Acute caffeine supplementation offsets the impairment in 10-km running performance following one night of partial sleep deprivation - a randomized controlled crossover trial

Authors:
Yi-Shan TSAI[1], Ting-Tzu CHEN[1], Yau-Ching CHAN[1], Chun-Chin HUANG[1], Ting-Fu LAI[2], Yung LIAO[2], Richard S. Metcalfe[3], Yung-Chih CHEN[1], and Ho-Seng WANG[1]*

[1] Department of Physical Education and Sport Sciences, National Taiwan Normal University, Taipei, Taiwan

[2] Graduate Institute of Sport, Leisure and Hospitality Management, National Taiwan Normal University, Taipei, Taiwan

[3] Applied Sports Technology, Exercise and Medicine Research Centre (A-STEM), Swansea University, Bay Campus, Fabian Way, Swansea, UK

Corresponding author:
Professor Ho-Seng WANG
Department of Physical Education and Sport Sciences
National Taiwan Normal University
Taipei, 162, Section 1, Heping E. Rd
Taiwan
Email: t08019@ntnu.edu.tw

Abstract

Introduction: Whether acute caffeine supplementation can offset the negative effects of one-night of partial sleep deprivation (PSD) on endurance exercise performance is currently unknown. **Methods**: Ten healthy recreational male runners (age: 27 ± 6 years; $\dot{V}O_{2max}$: 61 ± 9 ml/kg/min) completed 4 trials in a balanced Latin square design, which were PSD + caffeine (PSD-Caf), PSD + placebo (PSD-Pla), normal sleep (NS) + caffeine (NS-Caf) and NS + placebo (NS-Pla). 3 and 8 h sleep windows were scheduled in PSD and NS, respectively. 10-km treadmill time trial (TT) performance was assessed 45 min after caffeine (6 mg/kg/body mass)/placebo supplementation in the morning following PSD/NS. Blood glucose, lactate, free fatty acid and glycerol were measured at pre-supplementation, pre-exercise and after exercise. **Results**: PSD resulted in compromised TT performance compared to NS in the placebo conditions by 5% (51.9 ± 7.7 vs. 49.4 ± 6.9 min, *p* = 0.001). Caffeine improved TT performance compared to placebo following both PSD by 7.7% (PSD-Caf: 47.9 ± 7.3 min vs. PSD-Pla: 51.9 ± 7.7 min, *p* = 0.007) and NS by 2.8% (NS-Caf: 48.0 ± 6.4 min vs. NS-Pla: 49.4 ± 6.9 min, *p* = 0.049). TT performance following PSD-Caf was not different from either NS-Pla or NS-Caf (*p* = 0.185 and *p* = 0.891, respectively). Blood glucose, lactate, and glycerol concentrations at post-exercise, as well as heart rate and the speed/RPE ratio during TT, were higher in caffeine trials compared to placebo. **Conclusion:** Caffeine supplementation offsets the negative effects of one-night PSD on 10-km running performance.

Key words: endurance performance, sleep deprivation, sports nutrition, marathon, supplements

Introduction

Sleep loss, whether total sleep deprivation (TSD, >24 hours awake) or the more common partial sleep deprivation (PSD, total sleep time per day reduced to only a few hours), negatively impacts exercise training quality, competition preparation, exercise performance, and exercise-induced recovery processes (e.g., impaired muscle damage and repair and/or an increased exercise-induced inflammatory response) (Abedelmalek et al., 2013; Fullagar et al., 2016; Nédélec et al., 2015; Rae et al., 2017). PSD, which can occur due to a delayed onset of sleep or an earlier wake-up time, is particularly prevalent among athletes, often experienced before competition due to pre-game anxiety or travel schedules (Fullagar et al., 2015; Halson, 2014). For instance, surveys have indicated that approximately 65% athletes across various sports (Erlacher et al., 2011) and 70% of elite marathon runners have reported experiencing poorer sleep quality and/or reduced sleep duration (5:51 ± 1:25 hours) (Lastella et al., 2014) prior to competition.

Studies have shown that time-to-exhaustion and/or maximum power output during endurance exercise tests were significantly decrease after TSD (Azboy & Kaygisiz, 2009; Martin, 1981; Oliver et al., 2009). These findings extend to PSD as well, with studies by Mougin et al. (2001) and Souissi et al. (2020) demonstrating negative impacts on endurance exercise performance, including lower peak power output during cycling (~5%) and reduced distance covered during time-trial running (~6%). The reduction in exercise performance resulting from TSD or PSD is thought to be caused by a combination of both physiological and psychological stressors. Insufficient sleep can decrease alertness, neuronal reactivity, and cognitive function, which may impair decision-making (e.g., pacing strategies) and increase perceived effort during prolonged exercise (Fullagar et al., 2015; Rogers et al., 2003; Van Dongen et al., 2003; Van Helder & Radomski, 1989). Physiologically, sleep loss can reduce muscle glycogen concentrations and glucose tolerance, potentially affecting substrate utilization during exercise (Saghiv et al., 2019; Skein et al., 2011; Van Helder & Radomski, 1989). Additionally, cardiopulmonary function, including maximum oxygen consumption ($\dot{V}O2max$) and maximum heart rate, may be compromised due to inadequate sleep (Antunes et al., 2017).

