Title:

Acute taurine supplementation enhances thermoregulation and endurance cycling performance in the heat

Type: Original Investigation

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Abstract

This study investigated the effects of oral taurine supplementation on cycling time to exhaustion at a fixed-intensity and thermoregulation in the heat. In a double-blind, randomised crossover design, 11 healthy males participated in a time to exhaustion test in the heat (35 °C, 40% RH), cycling at the power output associated with ventilatory threshold, 2-h after ingesting: Taurine (50 $mg \cdot kg^{-1}$) or placebo (3 $mg \cdot kg^{-1}$ maltodextrin). Core and mean skin temperature, mean sweat rate, heart rate, rating of perceived exertion (RPE), thermal comfort and thermal sensation were measured during exercise and blood lactate concentration (B[La]) was measured after exercise. Taurine supplementation increased time to exhaustion by 10% (25.16 min *vs.* 22.43 min, $p = 0.040$), end sweat rate by 12.7% (687 $nL \cdot min^{-1}$ *vs.* 600 $nL \cdot min^{-1}$, $p = 0.034$) and decreased B[La] by 16.5% (5.75 $mmol \cdot L^{-1}$ *vs.* 6.85 $mmol \cdot L^{-1}$, $p = 0.033$). Core temperature was lower in the final 10% of the time to exhaustion (38.5 °C *vs.* 38.1 °C, $p = 0.049$). Taurine supplementation increased time to exhaustion and local sweating, while decreasing RPE and core temperature in the later stages of exercise, as well as reducing post-exercise B[La]. This study provides evidence of taurine's role in thermoregulatory processes. These findings have implications for the short-term preparation strategies of individuals exercising in the heat. Based on these findings, a single dose of taurine 2-h prior to training or competition would provide an ergogenic and thermoregulatory effect.

Keywords *amino acids; sweating; cooling; ergogenic aids.*

Introduction

Taurine, a sulphur containing amino acid, is abundantly expressed in mammalian tissue, accounting for 50–60% of the free amino acid pool (Huxtable, 1992). In human skeletal muscle, taurine concentration is approximately four times higher in type I compared to type II fibres (Harris, Dunnett & Greenhaff, 1998). Taurine is widely reported to facilitate a number of biological processes, such as sarcoplasmic reticulum Ca^{2+} handling in both type I and II fibres (Hamilton, Berg, Easton & Bakker, 2006; Dutka, Lamboley, Murphy & Lamb, 2014) and regulation of cell fluid volume (Cuisinier et al., 2002). Perhaps less well known are the anti-oxidative properties of taurine, elicited though a mitochondrial buffering action (Hansen, Andersen, Birkedal, Cornett & Wibrand, 2006; Hansen, Andersen, Cornett, Gradinaru & Grunnet, 2010), as well as its capacity to act as a neuromodulator, with a suggested influence on the thermoregulatory control centres in animal models (Frosini et al., 2000).

There is a lack of consensus regarding taurine's mechanisms of ergogenic action in humans, yet this semi-essential amino-acid (Huxtable, 1992) is responsible for improvements in endurance performance (Zhang, Izumi, Kagamimori, Sokejima, & Yamagami, 2004; Balshaw, Bampouras, Barry & Sparks, 2012; Ahmadian, Roshan & Ashourpore, 2017; Warnock, Jeffries, Patterson & Waldron, 2017; Waldron, Knight, Tallent, Patterson & Jeffries, 2018a) and is actively carried into most major organs via taurine transporters (TauT) (Ito et al., 2010). While oral taurine supplementation may not increase resting muscle content, increased plasma taurine levels following oral supplementation (Galloway, Talanian, Shoveller, Heigenhauser & Spriet, 2008) are likely to hinder its normal extrusion from skeletal cells (thus supressing intracellular release) as part of an osmoregulatory function during exercise (Cuisinier et al., 2002). Therefore, it is feasible that a combination of mechanisms explain the recent meta-analytic findings, demonstrating taurine's ergogenic effect on endurance performance in humans (Waldron, Patterson, Tallent, & Jeffries, 2018b).

