrawal symptoms was assessed before familiarization session (baseline), before PLA (pre-PLA) or TAU (pre-TAU) treatment and after PLA (post-PLA) or TAU (post-TAU) treatment using the validated 23-item Caffeine Withdrawal Symptoms Measurements Questionnaire, with all participants asked to rate each item on a 5-point scale from 0 (not at all) to 4 (extremely) (24). Eight of the positively worded items (e.g., energetic) were reverse scored before analyses.

Statistical Analyses

Data were analyzed for differences between TAU and PLA conditions using paired-samples *t*-tests, with significance set at $P < 0.05$. When variables were measured at more than two-time

points, two-way repeated-measures ANOVA followed by Fisher least significant difference *post hoc* test, with significance set at $P < 0.05$, was used to investigate the effect of both taurine and time. When variables were compared at more than two-time points and between noncaffeine and caffeine groups, two-way mixed-model ANOVA was adopted followed by Fisher least significant difference *post hoc* test, with significance set at $P < 0.05$.

RESULTS

Anthropometric and Lifestyle Variables

There were no significant difference in age ($P = 0.40$), weight ($P = 0.60$), height ($P = 0.92$), BMI ($P = 0.57$), sum of 6 ($P = 0.18$) and 8 ($P = 0.26$) skinfolds, and $\dot{V}O_{2peak}$ ($P = 0.32$) between the participants in the noncaffeine consumer and caffeine consumer groups, whereas a significant difference on daily caffeine intake was observed between the two groups ($P < 0.001$; Table 1). There were no significant differences between sleep quality ($P = 0.43$) and duration ($P = 0.30$) between PLA and TAU treatment days (Table 1). There were no significant differences between total energy intake, weight and kJ from proteins, fats, and carbohydrate ($P > 0.05$) between PLA and TAU treatment days (Table 1) in both noncaffeine consumer and caffeine consumer groups.

Effect of Taurine Ingestion on Maximal Voluntary Contraction

Isokinetic contraction. The effects of taurine ingestion on maximal voluntary peak torque, power, and rate of torque development were assessed by comparing either the first or the highest values achieved for each variable in response to PLA and TAU conditions. In the noncaffeine consumer group, taurine ingestion resulted in a significant fall in first (−16.1%; $P = 0.013$; effect size (ES) = −0.70; Fig. 1A) and best peak torque (−5.0%; $P = 0.016$; ES = −0.32; Fig. 1D), best rate of torque development (−4.6%; $P = 0.034$; ES = −0.25; Fig. 1E), and first (−17.7%; $P = 0.015$; ES = −0.86; Fig. 1C) and best power (−8.0%; $P = 0.008$; ES = −0.56; Fig. 1F). No significant difference was observed for the first rate of torque development ($P = 0.055$; ES = −0.65; Fig. 1B). In the caffeine-deprived caffeine consumer group, taurine ingestion resulted in significant improvements in best power (5.2%; $P = 0.045$; ES = 0.22; Fig. 1F). However, there was no significant changes in first ($P = 0.206$; ES = 0.24) and best peak torque ($P = 0.238$; ES = 0.09), first ($P = 0.399$; ES = 0.083) and best rate of torque development ($P = 0.454$; ES = −0.02), and first power ($P = 0.366$; ES = −0.16) between treatments (Figs. 1A–E). When all caffeine and noncaffeine consumer participants were grouped, only first power significantly deteriorated in response to taurine (−11.7%; $P = 0.043$; ES = −0.49; Fig. 1C), with no effect on best power ($P = 0.201$; ES = −0.11), first ($P = 0.129$; ES = −0.26) and