Caffeine is widely available in a variety of foods, drinks, and medications (Temple et al., 2017) and is commonly consumed by athletes prior to and/or during exercise training and competition to elevate performance (Pickering & Grgic, 2019). Studies have demonstrated that a single dose of caffeine (4-10 mg/kg body mass) before exercise can improve both running and cycling time-trial performance (Doherty & Smith, 2005; Ganio et al., 2009; Guest et al., 2021). The physiological and psychological mechanisms underlying caffeine's ergogenic effects overlap with those negatively impacted by sleep deprivation. This mechanistic convergence has led to the use of caffeine supplementation as a potential strategy to mitigate the performance impairments caused by insufficient sleep (Roehrs & Roth, 2008).

Accumulating evidence suggests that caffeine can offset, and in some cases even surpass, the performance decline observed following sleep deprivation (Irwin et al., 2020). To date, studies on TSD or PSD with caffeine supplementation have mainly focused on reaction time, skill related tests, and short duration high-intensity and/or repeated sprint performance (Cook et al., 2011; Donald et al., 2017; Romdhani, Souissi et al., 2021; Souissi et al., 2014; Souissi et al., 2018). However, due to the well-established beneficial effects of caffeine intake on prolonged endurance exercise performance and sleep loss (Guest et al., 2021; Roehrs & Roth, 2008), several studies have also investigated the effects of caffeine on prolonged endurance exercise performance following sleep loss (Khcharem et al., 2021; Khcharem et al., 2022; McLellan et al., 2004). However, it is important to note that these studies have all involved TSD (Khcharem et al., 2021; Khcharem et al., 2022; McLellan et al., 2004) and, up until now, the impact of caffeine supplementation on endurance exercise performance following PSD has not been investigated. This is an important research question as PSD is a common experience for athletes before competition. Therefore, this study aimed to examine the effects of caffeine intake on endurance running performance following a single night of PSD. We hypothesized that caffeine supplementation would mitigate the performance impairment observed after PSD.

Methods

Participants

Ten healthy young men (age: 27 ± 6 years, height: 173.6 ± 6.3 cm, body mass: 64.2 ± 5.8 kg, BMI: 21.3 ± 0.9 kg/m^2, $\dot{V}O_{2max}$: 61.1 ± 9.4 ml/kg/min) were recruited, all of whom were recreational runners performing regular endurance training (two to five times a week), and were familiar with and comfortable running a distance of 10 km (Bell et al., 2002). Because of the possible influences of menstrual cycle phases on exercise performance and sleep parameters (Besson et al., 2022; Greenhall et al., 2020; Hrozanova et al., 2021), we included only male and not female participants in the present study. All participants self-reported that their personal best times for a half-marathon and marathon were within 2 hours and 4.5 hours respectively. A power analysis was performed based on results of 8-km time trial running time from the study of Khcharem et al. (2022), wherein the influences of 5 mg/kg caffeine and 26-hours of TSD were assessed. We calculated that a minimum of 8 participants would be required to detect similar changes in the running performance, with at least 80% statistical power ($\alpha = 0.05$). Participants were non-smokers, non-habitual drinkers and did not report any history of medical conditions (e.g. diabetes or cardiovascular related diseases) and were not currently taking any prescription medications. Their habitual caffeine intake, evaluated with a modified questionnaire (Bühler et al., 2014), was less than 200 mg per day. Taiwanese versions of questionnaires adapted from the Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989) and Morningness/Eveningness Questionnaire (the Horne and Östberg (1976)) were assessed; all participants scored ≤ 5 points and no participant clearly identified as a morning/evening chronotype, respectively.

Study Design

Each participant completed a preliminary testing session and 4 main trials. In the preliminary session, one $\dot{V}O_{2max}$ test and a familiarization trial were assessed at least 48 hours before the main trials. The 4 main trials were partial sleep deprivation with (PSD-Caf) and without caffeine supplementation (PSD-Pla) and normal sleep with (NS-Caf) and without caffeine supplementation (NS-Pla), in a balanced Latin square and randomized cross-over design, with at least 7 days wash-out between trials to avoid order effects. Although the caffeine supplementation interventions were performed in a double-blinded way, due to the nature of sleep intervention, participants were not able to be blinded to the sleep situation. However, importantly the investigators performing time trial tests were blinded to both the sleep situation and supplementation intervention employed during each trial. The study protocol was approved by Research Ethics Committee of National Taiwan Normal University (No. 201912HM115). Informed consent was obtained from all participants before taking part.

All trials commenced at the same time of day to eliminate the influence of circadian variation. Ambient (humidity and temperature) and environmental (brightness and noise) conditions in the sleep room and in the laboratory were monitored and carefully controlled throughout the trials. Temperature was between 24.1-25.6 °C in the Exercise Physiology laboratory and was between 25.2-26.1 °C in the sleeping room. Mean relative humidity was about 60% in both Exercise Physiology laboratory and sleeping room. Identical running shoes and clothes (individually) were used to minimize potential effects from external factors.