Thermal strain is exacerbated by a combination of prolonged exercise and high environmental temperatures or humidity, which can cause a continuous rise in core temperature beyond homeostatic ranges (Nybo, 2008). The net effects of thermal strain are characterised by the heat balance equation, where heat energy is gained or dissipated via evaporation, conduction, convection and radiation (Gagge & Gonzalez, 1996). In thermally-strained humans, heat is

chiefly dissipated via evaporative and convective processes, as governed by the thermoregulatory centre in the hypothalamus (Gagge & Gonzalez, 1996). Cutaneous vasodilation and sweating (among other processes) support these avenues of heat loss by attenuating the rate of rise in core temperature during exercise (Marino et al., 2000; Sawka, Leon, Montain & Sonna, 2011). In relation to the current study, smaller increases in core temperature have been observed in heat-stressed rabbits compared to thermoneutral controls after intrathecal infusion of taurine (Frosini et al., 2000). It is possible that oral ingestion, rather than central infusion, could elicit similar effects in humans. Furthermore, taurine has a number of peripheral actions, particularly as a vaso-relaxant (Sun et al., 2016), which could facilitate convective and evaporative cooling processes. Taurine's inotropic effects on the heart (Satoh & Sperelakis, 1998) and possible influence on stroke volume (Baum & Weiß, 2001) might also offset the deleterious effects of heat stress on cardiac efficiency and subsequent endurance performance (Gonzalez-Alonso, 2012). However, none of these questions have been addressed in exercising, thermally-stressed humans.

The purpose of this study was to investigate the effects of acute oral taurine supplementation on thermoregulation during cycling exercise of a fixed intensity in the heat (35 °C; 40% relative humidity). It was hypothesised that taurine would extend time to exhaustion in the heat and delay the rate of rise in core temperature by enhancing convective or evaporative cooling potential.

Materials and methods

Participants

Eleven non-heat acclimated healthy males volunteered for this study (age 23 ± 2 years, stature 180 ± 60 cm, body mass 83.0 ± 9.5 kg, maximal oxygen uptake ($\dot{V}O_{2max}$) 46.0 ± 6.3 $ml \cdot min^{-1} \cdot kg^{-1}$, maximal aerobic power output 305 ± 29 W). Given the typical effect sizes (Cohen's d = 0.4) reported using taurine to improve endurance performance (Waldron et al., 2018b) in thermoneutral environments, G*Power (Version 3.0.10; Universität Düsseldorf, Germany) was used to calculate an *a-priori* sample size of 11, which was sufficient to identify differences between groups with a statistical power of 0.80. Participants were asked to refrain from alcohol and caffeine consumption for 24-h and avoid strenuous exercise for 48-h prior to testing. All

participants gave written informed consent. Ethical approval was provided by the institutional ethics committee, which was conducted in accordance with the 1964 Helsinki declaration.

Design

The study adopted a double-blind, randomised crossover design. All participants reported to the laboratory on three separate occasions, each separated by a maximum of 96-h and minimum 72-h to minimise acclimation effects. The first visit comprised preliminary testing and familiarisation. On visits two and three, the participants completed the experimental trials, where they ingested taurine or placebo 2-h before exercise. Randomisation was conducted by generating random numbers for all participants, across each condition using online software. Each laboratory visit was conducted at the same time of day.

Preliminary testing

During visit one, participants undertook an incremental exercise test to volitional exhaustion on a mechanically-braked cycle ergometer (Monark Exercise AB, Ergomedic 874E, Varberg, Sweden) in thermoneutral conditions (19.2 ± 0.8 °C) to determine $\dot{V}O_{2max}$ and the power output at ventilatory threshold. Participants cycled for 5-min at 80 W to warm-up and rested for 5-min before starting the test. The test started at a workload of 120 W and increased 24 W·min^{-1} at a fixed cadence of 80 rev·min^{-1} until volitional exhaustion or when cadence dropped below 70 rev·min^{-1} for more than 10-s. Oxygen uptake ($\dot{V}O_2$) was measured using breath-by-breath expired air analysis (Jaeger Vyntus CPX, Hoechberg, Germany). The gas analyser was calibrated before every trial with gases of known concentration (15.95% O_2, 4.97% CO_2, BAL. N_2) and the turbine volume transducer was calibrated automatically by the system at flow values of 2 L·s^{-1} and 0.2 L·s^{-1}. Heart rate (HR) was recorded throughout the trial (Polar Heart Rate Monitor M400, Warwick, UK). $\dot{V}O_{2max}$ was calculated by measuring the highest 30-s average $\dot{V}O_2$ peak power was measured as the highest power output recorded during the test for a full minute. After 20-min of rest, participants conducted a familiarisation, comprising maximal constant load exercise at the power output associated with ventilatory threshold in an environmental chamber (Sporting Edge UK, Basingstoke, UK) set to experimental conditions (35 ± 0.3 °C, 40 ± 0.3 % RH). Breath-by-breath $\dot{V}O_2$ and $\dot{V}CO_2$ data from the incremental cycling test was used to plot ventilatory threshold, using the simplified v-slope method (Scheider, Phillips & Stoffolano, 1993). The mean power output at thermoneutral ventilatory threshold was 203 ± 23 W, which was fixed for all experimental trials. This threshold was

selected as it was deemed appropriate to evaluate endurance capacity at a repeatable, sub-maximal fixed intensity, while increasing the rate of metabolic heat production sufficient to induce thermoregulatory responses.