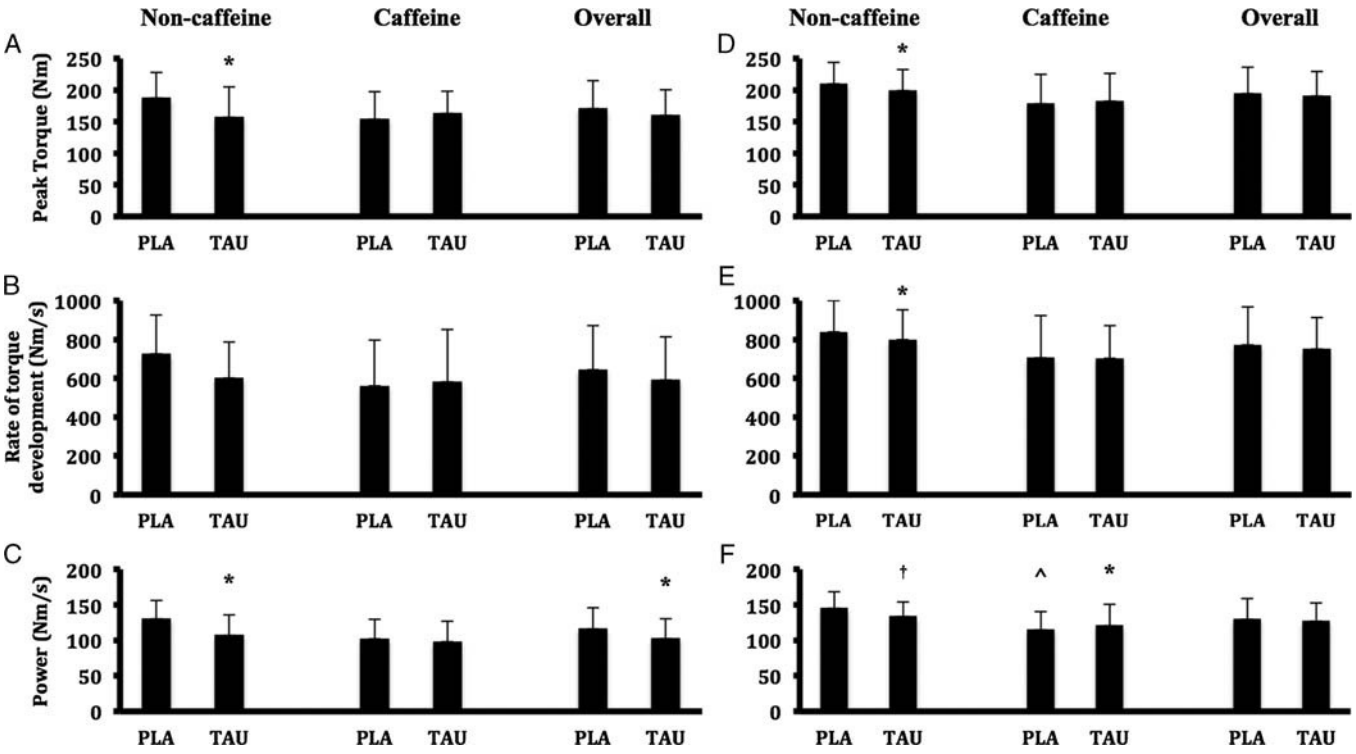

FIGURE 1—Effect of PLA and TAU treatments on peak torque (A), maximal rate of torque development (B), and power for the first maximal isokinetic voluntary contraction (C) as well as on peak torque (D), maximum rate of torque development (E), and power for the best maximal isokinetic voluntary contraction (F) in the noncaffeine group, caffeine group, and all participants. A significant difference to PLA (*$P < 0.05$ and †$P < 0.001$, respectively) and to noncaffeine (^$P < 0.05$). Values are expressed as mean ± SD.

best peak torque ($P = 0.193$; ES = −0.08), and first ($P = 0.175$; ES = −0.23) and best rate of torque development ($P = 0.113$; ES = −0.12) in response to taurine (Fig. 1A, B, D–F).