Preliminary Session

$\dot{V}O_{2max}$ measurement

Prior to the familiarization trial, a Bruce protocol test on a treadmill (h/p cosmos mercury 4.0, Germany) was employed for determining $\dot{V}O_{2max}$ (Trabulo et al., 1994). Expired gas samples were collected throughout the test using an automatic gas analyzer (V_{max}29, Sensor Medics the Corp., Yorba Linda, CA, USA). $\dot{V}O_{2max}$ was determined when the participants had obtained at least two of the following conditions: (1) respiratory exchange ratio (RER) was above 1.15; (2) $\dot{V}O_2$ increased less than 2 m/kg/min with the adding loads; (3) heart rate (HR) (Polar V800, Kempele, Finland) reached between ±10 (beats/min) of personal calculated maximal heart rate [HR_{max} = 207-(0.7 × age)] (Tanaka et al., 2001); and/or (4) volitional exhaustion was reached with a corresponding rating of perceived exertion (RPE) on the Borg scale of 19-20 (Borg, 1982).

Familiarization Trial

To familiarize with environment and experimental procedures, participants arrived at the same time of day as for the main trials. 8 hours of sleep was scheduled in the sleeping room to familiarize with sleep conditions, followed by a 4-km familiarization run the next morning.

Main trial days

Participants were required to document food and beverage intake, physical activities, and sleep diaries (bedtime, wake-up time and self-reported five-point general sleep quality) for three days prior to the first main trial, and they were then asked to replicate these behaviours prior to the following visits. Participants were also instructed to avoid all dietary sources of caffeine, alcohol, vigorous physical activity, and any nutritional supplements for 24 h preceding each main trial.

At approximately 9 p.m. participants reported to the Exercise Physiology laboratory and rested for 10 min before a venous blood sample was collected (Pre-Sleep). Uniform light snacks (crackers and a small cup cake, about 250 kcal) were then provided, and then no food was allowed until the next morning (only water was permitted). The NS trials included a bedtime from 10 p.m. and awakening at 6 a.m. the following morning, giving a total of 8 h permitted sleep duration. As for the PSD trials, participants remained awake until 3 a.m. and were awoken at 6 a.m. for a total of 3 h permitted sleep duration. During the waking hours, participants were allowed to engage in sedentary activities such as reading, listening to music, watching videos, or using a computer.

After baseline blood sampling at 6 a.m. (Pre-Sup), a standardized breakfast including toast and juice (284 kcal; carbohydrate: 71%, fat: 20%, protein: 9%) was consumed within 10 min, followed by caffeine (6 mg/kg body mass) (Southward et al., 2018) or placebo (sweetener, Zerose®, Cargill Inc., MN, USA, same weight as caffeine) supplementation in non-transparent capsules with 100 ml water. After the supplementation, participants rested quietly for 45 min and another blood sample was taken before (Pre-Ex) performing the 10-km running time trial (TT).

Prior to the 10-km TT, participants completed a 1 km warm up at a running speed equivalent to 60% of $\dot{V}O_{2max}$. The slope of treadmill was set to 1% inclination to reflect the energy demands of over ground outdoor running at a moderate-to-high intensity (Miller et al., 2019; Jones & Doust, 1996). During the 10-km TT, participants were instructed to complete the TT at the fastest speed and they were blinded to all performance and physiological related feedback, except for distance remaining which was displayed on the treadmill dashboard. Heart rate, RPE and respiratory gas exchange data were collected every 2 km during the 10-km TT. Within 3 min after completing the TT, a final blood sample was collected (Post-Ex).