Experimental trials

All subsequent tests were conducted in the heat (35 ± 0.3 °C, 40 ± 0.6 % RH). Upon arrival at the laboratory, 1-h prior to testing, participants were instructed to insert a rectal thermistor (Edale Instruments Ltd, Cambridge, UK) 10 cm past the anal sphincter to measure core temperature (T_{core}). T_{core} was recorded every 1-min via a scanning thermometer type CDS 1.0 (Edale Instruments Ltd, Cambridge, UK). A urine sample was also provided to determine hydration status using a refractometer (Pocket Osmochek, Vitech Scientific Ltd, West Sussex, UK). A reading of > 600 mOsm·kg^{-1}·H_2O^{-1} indicated the threshold of hypohydration, in which case the participant consumed 500 ml of water and waited 30-min before testing.

The participants' body mass was recorded wearing cycling shorts and rectal thermistor inserted. The participants were instructed to abstain from drinking fluids until the end of the test. Skin thermistors (Grant Instruments Ltd., Cambridge, UK) were then attached to four sites on the participants' right side: upper chest, mid humerus, mid-calf and mid-thigh. Skin temperature was recorded continuously via a Squirrel data logger (SQ2010, Grant Instruments Ltd., Cambridge, UK) and reported every 1-min. Mean skin temperature (T_{skin}) was calculated using Ramanathan's equation (Ramanathan, 1964): $T_{skin} = 0.3 \times (T_{chest} + T_{arm}) + 0.2 \times (T_{thigh} + T_{calf})$. Prior to fitting skin thermistors, the skin was cleaned with soap and water and dry shaved, before being thoroughly dried. Local sweat rate was measured using a Q-Sweat system (WR Medical Electronics Co., Stillwater, MN). Ventilated capsules were fixed proximal to the skin thermistors on the right-side of the body. Dry air was continuously delivered through Teflon-lined tygon tubing at a fixed flow rate of 60 sccm·min^{-1} between four separate sweat capsules mounted on the skin's surface and the Q-sweat device. Fluid released from the sweat glands is transiently incorporated into the dry air, with the change in temperature and relative humidity monitored by the Q-Sweat device. The capsule size was 0.781 cm^2. Sweat rate was calculated using standard vapour pressure equations and expressed in nL·min^{-1}. Mean sweat rate was calculated by averaging the four sweat sites and reported every 1-min.

Participants then entered the environmental chamber and completed a 3-min warm-up at 80 W, followed by 2-min of rest. Participants were instructed to maintain a pedal cadence of 80

rev·min^{-1} at an intensity equivalent to thermoneutral ventilatory threshold until complete exhaustion. Exhaustion was defined as voluntary withdrawal or when pedal cadence dropped below 70 rev·min^{-1} for more than 10-s. The coefficient of variation for this test in our laboratory is 3.8% while cycling in the heat. Ratings of perceived exertion (RPE) were recorded on a 6 to 20 point Borg scale. Thermal comfort (TC) was recorded on a 7-point scale where -3 = "much too cool", 0 = "comfortable", and 3 = "much too warm" (Bedford, 1936). Thermal sensation (TS) was recorded on a 9-point scale where -4 = "very cold", 0 = "neutral", and 4 = "very hot". RPE, TC and TS were recorded every 3-min and at completion. One-min post exercise, a blood lactate sample was taken from the right ear lobe using a lancet and analysed by an automated analyser (Biosen C_Line, EKF diagnostic GmbH, Barleben, Germany). Skin thermistors and sweat capsules were also removed post-exercise, before being towel-dried and re-weighed to indicate fluid losses during exercise.

Supplementation

All supplements were prepared in powder form, which were measured using an analytical balance (Precisa 125A, Precisa Gravimetrics AG, Zurich, Switzerland) and ingested in a gelatine capsule. The capsules contained one of the following: taurine (T) (50 mg·kg^{-1} BM) or a placebo (P) (maltodextrin) (3 mg·kg^{-1} BM) and were administered to participants 2-h prior to exercise. The 2-h timing was chosen as it captures the likely range of peak plasma availability of taurine after oral administration (Galloway et al., 2008; Ghandforoush-Sattari, Mashayekhi, Krishna, Thompson & Routledge, 2010). Participants' body mass was taken prior to each trial to measure the correct dose and the supplements were balanced such that an equal number of capsules were ingested between conditions. The taurine dosage followed published recommendations (Warnock et al. 2017; Waldron et al., 2018a). The supplements were sourced from the company (My Protein, Manchester, UK).