Isometric contraction. The effects of TAU ingestion on maximal peak torque, rate of torque development, and reaction time were assessed by comparing either the first or highest values achieved for each variable in response to PLA and TAU conditions. In the noncaffeine consumer group, there was a significant decrease in the first (−5.1%; $P = 0.002$; ES = −0.25; Fig. 2A) and best peak torque (−4.3%; $P = 0.032$; ES = −0.20; Fig. 2D), but no significant differences in first ($P = 0.304$; ES = −0.16; Fig. 2B) and best rate of torque development ($P = 0.077$; ES = −0.16; Fig. 2E) and first ($P = 0.170$; ES = 0.58; Fig. 2C) and best reaction time ($P = 0.203$; ES = 0.33; Fig. 2F). In the caffeine-deprived caffeine consumer group, there was no significant difference for first ($P = 0.302$; ES = −0.21) and best peak torque ($P = 0.177$; ES = −0.16), first ($P = 0.332$; ES = −0.20) and best rate of torque development ($P = 0.286$; ES = −0.15), and first ($P = 0.170$; ES = −0.38) and best reaction time ($P = 0.071$; ES = −0.57) between treatments (Fig. 2A–F). When both caffeine and noncaffeine groups were combined, there was a significant fall in best peak torque (−3.6%; $P = 0.024$; ES = −0.19; Fig. 2D) in response to taurine, but no significant difference with the other isometric variables, including first peak torque ($P = 0.086$; ES = −0.24), first ($P = 0.239$; ES = −0.18) and best rate of torque development ($P = 0.081$; ES = −0.15), and first ($P = 0.359$; ES = 0.13) and best reaction time ($P = 0.196$; ES = −0.24) in response to taurine (Fig. 2A–C, E, F).

There were no significant differences for all the isokinetic and isometric variables between noncaffeine and caffeine groups for the PLA condition ($P > 0.05$), except for isokinetic best power ($P = 0.034$; Fig. 1F). Test–retest reliability performed between the familiarization and control sessions resulted in a coefficient of variation of 2.5%, 3.1%, and 3.1% for peak power, peak isokinetic torque, and peak isometric torque, respectively.

Level of Adverse Reaction to Taurine Ingestion

No participants exhibited any changes in adverse reaction variables with TAU and PLA treatment ($P > 0.05$), except for reduced sleepiness in PLA treatment ($P = 0.040$; Table 2), and no participants reported any noticeable difference in the appearance or taste between PLA and TAU conditions.

Effect of Taurine Ingestion on Blood Variables

Plasma taurine levels were higher 1 h after ingesting taurine than placebo ($P = 0.004$; Table 3). However, there were no significant differences in the concentrations of all the other amino acids tested here between PLA and TAU treatments ($P > 0.05$). No significant differences were observed for tryptophan/BCAA and tryptophan/LNAA ratios ($P > 0.05$; Table 3).