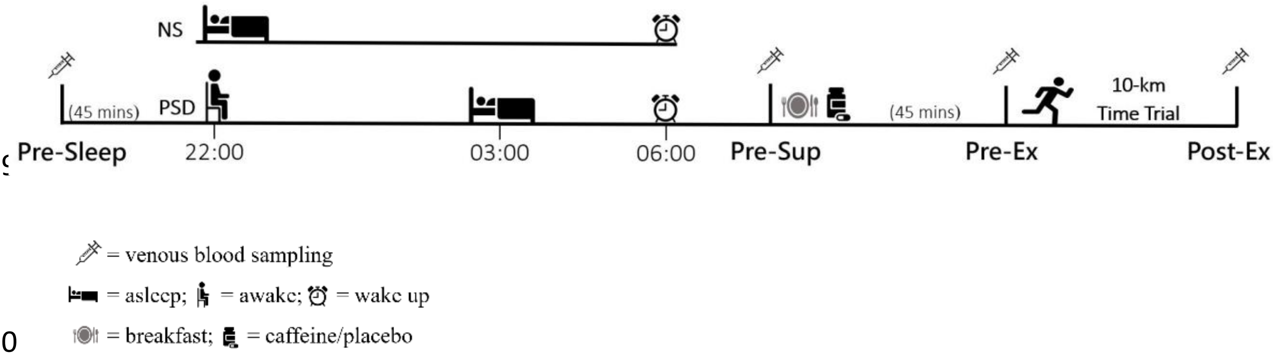

Fig 1. Experimental protocol. PSD: partial sleep deprivation; NS: normal sleep

Sleep measurements

In both preliminary session and main trials, participants wore an actigraphy device (wGT3X-BT, Actigraph, Pensacola, FL, USA) on the nondominant wrist to monitor sleep characteristics. The ActiLife software version 6.0 was used to analyze the data; all data were processed using 60-s epochs with default sampling frequency (i.e., 30 Hz). The Cole-Kripke algorithm was used (Cole et al., 1992) for sleep analysis (Quante et al., 2018), following recommended data collection and processing criteria (Migueles et al., 2017). The results of total time in bed, total sleep time and sleep efficiency (i.e., time in bed relative to actual sleep time) were obtained to compare sleep characteristics between trials.

Blood parameters analysis

Venous blood samples (~8 mL) were drawn from the antecubital vein (Pre-Sleep, Pre-Sup, Pre-Ex and Post-Ex), and 1 mL whole blood was immediately analyzed for complete blood count (CBC), glucose (Accu-Chek® Guide) and lactate (Lactate Pro 2). The remainder of the sample was then centrifuged at 3,000 g for 10 min at 4 °C after 15 min of clotting, and the separated serum was then stored at -80°C until subsequent analysis for FFA (#NEFA, Wako; Hitachi 7020) and glycerol (#10010755; Cayman Chemical) by Lezen Reference Laboratory (Taipei, Taiwan) following the manufacturer's instructions. Due to the possible changes in hydration status and fluid shifts during/after aerobic exercise, plasma volume changes were calculated, and the concentrations of biochemical variables were corrected following the formula from Dill and Costill (1974).

Statistical Analysis

All data are reported as the means ± standard deviation with 95% confidence interval (CI). Data were tested for normality of distribution using the Shapiro-Wilk test and Q-Q plot. Two-factor repeated-measures ANOVA was used to analyze the 10-km TT performance; the factors were supplementation (caffeine and placebo) and sleep situation (partial sleep deprivation and normal sleep). Three-factor repeated-measures ANOVA was used to determine the effects of supplementation (caffeine and placebo) and sleep situation (partial sleep deprivation and normal sleep) on the dependent variables at different running distances (2km, 4km, 6km, 8km and 10km). Three-factor repeated-measures ANOVA was also used to analyze time-dependent blood metabolite data; the factors were supplementation, sleep situation and time points (Pre-Sup, Pre-Ex and Post-Ex). The Greenhouse-Geisser correction was employed when violating the assumption of sphericity. When a main effect of supplementation, sleep situation, time or distance level, or interaction was detected, the *Bonferroni* procedure was applied for *post-hoc* comparisons. Two-way ANOVA were used to compare participants' sleep characteristics between trials. Partial eta squared (η_p^2) and Cohen's *d* were used as measures of effect size in the case of ANOVA and t-test analysis, respectively. Furthermore, the correlation between TT finishing time and all blood parameters were assessed with Pearson's correlation analysis. Analyses were performed with SPSS 20.0 and statistical significance was set at an α level of 0.05.

Results

Sleep characteristics

There was no interaction or main effect of supplementation for time in bed, total sleep time and sleep efficiency (all, $p > 0.05$). Time in bed was significantly lower in PSD than NS ($p < 0.001$, $\eta_p^2 = 1.000$). Total sleep time was significantly lower in PSD than NS ($p < 0.001$, $\eta_p^2 = 0.999$). Sleep efficiency was significantly higher in PSD than NS ($p = 0.012$, $\eta_p^2 = 0.523$) (Table 1).

Table 1. Sleep characteristics during main trials

	PSD-Caf	PSD-Pla	NS-Caf	NS-Pla
Time in Bed (min)	177 ± 2.8* (172 - 180)	176 ± 3.6* (169 - 179)	470 ± 5.2 (460 - 478)	472 ± 5.6 (458 - 476)
Total Sleep Time (min)	162 ± 3.7* (158 - 170)	159 ± 5.7* (155 - 174)	417 ± 7.0 (406 - 430)	415 ± 7.4 (405 - 428)
Sleep Efficiency (%)	91.5 ± 2.4* (88.3 - 95.5)	90.6 ± 3.0* (86.6 - 97.8)	88.6 ± 2.1 (84.9 - 91.5)	88.0 ± 1.7 (85.3 - 90.7)

Mean ± SD (minimal value - maximal value). PSD: partial sleep deprivation; NS: normal sleep; Caf: caffeine; Pla: placebo. *$p < 0.05$, main effect for sleep situation, PSD significantly different from NS.