Statistical analysis

A two-way repeated measures analysis of variance (RM-ANOVA) was conducted, with condition (taurine or placebo) and time (10% epochs across the trials) as the independent variables. A Greenhouse-Geisser correction was applied when the assumption of sphericity was violated. Where interaction effects were found, *post-hoc* analysis was performed with Bonferroni tests to identify pairwise differences. Two-tailed paired samples *t-test* identified significant differences between the performance trials. A Wilcoxon Signed-Rank test was performed on non-parametric data across time points. Statistical significance was accepted at *P*

< 0.05 and all analyses were performed on IBM SPSS Statistics (Version 21, IBM Corp., Armonk, NY, USA). The magnitude of effect was calculated with partial eta-squared (η_p^2) according to the following criteria: 0.02, a small difference; 0.13, a moderate difference; 0.26 a large difference (Cohen, 1988). Cohen's *d* was calculated to interpret the effect of pairwise changes.

Results

Time to exhaustion at the power output associated with ventilatory threshold in 35 °C/40% RH was increased by ~ 10% with nine out of the eleven participants extending their performance after taurine supplementation (Taurine = 25.16 ± 5.25 min; Placebo = 22.43 ± 4.28 min) ($t_{(10)}$ = 2.353, p = 0.040, Cohen's d = 0.70) (Figure 1). There were also no trial order effects of the time to exhaustion (P = 0.203).

***** Insert Figure 1 here *****

Core temperature increased with time ($F_{(19,190)}$ = 174.954 , p < 0.001, η_p^2 = 0.946), with no main effect for condition ($F_{(1,10)}$ = 0.035 , p = 0.855, η_p^2 = 0.004); however, there was a condition × time interaction ($F_{(19,190)}$ = 3.715 , p < 0.001, η_p^2 = 0.271). Pairwise analysis confirmed that core temperature was reduced in the taurine condition only at the 100% epoch (Placebo = 38.5 ± 0.4 °C; Taurine = 38.1 ± 0.4 °C) ($t_{(10)}$ = 2.241, p = 0.049) (Figure 2 A). Mean skin temperature increased with time in both conditions ($F_{(19,190)}$ = 77.692 , p < 0.001, η_p^2 = 0.886), without condition effects ($F_{(1,10)}$ = 1.235 , p = 0.293, η_p^2 = 0.110) or an interaction with time ($F_{(19,190)}$ = 1.562, p = 0.069, η_p^2 = 0.135) (Figure 2 C).

***** Insert Figure 2 here *****

Mean sweat rate increased with time in both conditions ($F_{(19,190)}$ = 51.921 , p < 0.001, η_p^2 = 0.839) and was increased in the taurine condition relative to placebo ($F_{(1,10)}$ = 23.218 , p = 0.001, η_p^2 = 0.699). There was also a large condition × time interaction effect ($F_{(19,190)}$ = 3.654, p < 0.001, η_p^2 = 0.268) (Figure 3). Post-hoc analysis revealed that mean sweat rate was higher

at all stages beyond 20 % into the exercise test ($p < 0.05$). At the end of the exercise trial, mean local sweat rate was ~13% higher in the taurine condition (Taurine = 687 ± 144 nL·min^{-1}; Placebo = 600 ± 194 nL·min^{-1}) ($t_{(10)} = 9.624$, $p = 0.034$). Pre-to-post changes in body mass were also increased ~ 42 % in the taurine condition (Taurine = 786 ± 232 g; Placebo = 455 ± 281 g) ($t_{(10)} = 5.434$, $p = 0.024$).

***** Insert Figure 3 here *****

Heart rate increased with time in both conditions ($F_{(1.743,17.432)} = 166.847$, $p < 0.001$, $\eta_p^2 = 0.943$); however, there were no main effects for condition ($F_{(1,10)} = 0.410$, $p = 0.536$, $\eta_p^2 = 0.039$), nor was there an interactions ($F_{(1.706,17.062)} = 0.512$, $p = 0.863$, $\eta_p^2 = 0.049$). A Wilcoxon Signed-Rank test indicated differences in RPE at 18-min (Taurine = 18 ± 1; Placebo = 19 ± 1; $Z = -2.264$, $p = 0.024$) and end exercise (Taurine = 19 ± 1; Placebo = 20 ± 1; $Z = -2.449$, $p = 0.014$) (Figure 4 A). There were no differences between conditions for thermal comfort or thermal sensation ($p > 0.05$) (Figure 4 B&C).