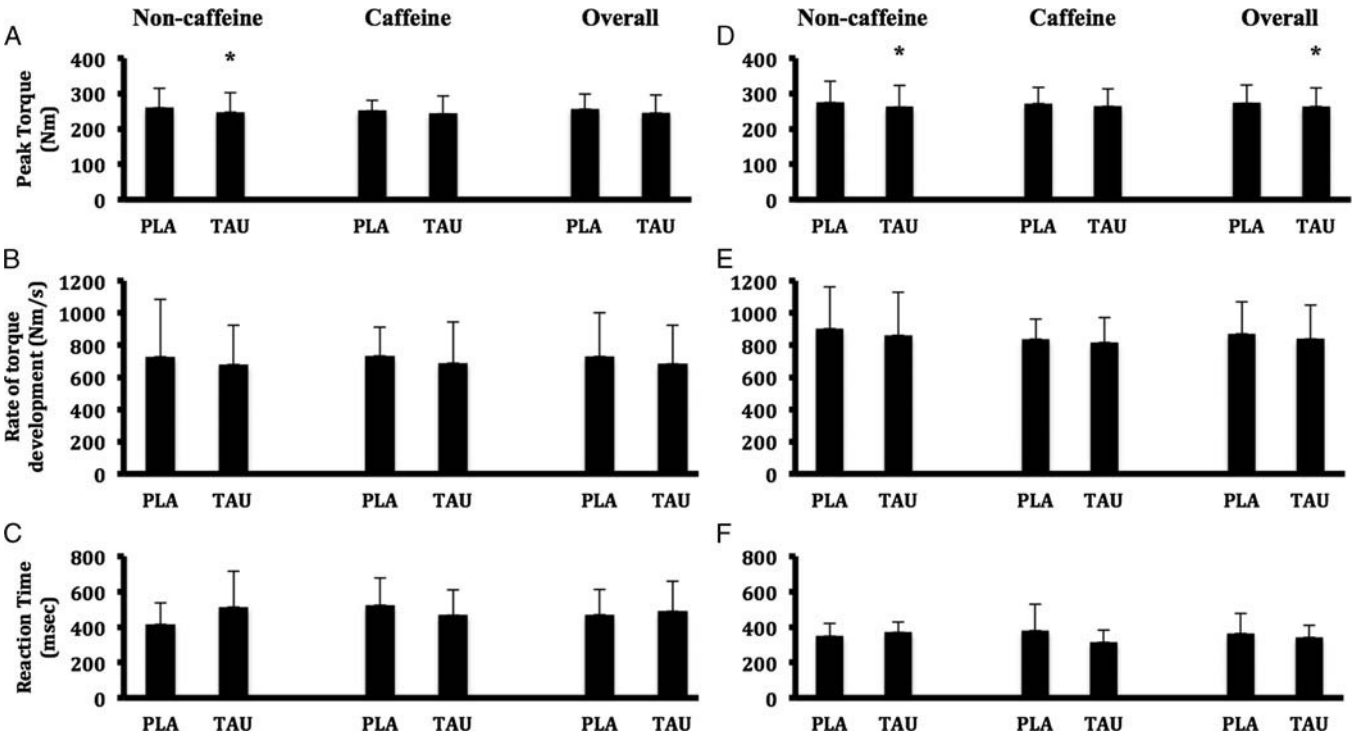

FIGURE 2—Effect of PLA and TAU treatments on peak torque (A), maximum rate of torque development (B), and reaction time for the first maximal isometric voluntary contraction (C) as well as on peak torque (D), maximum rate of torque development (E), and reaction time for the best maximal isometric voluntary contraction (F) in the noncaffeine group, caffeine group, and all participants. *A significant difference to PLA ($P < 0.05$). Values are expressed as mean ± SD.

TABLE 2. Adverse reaction to capsules ingestion.

	PLA			TAU		
Reaction	Before	After	Change	Before	After	Change
Upset stomach	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0
Nausea	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0
Stomach or intestinal gas	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0	1.0 ± 0.0	1.1 ± 0.3	−0.1 ± 0.3
Sleepiness	1.8 ± 0.7	1.4 ± 0.7[a]	0.4 ± 0.6	1.6 ± 0.7	1.1 ± 0.4	0.4 ± 0.5
Metallic taste	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0
Light headedness	1.1 ± 0.3	1.1 ± 0.3	0.0 ± 0.0	1.1 ± 0.4	1.1 ± 0.4	0.0 ± 0.0
Redness of the eye, face, or hand	1.2 ± 0.4	1.1 ± 0.4	0.1 ± 0.3	1.2 ± 0.6	1.0 ± 0.0	0.2 ± 0.6
Cough	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0	1.2 ± 0.6	1.1 ± 0.4	0.1 ± 0.3
Others	1.1 ± 0.3	1.1 ± 0.4	−0.1 ± 0.3	1.0 ± 0.0	1.0 ± 0.0	0.0 ± 0.0

Values are expressed mean ± SD.
[a]Significantly different from before ($P < 0.05$).

Effect of Taurine Ingestion on Caffeine Withdrawal Symptoms

In the caffeine consumer group, drowsiness/fatigue was significantly increased in the pre-TAU treatment compared with baseline, and decreased alertness was significantly more pronounced in pre-PLA treatment compared with baseline, but pre-TAU drowsiness/fatigue returned to baseline after taurine ingestion (Table 4). There were no significant changes in the other variables (Table 4). In the noncaffeine group, taurine ingestion had no significant effect on any of the withdrawal symptoms except improving flu-like symptoms (Table 4).

DISCUSSION

Because taurine increases the force generated by isolated rat fast twitch muscle fiber preparations (3), we examined whether acute taurine ingestion could enhance maximal voluntary power and isometric/isokinetic peak torque in healthy trained individuals, an issue that has never been examined before. Against our expectation, taurine ingestion in noncaffeine consumers resulted in a fall rather than an increase in isokinetic power (8.0%), isokinetic peak torque (5.1%), and isometric peak torque (4.3%). Another unexpected finding was the 5.2% improvement in isokinetic power after taurine ingestion in the caffeine-deprived habitual caffeine consumers, whereas the other isokinetic and isometric variables were unaffected. These findings indicate that the effect of taurine ingestion is affected by one's caffeine consumption habits.