10-km TT finishing time

There was a significant interaction for 10-km TT finishing time ($p = 0.020$, $\eta_p^2 = 0.470$, Fig 2). Partial sleep deprivation resulted in an increase in 10-km TT finishing time by 5% compared to normal sleep in the placebo trials (PSD-Pla: 51.9 ± 7.7 min, 95% CI [47.3, 56.5] vs. NS-Pla: 49.4 ± 6.9 min, 95% CI [45.4, 53.5]; $p = 0.001$, $d = 0.34$). Caffeine supplementation decreased TT time compared to placebo following both partial sleep deprivation (-7.7%; PSD-Caf: 47.9 ± 7.3 min, 95% CI [43.6, 52.1] vs PSD-Pla; $p = 0.007$, $d = 0.53$) and following normal sleep (-2.8%; NS-Caf: 48.0 ± 6.4 min, 95% CI [44.3, 51.7] vs. NS-Pla; $p = 0.049$, $d = 0.21$). 10-km TT finishing time in PSD-Caf did not differ from either NS-Pla or NS-Caf ($p = 0.185$, $d = 0.22$; $p = 0.891$, $d = 0.02$, respectively). Intra-individual (among trials) coefficient of variation was 4.55%, and the inter-individual (among participants) coefficient of variation was 14.37%.

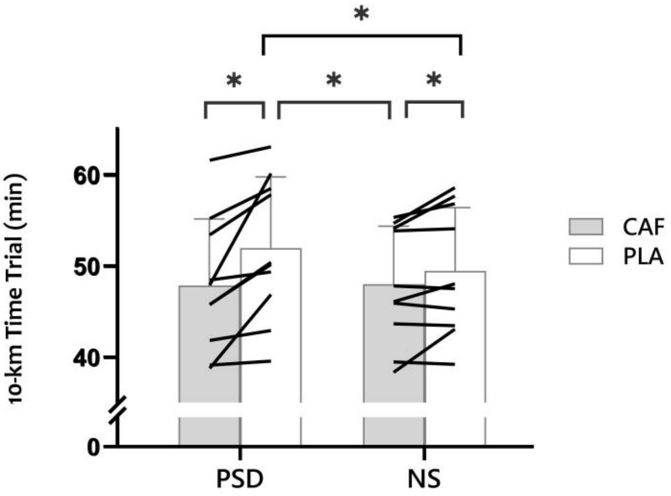

Fig 2. 10-km time trial running finishing time. PSD: partial sleep deprivation; NS: normal sleep; CAF: caffeine; PLA: placebo. *$p < 0.05$, significantly different between trials.

Physiological responses and RPE during TT

No significant three-way interactions or main effect of sleep situation were found for any measured cardiorespiratory parameter or for RPE (Fig 3.). A main effect of supplementation was detected for heart rate, Speed/RPE ratio and VE ($p = 0.001$, $\eta_p^2 = 0.710$; $p = 0.006$, $\eta_p^2 = 0.588$; $p = 0.004$, $\eta_p^2 = 0.613$; respectively), with all having greater response during Caf trial compared to Pla trial (heart rate: 4.9%; 172 ± 10 bpm, 95% CI [168, 176] vs. 164 ± 11 bpm, 95% CI [159, 169]; Speed/RPE ratio: 0.92 ± 0.13, 95% CI [0.86, 0.98] vs. 0.85 ± 0.10, 95% CI [0.81, 0.89]; VE: 12.1%; 95.6 ± 20.9 L/min, 95% CI [86.4, 104.8] vs. 85.3 ± 21.5 L/min, 95% CI [75.9, 94.7]). There were also main effects of distance (all, $p < 0.001$): heart rate, RPE, VE and percentage of maximal oxygen consumption gradually increased and peaked at 10km (Fig 3. A, B, E, F, respectively), whilst Speed/RPE ratio decreased and reached the lowest value at 10km (Fig 3. C).