***** Insert Figure 4 here *****

Post-test B[La] was reduced by 16.5% following taurine supplementation (Taurine = 5.75 ± 2.15 mmol·L^{-1}; Placebo = 6.85 ± 1.77 mmol·L^{-1}) ($t_{(10)} = -2.470$, $p = 0.033$).

Discussion

We investigated the effects of taurine supplementation on thermoregulation and time to exhaustion in the heat. The key findings were that time to exhaustion in the heat (35 °C) increased by ~ 10% and sweat rate by ~ 12.7% at end exercise following taurine supplementation. The 10% increase in time to exhaustion surpassed the CV% of 3.8%, thus denoting a true change in performance. Whilst there were no consistent differences in core and skin temperature between conditions during the earlier stages of the time to exhaustion, a

lower core temperature was found during the final 10% of the trial and there was a trend for 'small' reductions in skin temperature based on effect sizes. Collectively, these thermoregulatory changes might partly explain the observed improvement in performance.

There is growing evidence to support the efficacy of taurine as an ergogenic aid for exercise performance (Souza, Del Coso, Casonatto & Polito, 2016; Waldron et al., 2018b). These changes are commonly attributed to improved mechanical efficiency (Paulucio et al., 2017) or improved efficiency of ATP turnover in the muscle cell (Hansen et al., 2010). Here, we extend these observations to report an increase in performance of 10% in the heat (35 °C). This change was larger than the typical 2-6% increases in performance reported following oral taurine administration in thermoneutral environments (Zhang et al., 2004; Balshaw et al., 2012; Waldron et al., 2018a). Given that taurine's capacity to elicit changes in time to exhaustion is markedly larger in the heat, our findings naturally infer a role for taurine in thermoregulatory control processes.

Endurance exercise in the heat ceases for number of physiological reasons (Tucker & Noakes, 2009). For example, during exhaustive, fixed-resistance exercise in hot conditions, reductions in cycling power output have been reported, alongside declines in cardiac output and stroke volume, thus impairing muscle blood flow and oxygenation (Gonzalez-Alonso & Calbet, 2003). Taurine has a well-established inotropic action on cardiac muscle fibre (Satoh & Sperelakis, 1998) and has been shown to increase stroke volume when consumed as part of an energy drink (Baum & Weis, 2001). Given the established effects of taurine on cardiac myocytes, this might have been anticipated to offset fatigue-induced reductions in cardiac efficiency during performance. However, there were no changes in heart rate, which are likely to have been lower in the taurine condition if this mechanism were to explain the change in performance. As such, the increased performance and delayed fatigue in the taurine condition most likely occurred for different reasons.

The changes observed in local sweat rate, total body fluid losses and core temperature were remarkable and highlight, for the first time, a significant role for taurine in human thermoregulation. Sweat response from eccrine glands is governed by the thermoregulatory centre in the hypothalamus, which directs the sympathetic nervous system to increase sweat secretion (Shibasaki & Crandall, 2010). These changes in sweat response occurred from the early stages of the exercise bout, prior to excessive exercise or external thermal stimulus, and

appear to have influenced skin temperature, which was also lower in the taurine condition. Core temperature is the net result of heat gain and dissipation, with sweating contributing > 70% to heat loss during exercise in hot conditions (Marino et al., 2000). Thus, thermoregulation is increasingly dependent upon evaporative cooling as an avenue of heat loss during exercise. Taurine ingestion appears to augment the local sweating responses, such that the rate of rise in core temperature at the later stages of the trial was reduced compared to placebo, culminating in a lower core temperature at the end-point of the trial. The cooler skin temperature also generates a greater core-shell gradient, thus facilitating convective heat loss. The increased thermal stress observed during the placebo condition is most likely responsible for the earlier cessation of exercise. Indeed, hyperthermia may cause sub-conscious anticipatory reductions in skeletal muscle recruitment and subsequent force production of the lower limbs prior to the attainment of the hypothesised 'critical' (~ 40 °C) core temperature (Tucker, Rauch, Harley & Noakes, 2004). Others have reported relationships (r^2 = 0.98) between core temperature and electroencephalographic $\alpha - to - \beta$ wave ratios, which indicate supressed arousal levels and can be used to infer a reduction in central drive (Nielsen, Hyldig, Bidstrup, Gonzales-Alonso & Cristoffersen, 2001). Therefore, hyperthermic fatigue is mostly commonly ascribed to centrally-acting mechanisms (Nybo, 2008).