Our finding that the caffeine habits of our participants affect markedly the pattern of responses of their isokinetic power, peak torque, and isometric peak torque to taurine ingestion was unexpected. As in most studies examining the role of ergogenic aids on exercise performance, both noncaffeine consumers and 24-h caffeine-deprived habitual caffeine consumers were recruited to our study. However, given the overlap in caffeine and taurine effects on sarcoplasmic calcium release (3,14), we designed this study to examine the extent to which taurine effect on muscle contractile performance differs between caffeine-deprived caffeine consumers and noncaffeine consumers, expecting little or no difference between these groups. Against expectations, the effect of taurine on muscle contractile performance was

TABLE 3. Effect of taurine and placebo ingestion on plasma amino acid concentrations, BCAA, LNAA, Tryp/BCAA, and Tryp/LNAA ratio 1 h after ingestion.

	Noncaffeine		Caffeine		Overall	
	PLA	TAU	PLA	TAU	PLA	TAU
Alanine, $\mu mol \cdot L^{-1}$	316.2 ± 56.5	316.1 ± 78.8	308.8 ± 82.9	336.1 ± 162.2	312.5 ± 67.0	326.1 ± 120.7
Arginine, $\mu mol \cdot L^{-1}$	97.7 ± 26.2	96.4 ± 25.2	89.4 ± 12.3	96.9 ± 22.7	93.5 ± 19.8	96.6 ± 22.6
Aspartic acid, $\mu mol \cdot L^{-1}$	13.2 ± 8.7	14.4 ± 12.3	6.9 ± 9.8	14.7 ± 9.0	10.0 ± 9.4	14.6 ± 10.2
Cystine, $\mu mol \cdot L^{-1}$	29.8 ± 17.1	30.3 ± 17.8	6.9 ± 15.4	5.9 ± 13.2	18.3 ± 19.5	18.1 ± 19.6
Glutamic acid, $\mu mol \cdot L^{-1}$	76.2 ± 8.8	87.1 ± 13.9	86 ± 10.2	85.0 ± 18.7	81.1 ± 10.4	86.0 ± 15.6
Glycine, $\mu mol \cdot L^{-1}$	326.3 ± 29.3	311.1 ± 41.1	261.3 ± 40.5	288.1 ± 62.3	293.8 ± 47.8	299.6 ± 51.3
Histidine, $\mu mol \cdot L^{-1}$	94.1 ± 4.6	91.1 ± 4.6	85.2 ± 12.2	83.7 ± 3.2	89.6 ± 9.9	87.4 ± 5.4
Isoleucine, $\mu mol \cdot L^{-1}$	74.4 ± 7.9	74.3 ± 13.6	66.4 ± 26.2	72.3 ± 11.3	70.4 ± 18.7	73.3 ± 11.8
Leucine, $\mu mol \cdot L^{-1}$	134.8 ± 12.1	136.9 ± 23.8	144.5 ± 25.5	133.4 ± 16.6	139.6 ± 19.5	135.2 ± 19.4
Lysine, $\mu mol \cdot L^{-1}$	246.4 ± 72.5	250.8 ± 77.3	194.3 ± 46.7	199.6 ± 37.4	220.4 ± 63.7	225.2 ± 63.3
Methionine, $\mu mol \cdot L^{-1}$	30.5 ± 3.0	32.3 ± 2.5	26.8 ± 5.2	23.3 ± 3.2	28.7 ± 4.4	27.8 ± 5.5
Phenylalanine, $\mu mol \cdot L^{-1}$	57.7 ± 11.0	60.5 ± 9.4	56.6 ± 4.6	52.1 ± 4.0	57.2 ± 8.0	56.3 ± 8.1
Proline, $\mu mol \cdot L^{-1}$	252.0 ± 77.7	277.5 ± 97.3	242.1 ± 63.2	224.4 ± 25.0	247.0 ± 67.0	251.0 ± 72.6
Serine, $\mu mol \cdot L^{-1}$	160.3 ± 15.6	168.9 ± 38.1	146.3 ± 18.0	146.3 ± 12.8	153.3 ± 17.5	157.6 ± 29.3
Taurine, $\mu mol \cdot L^{-1}$	77.8 ± 25.9	758.6 ± 311.0[a]	59.3 ± 15.7	790.0 ± 85.7[a]	68.6 ± 22.4	774.3 ± 215.7[a]
Threonine, $\mu mol \cdot L^{-1}$	158.1 ± 26.6	147.6 ± 33.2	137.6 ± 11.9	136.9 ± 20.7	147.9 ± 22.2	142.3 ± 26.7
Tryptophan, $\mu mol \cdot L^{-1}$	54.7 ± 6.9	58.3 ± 8.8	53.7 ± 9.0	50.0 ± 6.5	54.2 ± 7.6	54.2 ± 8.5
Tyrosine, $\mu mol \cdot L^{-1}$	60.7 ± 7.1	65.1 ± 9.2	58.9 ± 13.7	53.5 ± 8.9	59.8 ± 10.3	59.3 ± 10.5
Valine, $\mu mol \cdot L^{-1}$	252.5 ± 26.6	252.7 ± 52.8	275.3 ± 38.4	254.6 ± 48.3	263.9 ± 33.4	253.6 ± 47.7
BCAA, $\mu mol \cdot L^{-1}$	461.7 ± 42.9	464.0 ± 89.9	486.2 ± 85.7	460.3 ± 70.9	474.0 ± 65.2	462.1 ± 76.4
LNAA, $\mu mol \cdot L^{-1}$	580.2 ± 46.5	589.6 ± 103.1	601.8 ± 98.9	566.0 ± 70.0	591.0 ± 73.8	577.8 ± 84.0
Tryp/BCAA	0.12 ± 0.01	0.13 ± 0.03	0.11 ± 0.02	0.11 ± 0.02	0.12 ± 0.02	0.12 ± 0.03
Tryp/LNAA	0.09 ± 0.01	0.10 ± 0.02	0.09 ± 0.01	0.09 ± 0.01	0.09 ± 0.01	0.10 ± 0.02