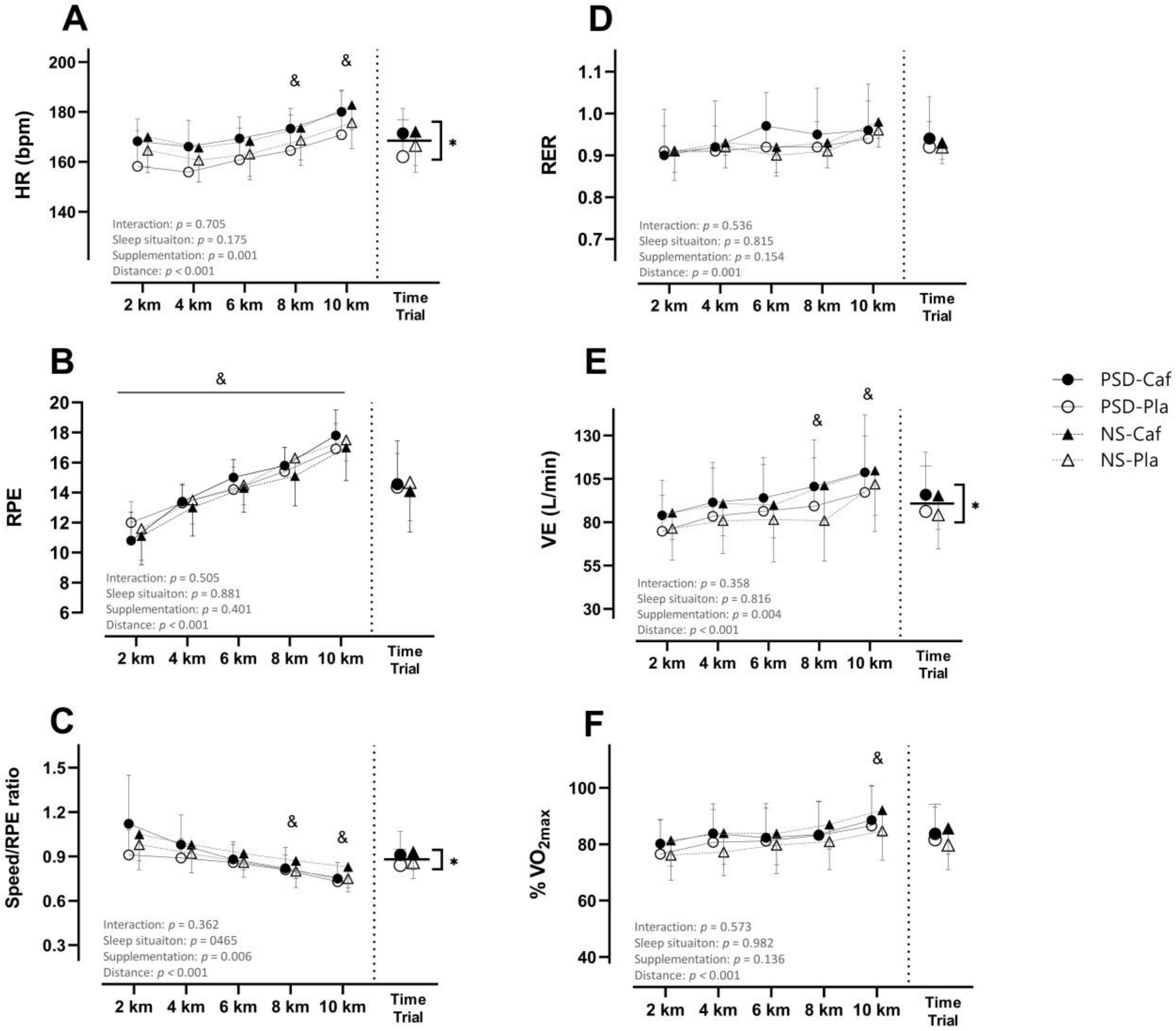

Fig 3. (A) HR, (B) RPE, (C) Speed/RPE ratio, (D) RER, (E) VE and (F) %$\dot{V}O_{2max}$ during time trial. PSD: partial sleep deprivation; NS: normal sleep; Caf: caffeine; Pla: placebo. Time Trial: average value throughout TT; Speed/RPE ratio = running speed (km/hr) divided by RPE; RER: respiratory exchange rate; VE: minute ventilation; %$\dot{V}O_{2max}$: percentage of maximal oxygen consumption. *$p < 0.05$, main effect, significantly different from placebo. $^{\&}p < 0.05$, main effect, significantly different from all other distances.

Blood parameters

No significant three-way interactions or main effect of sleep situation were detected for any measured blood metabolite (Fig 4.). A supplementation x exercise duration interaction was found for glucose, lactate and glycerol (p = 0.044, η_p^2 = 0.293; p = 0.005, η_p^2 = 0.450; p = 0.004, η_p^2 = 0.466; respectively). Post-hoc analysis revealed that glucose, lactate and glycerol levels were higher at Post-Ex in the Caf trial compared to Pla trial (Glucose: 8.7%; 132 ± 29 mg/dL, 95% CI [119, 144] vs. 121 ± 23 mg/dL, 95% CI

[111, 131]; $p = 0.015$, $d = 0.40$. Lactate: 20.8%; 7.9 ± 4.0 mmol/L, 95% CI [6.2, 9.7] vs. 6.6 ± 4.2 mmol/L, 95% CI [4.7, 8.4]; $p = 0.012$, $d = 0.33$. Glycerol: 14.5%; 4.4 ± 1.0 mg/L, 95% CI [4.0, 4.8] vs. 3.8 ± 1.1 mg/L, 95% CI [3.3, 4.3]; $p = 0.005$, $d = 0.53$). Glucose, lactate, FFA and glycerol all peaked at Post-Ex (Fig 4. A-D).