Taurine acts as a neuromodulator in various brain areas and is a $GABA_A$ receptor agonist (Jia et al., 2008). Heat stress induces the release of both GABA and taurine from the hypothalamus into the cerebrospinal fluid (CSF), indicating their activity in response to temperature changes in the domain of thermoregulatory control (Frosini et al., 2000). Similarly, patients with heat stroke, who exhibit an array of neurological disorders, demonstrate increases in urinary taurine concentrations following cellular release, that are subsequently reduced following recovery from hyperthermic states (Bouchama, El-Yazigi, Yusuf & Al-Sediary, 1993). Whilst we did not intend to directly measure central mechanisms in this study, the marked increase in autonomic sudomotor responses demonstrated across most stages of the trials in the taurine condition strongly infers a change in the centrally-mediated thermoregulatory set-point. More specifically, we postulate that the resulting increase in the onset and volume of sweating in the taurine condition was sufficient to delay the progression of core temperature, thus delaying hyperthermic fatigue and extending time to exhaustion. Interestingly, plasma taurine can readily cross the blood-brain barrier via a TauT (Kang, 2000) and infiltrate hypothalamic regions of the brain, albeit more slowly than central

infusions. The accumulation of taurine in these central locations presumably reduces core temperature via taurine binding sites or antagonism of GABA receptors, which are established effectors of hypothermia via distinct neural pathways (Quéva et al., 2003; Frosini et al., 2003). Higher plasma availability following oral ingestion is likely to drive the transport of taurine across the blood-brain barrier into these central areas, where it can interact with target receptors and instigate thermoregulatory responses, such as increasing sudomotor function. The changes found in local sweating and core temperature in the taurine condition have implications beyond sports and exercise, which could include the application to occupations requiring routine heat exposure, such as the emergency or military services. Supplements that augment the sweating response could also be useful to counteract milder forms of hyperhidrosis. Further work is needed to corroborate these findings and investigate the application to other populations.

Owing to its molecular structure, taurine is an endogenously free sulfonic acid and acts to control the movement of fluids via osmosis (Cuisinier et al., 2002). Indeed, it is this function that explains its extrusion from intra-cellular compartments during exercise, where it is transported into the extra-cellular space to prevent cell swelling (Stutzin et al., 1999). It is not yet apparent how exogenous supply of taurine effects this process and it has been speculated that high plasma concentrations observed after oral ingestion could supress its normal release from the cell (Ishikura, Song-Gyu & Ohmori, 2013). It is also possible that higher plasma taurine concentrations could attract fluid to the vascular space, thus contributing to plasma volume expansion; however, plasma taurine's relationship with plasma volume is weaker than other osmolytes (Cuisinier et al., 2002). It is unlikely that these osmoregulatory effects would alter sweat production but there are possible cardiovascular implications that require further investigation in the heat.

The similarity in thermal perception between conditions across the relative stages of the protocol was surprising, given the improved thermoregulation in the taurine condition. The trend for a lower thermal sensation at the end stages of the trial in the taurine condition indicates a possible improved perception, yet this didn't reach significance. On the other hand, RPE was reduced at end stages of the trial in the taurine condition. While the power output was fixed between conditions and HR was similar, the increased sweat rate, subsequent reduction in body temperatures and post-exercise B[La] appear to have collectively contributed to the lower RPE. The lower B[La] in the taurine condition was unanticipated,

particularly given the longer time to exhaustion. Whilst changes in B[La] have not typically reported in thermoneutral environments following taurine supplementation (Waldron et al., 2018a; Warnock et al., 2017), it is possible that the reduced thermal strain of the taurine group lowered the rate of glycolytic flux (Febbraio, 2001). This finding is also worthy of further investigation, as it might indicate an indirect influence of taurine on glycolytic metabolism.

A limitation of this study was the absence of plasma or muscle taurine measurements, which would confirm the presence of taurine during the exercising period. However, the timing and availability of plasma taurine following oral ingestion has been established using identical procedures to those adopted here, albeit at lower doses (1.66 g; Galloway et al., 2008), thus, it was assumed that the 50 mg/kg would provide adequate delivery of taurine to exert an ergogenic effect.

Conclusion

This is the first study to examine the effect oral taurine ingestion on thermoregulation during cycling in the heat. Compared to placebo, taurine supplementation of 50 $mg \cdot kg^{-1}$ body mass increased time to exhaustion (~ 10%) and local sweating (~ 12.7%), while decreasing RPE and core temperature in the later stages of exercise, as well as reducing post-exercise B[La]. Therefore, this study also provides the first evidence of taurine's role in thermoregulatory processes. Notably, the profound influence of taurine on sudomotor function was an important finding of the current study and indicates a potential central mechanism, as previously described in animal models. These findings have implications for short-term preparation strategies of individuals exercising in the heat. Based on our findings, a single dose of taurine 2-h prior to training or competition would provide an ergogenic and thermoregulatory effect. Given that this is the first study to report on this topic, future research should attempt to corroborate our findings across different populations and investigate the potential explanatory mechanisms.