Values are expressed as mean ± SD.
[a]A significant difference to PLA ($P < 0.05$).

TABLE 4. Effect of taurine on caffeine withdrawal symptoms in caffeine and noncaffeine consumers.

	Drowsiness/ Fatigue	Decreased Alertness/ Difficulty Concentrating	Mood Disturbances	Decreased Sociability/ Motivation to Work	Nausea/Upset Stomach	Flu-like Symptoms	Headache	Total Withdrawal
Caffeine consumers								
Baseline	0.58 ± 0.41	1.17 ± 0.53[b]	0.17 ± 0.41	1.96 ± 1.19	0.00 ± 0.00	0.72 ± 0.61	0.17 ± 0.41	4.76 ± 2.58
Pre-PLA	0.67 ± 0.61	1.50 ± 0.68[a]	0.38 ± 0.80	2.71 ± 1.02	0.00 ± 0.00	0.39 ± 0.49	0.17 ± 0.41	5.81 ± 2.87
Post-PLA	0.42 ± 0.30[b]	1.37 ± 0.72	0.13 ± 0.21	2.29 ± 0.84	0.00 ± 0.00	0.44 ± 0.34	0.33 ± 0.52	4.98 ± 2.32
Pre-TAU	1.17 ± 0.63[a]	1.43 ± 0.45	0.17 ± 0.20	2.50 ± 0.27	0.00 ± 0.00	0.94 ± 0.65	0.00 ± 0.00	6.21 ± 1.34
Post-TAU	0.75 ± 0.67	1.30 ± 0.41	0.13 ± 0.21	2.42 ± 0.38[c]	0.00 ± 0.00	0.72 ± 0.39	0.00 ± 0.00	5.31 ± 0.87
Noncaffeine consumers								
Baseline	0.96 ± 0.49	1.87 ± 0.69	0.42 ± 0.56	2.58 ± 0.94	0.08 ± 0.20	0.56 ± 0.34	0.00 ± 0.00	6.46 ± 2.62
Pre-PLA	0.65 ± 0.38	1.24 ± 0.43	0.05 ± 0.11	2.20 ± 0.91	0.20 ± 0.27	0.53 ± 0.61	0.20 ± 0.45	5.07 ± 1.74
Post-PLA	0.95 ± 0.54	1.32 ± 0.61	0.10 ± 0.22	2.45 ± 0.78	0.10 ± 0.22	0.47 ± 0.65	0.00 ± 0.00	5.39 ± 1.74
Pre-TAU	0.65 ± 0.45	1.52 ± 0.54	0.25 ± 0.35	2.45 ± 0.78	0.20 ± 0.45	0.47 ± 0.30	0.00 ± 0.00	5.54 ± 2.03
Post-TAU	0.70 ± 0.89	1.64 ± 0.77	0.30 ± 0.45	2.45 ± 0.69	0.10 ± 0.22	0.33 ± 0.24[a]	0.00 ± 0.00	5.52 ± 2.31