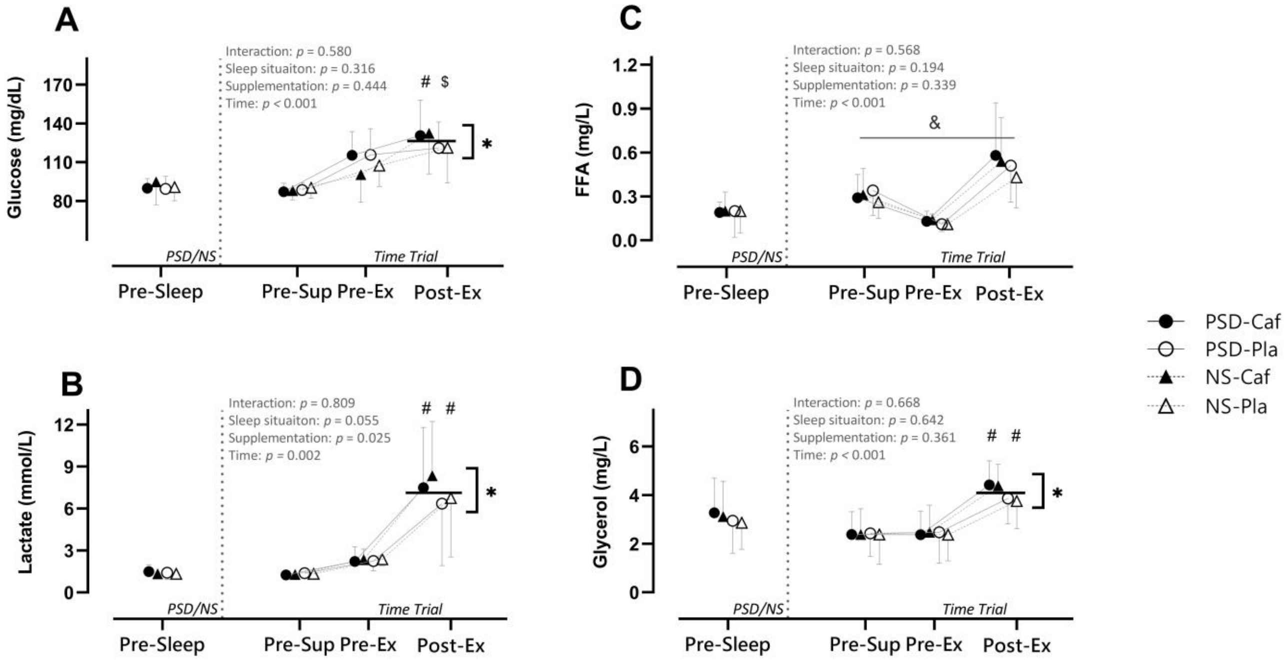

Fig 4. (A) glucose (B) lactate (C) free fatty acid (D) glycerol concentrations among trials. PSD: partial sleep deprivation; NS: normal sleep; Caf: caffeine; Pla: placebo. *$p < 0.05$, simple main effect, significantly different between caffeine and placebo. $^{\#}p < 0.05$, simple main effect, significantly different from other time points in the same Caf/Pla trial. $^{\$}p < 0.05$, simple main effect, significantly different from Pre-Sup in the same Caf/Pla trial. $^{\&}p < 0.05$, main effect, significantly different from other time points.

Discussion

The purpose of current study was to determine whether caffeine supplementation (6 mg/kg body mass) following a single night of PSD (i.e., 3 vs. 8 h sleep window) could mitigate the unfavourable effects of PSD on prolonged running performance. Our findings show that PSD decreased performance during 10-km TT running, while acute caffeine supplementation restored running performance following PSD, with performance even reaching comparable levels as observed following normal sleep with caffeine ingestion. These findings support the use of caffeine supplementation as an ergogenic strategy for athletes experiencing disturbed sleep duration/quality prior to competition.

In the present study, we observed that one night of PSD negatively impacts 10-km endurance running performance by approximately 5%. This aligns with previous findings, where performance was impaired by 4-10% in short, high-intensity time-to-exhaustion exercise tasks after a single night of PSD (Keramidas et al., 2018; Mejri et al., 2016; Mougin et al., 2001). Similarly, negative effects of PSD on self-paced exercise have been reported in 12-minute running time trials (Souissi et al., 2020) and 15-minute cycling time trials (Cullen et al., 2019). The duration of exercise was between 40 to 60 min in our study, providing evidence that the negative impact of PSD is observed not only during short and high-intensity intermittent exercise, but also during self-paced prolonged endurance exercise tasks requiring maximal effort.

Interestingly, physiological demand (i.e., HR), perception of effort (i.e., RPE) and markers of energy metabolism (such as RER, FFA, glucose and lactate) demonstrated similar responses between sleep situations during and following the 10-km TT. This is in line with previous studies also reporting no effect of PSD on RPE or heart rate during cycling time trials or time to exhaustion tests despite differences in performance (Chase et al., 2017; Cullen et al., 2019; Dean et al., 2023; Mamiya et al., 2021). During self-paced exercise (e.g. time trials), the remaining distance/duration becomes a crucial message to the brain to interpret RPE (Renfree et al., 2012; Tucker, 2009). As such, to achieve maximal attainable performance, experienced athletes adjust their pacing strategy accordingly to maintain reasonable RPE at intermediate points during the trial, thus possibly resulting in different performance but similar RPE under different experimental interventions (Renfree et al., 2012; Tucker, 2009). The impaired performance after PSD might be due to other potential mechanisms that we did not measure in this study, which could include physiological demands, such as impaired mitochondrial function (Liu et al, 2020), or lower pre-exercise glycogen availability (Skein et al., 2011), that have previously been observed following PSD. In addition, given that participants cannot be blinded to the sleep conditions due to the nature of the study design, there might be the potential psychological effects from a perceptual expectation of poorer running performance due to sleep deprivation. Sleep efficiency was slightly higher in PSD-Pla compared to NS-Pla, but self-paced maximal exercise performance was still compromised in the PSD-Pla trial, indicating the importance of sufficient sleep *duration* on maintaining next day exercise performance.