Declaration of interest statement

The authors have no conflicts of interest to declare.

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List of Figures

Figure 1

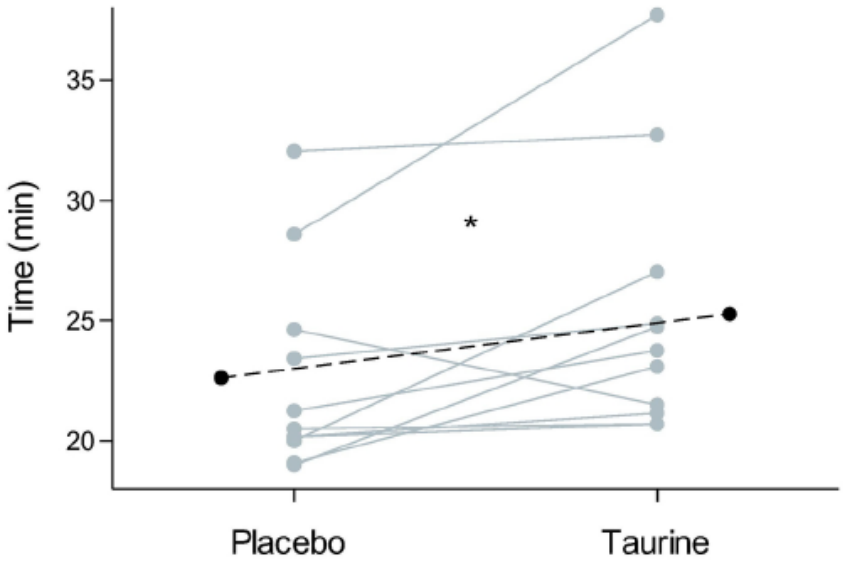


Figure 2

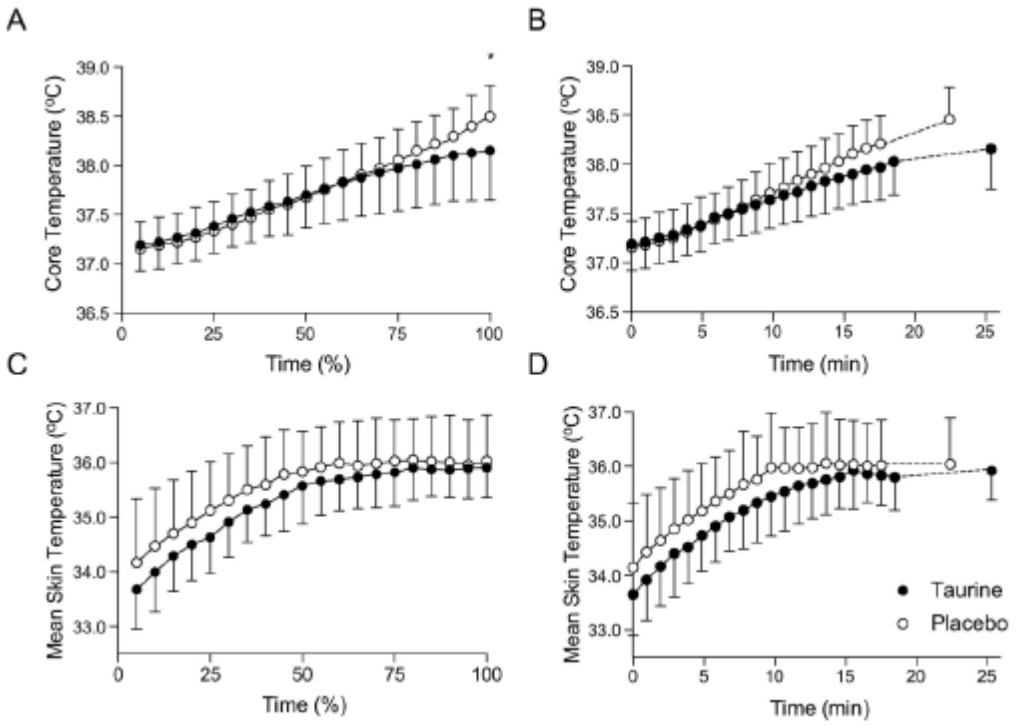

A
B
C
D
Core Temperature (ºC)
Mean Skin Temperature (ºC)
Time (%)
Time (min)
Taurine
Placebo