Values are expressed as mean ± SD.
[a]A significant difference to baseline ($P < 0.05$).
[b]A significant difference to noncaffeine consumers ($P < 0.05$).
[c]A significant difference to PLA, respectively ($P < 0.05$).

markedly affected by one's caffeine intake habits. This finding has important implications for the design of studies investigating the ergogenic benefit of taurine because it implies that the proportion of habitual caffeine consumers recruited to a given study has the potential to affect markedly its findings. This is best illustrated by our own results showing that when all the noncaffeine consumers and caffeine-deprived caffeine consumers recruited to our study were grouped together, as one would normally do for this type of study, the resulting effect of taurine administration differed from those obtained with either noncaffeine or caffeine-deprived caffeine consumers (Fig. 1). In other words, positive, negative, or no effect of taurine is a likely outcome depending on the proportion of habitual caffeine consumers being recruited. Because the proportion of habitual caffeine consumers was not reported in previous studies concerning the ergogenic benefits of taurine on different types of exercise modalities (6–11), this raises the issue of whether the contradicting results between some of these studies are due to possible differences between studies in their proportions of noncaffeine consumers.

Given the overlap between caffeine and taurine effects on intramuscular calcium release (3,14), maybe the removal of caffeine from the caffeine consumers involved in our study contributed to a fall in their muscle contractile performance that was overcome, in part, by taurine ingestion, thus explaining why no negative effect was detected. In support of this interpretation, short-term caffeine abstinence in regular caffeine consumers is associated with caffeine withdrawal symptoms (25) that can adversely affect several aspects of human performance such as reaction time reaction time (26) and mood (25), with caffeine ingestion being proposed to restore performance to baseline rather than improving it (27,28). Maybe taurine acts in a similar way in caffeine-withdrawn individuals. However, this interpretation is difficult to reconcile with our finding of the negative effect taurine administration has on muscle strength and power in nonhabitual caffeine consumers and with the findings by others that some aspects of exercise performance are not significantly affected by caffeine withdrawal (29,30). Moreover, the intensity of the caffeine withdrawal symptoms experienced by our participants was at best minor, including marginally more fatigue from baseline scores after caffeine abstinence, but not after taurine ingestion.

Because this is the first study to have examined the effect of taurine ingestion on maximal muscle contractile performance in humans, this limits our ability to compare our findings with those of others and to identify the mechanisms underlying the inhibitory effect of taurine. Of note, however, a recent study using isolated smooth muscle preparations showed that the effect of taurine on smooth muscle contractility is dependent on muscle contractility state, with taurine stimulating contraction in muscles in a low-contractile state, but inhibiting muscle contraction when in a high-contractile state (31). Furthermore, evidence was provided that the inhibitory effect of taurine was β-adrenergic and nitric oxide mediated. Although taurine administration has been shown to inhibit sympathoadrenal activity in humans (32), the absence of any inhibitory effect of β-adrenoceptor blockade on maximal anaerobic power in humans (33,34) despite its marked inhibitory effect on maximal aerobic capacity (35) suggests that the inhibitory effect of taurine on muscle maximal power and torque reported here is not mediated by impaired sympathoadrenal activity. A role for nitric oxide is also questionable. Indeed, because taurine increases nitric oxide production by endothelial cells (36), one may propose that a taurine-mediated endothelial rise in nitric oxide in the blood vessels perfusing skeletal muscles might be involved because nitric oxide has been shown in some but not all studies to inhibit muscle contraction (37,38). However, the involvement of such a mechanism is challenged by nitric oxide short half-life. It is also unlikely that changes in intramuscular taurine levels and associated effect on intramuscular osmotic pressure mediate taurine's inhibitory effect via a direct effect on sarcoplasmic calcium release and storage (3). This is because others have reported that taurine fails to accumulate within human muscle even after 7 d of chronic supplementation (39).

The inhibitory effect of taurine ingestion on some aspects of muscle contractile performance in noncaffeine consumers is unlikely to be explained by a placebo effect. This is because an increase rather than a decrease in performance would be expected to result from such an effect. Also, both placebo and taurine were encapsulated, with no participants noticing any difference in the taste and appearance of the

capsules between testing sessions, and no participants reporting any change in adverse reactions in response to both treatments. Finally, this study not only adopted a counterbalanced experimental design but also was designed in such a way that participants and experimenters were double blinded to the experimental treatments, and participants were deceived about the true purpose of the study.

Given that taurine has been reported to inhibit the release and synthesis of serotonin (5-HT) from tryptophan in primary brain cell cultures (40), we undertook to examine if the detrimental effect of taurine ingestion could be mediated by acute changes in plasma tryptophan/BCAA ratios, particularly in noncaffeine consumers. An increase in this ratio has been reported to be associated with an increase in tryptophan uptake by the brain. Because this amino acid is the precursor of 5-HT, which in turn is believed to mediate fatigue and deteriorate exercise performance (41), a high ratio would be expected to be detrimental to exercise performance. In contrast, when plasma BCAA levels are elevated, tryptophan uptake by the brain is reduced, and this has been reported to improve exercise performance (42). Because, in the current study, taurine ingestion did not increase plasma tryptophan/BCAA ratios, with these ratios being comparable to those published by others (42), it is unlikely that this variable explains the detrimental effect of taurine on muscle contractile performance. It is important to note, however, that changes in the levels of brain neurotransmitters other than 5-HT might be implicated. For instance, taurine is a partial agonist of glycine receptors *in vitro* (43) and acts as both a neurotransmitter and a neuroprotectant in the brain (44). It is unclear, however, whether any of these effects of taurine contribute to the detrimental effect of taurine, because the role of these neurotransmitters in muscle maximal voluntary power and torque remains to be examined. Of note, it is possible that the taurine dose used in our study might have been detrimental to the muscle contractile performance of the noncaffeine consumers. This interpretation is indirectly supported by the finding that caffeine at an optimal dose improves some aspects of human performance, such as reaction time and alertness, whereas ingesting caffeine beyond such an optimal dose results in detrimental effects (45).

Given our finding that taurine is detrimental to muscle contractile performance in noncaffeine consumers, this raises the obvious question of whether this is also the case with taurine-rich commercial energy drinks. It would be premature to make such an inference on the basis of our results because these energy drinks contain other additives (e.g., caffeine) that have the capacity to affect human exercise performance (5,46). Also, because the taurine content per serve of these drinks is lower than that used in our study, maybe the amount of taurine in these drinks is not high enough to be detrimental to maximal voluntary power and torque while being high enough to enhance endurance performance (5,46). Is the presence of taurine in these energy drinks opposing or enhancing the benefits of other additives, such as caffeine? This is an important issue to address given the evidence that caffeine/taurine-based energy drinks do not improve sprint performance in trained athletes (12,13).

In conclusion, this study shows for the first time that acute taurine ingestion in overnight fasted noncaffeine consumers impairs maximal voluntary muscle power and both maximal isokinetic and isometric voluntary peak torque. In contrast, taurine ingestion in caffeine-deprived caffeine consumers increases muscle power without affecting any of the other indicators of muscle contractile performance examined here. These findings thus imply that the caffeine habit of an individual can affect her/his response to ergogenic aids, with the overall effect of taurine on the average performance of a group of participants being affected by the proportion of habitual caffeine consumers. The mechanisms underlying these confounding effects of caffeine habits on taurine effects remain to be elucidated.

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