Figure 3

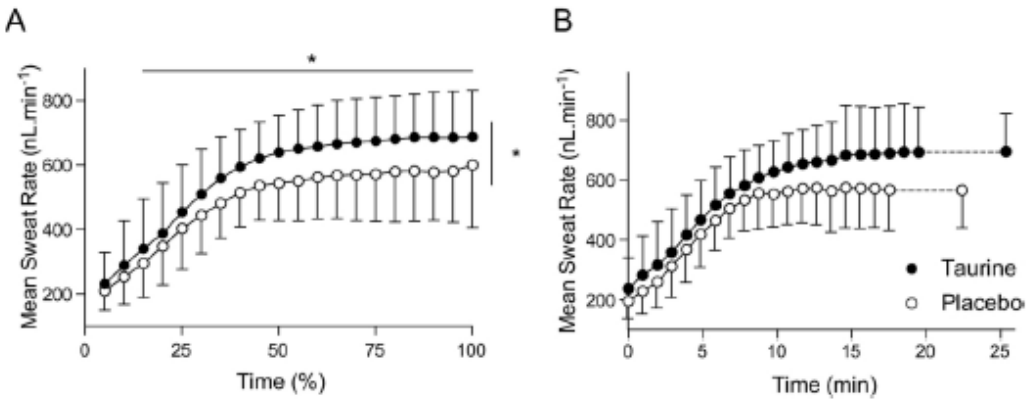
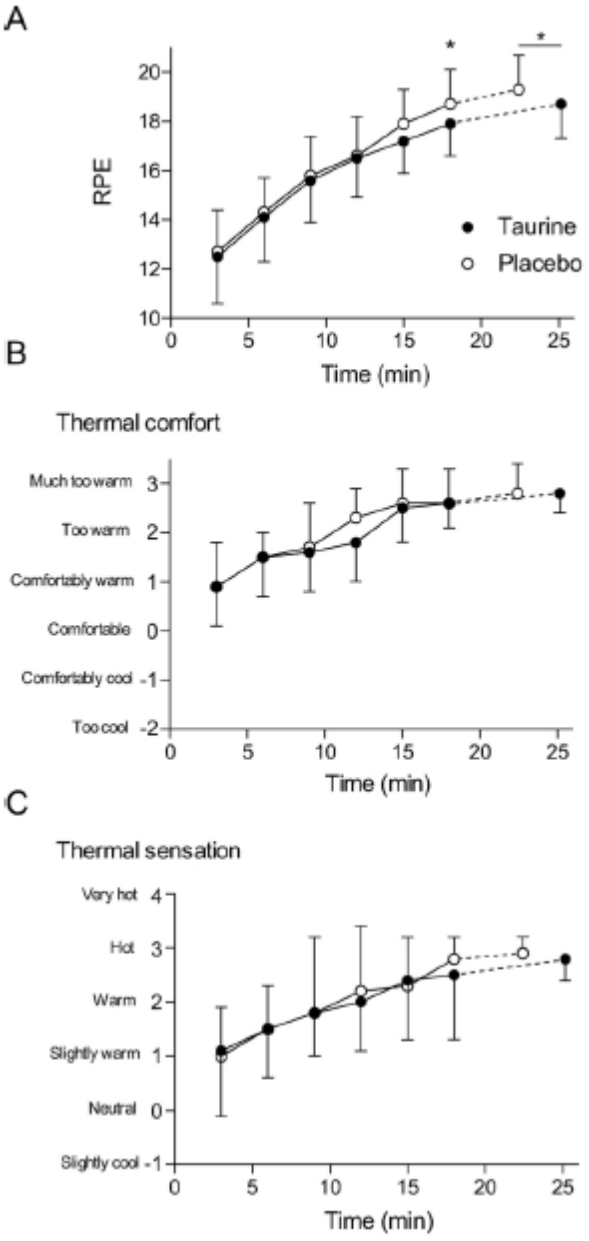

A
B
Mean Sweat Rate (nL.min⁻¹)
800
600
400
200
0
25
50
75
100
Time (%)
*
*
Mean Sweat Rate (nL.min⁻¹)
800
600
400
200
0
5
10
15
20
25
Time (min)
Taurine
Placebo

Figure 4


A
RPE
Taurine
Placebo
Time (min)
B
Thermal comfort
Much too warm
Too warm
Comfortably warm
Comfortable
Comfortably cool
Too cool
Time (min)
C
Thermal sensation
Very hot
Hot
Warm
Slightly warm
Neutral
Slightly cool
Time (min)