To the best of our knowledge, this is the first study to investigate the effects of caffeine ingestion after a single night of PSD on TT endurance performance. We found that caffeine supplementation completely offset (~7.7%) the decline in TT performance caused by PSD. Previously, McLellan et al. (2004) reported a 25% longer (~4.5 min) time to exhaustion during running at 85% maximal aerobic power with caffeine compared to placebo after a period of TSD. Similarly, Khcharem et al. (2021) found that running time to exhaustion was ~9% longer with caffeine intake after TSD, whilst Khcharem et al. (2022) also revealed that after caffeine ingestion, 8-km running TT performance was improved by 5.4% following TSD and by 2.4% following normal sleep, in comparison with placebo supplementation. Accordingly, the results of present study are in line with previous TSD studies and extend our understanding by showing that that caffeine supplementation also reinstates endurance exercise performance under PSD, the most common scenario that athletes might experience prior to the competition (Fullagar et al., 2015; Halson, 2014). The fact that performance after PSD with caffeine supplementation was restored to similar levels compared to following normal sleep further demonstrates the ergogenic potential of acute caffeine intake following insufficient sleep.

Studies showed that caffeine tended to promote glycogen catabolism and found that glycogen breakdown was enhanced in the fast-oxidative fibers exposed to caffeine (Chesley et al., 1998; Vergauwen et al., 1997). As such, caffeine-facilitated glycogenolysis may have contributed to both the improvement in performance and the greater post-exercise lactate concentrations with caffeine in our study. We also observed greater post-exercise blood glucose concentrations following caffeine supplementation and this may be explained by an increase in hepatic glucose output (Zaharieva & Riddell, 2013). In terms of energy metabolism, similar to the findings of Bell et al. (2002) during a 10-km run, we found that caffeine did not affect RER during time trial running. It is well-established via systematic review that pre-exercise intake of caffeine may effectively increase fat oxidation during aerobic exercise at submaximal intensity exercise (Collado-Mateo et al., 2020); when performing higher intensity exercise (e.g. during a time trial), then effects of caffeine on substrate metabolism (e.g. RER) might not be observed (Hulston & Jeukendrup, 2008). Therefore, though higher glycerol levels after exercise in our results likely reflects an increase in adipose tissue lipolysis facilitated by caffeine, the high exercise intensity (>80% $\dot{V}O_{2max}$ in all trials) meant that this did not translate into differences in substrate oxidation and RER. Finally, we observed that higher heart rate response corresponding to the greater performance during caffeine trials and this likely reflects the greater physiological demand as a result of the ergogenic effects of caffeine (Bridge & Jones, 2006; Graham, 2001; Hulston & Jeukendrup, 2008). On the other hand, despite similar absolute ratings of RPE between caffeine and placebo trials, when expressed relative to running speed (which was increased in the caffeine trials), we observed a higher Speed/RPE ratio with caffeine compared to placebo. This indicates that caffeine influenced performance via mechanisms that result in a reduced perception of effort for a given relative exercise intensity (Smirmaul et al., 2017).

Similar to previous study (Khcharem et al., 2022), we also found that time-trial endurance performance was increased in the NS-Caf trial compared to the NS-Pla trial. However, in contrast to previous studies investigating effects of caffeine supplementation following a night of NS and TSD on performance during an 8-km running TT (lasting 33-40 min) (Khcharem et al. 2022) or on run time to exhaustion at a fixed high intensity (Khcharem et al., 2021), we found no significant difference in exercise performance with caffeine supplementation following normal sleep (NS-Caf) and partial sleep deprivation (PSD-Caf). It is possible that the negative influences of PSD are less severe compared to TSD and therefore the ergogenic effects of acute caffeine supplementation were sufficient to restore exercise performance completely rather than just partially. In support of this, studies have revealed that the harmful effects of sleep loss on endurance exercise performance are more pronounced following more prolonged sleep deprivation (i.e., PSD vs. TSD) (Cullen et al., 2019; Cullen et al., 2020; Reynolds & Banks, 2010). Another potential explanation is that the deleterious effect of sleep loss on endurance performance is dependent on exercise duration, i.e., exercise lasting longer is more negatively affected by sleep deprivation than shorter exercise bouts (Lopes et al., 2023). Additionally, there might be a relationship between the duration of exercise and caffeine supplementation, such that the beneficial effects of caffeine become more pronounced with longer exercise durations (Shen et al., 2019). Taken together, the smaller magnitude of sleep loss (via PSD), and the longer exercise duration (10-km running TT) combined with greater ergogenic effects of caffeine supplementation, are likely reasons why we did not observe a difference in 10-km TT performance between the PSD-Caf trial and the NS-Caf trial.

While this study was highly controlled, certain limitations warrant consideration. First, considering it is not possible to blind participants in the sleep conditions, there is the potential for psychological effects (i.e. expectancy of poorer running performance) to occur. However, the finding that caffeine completely restored performance compared to placebo (and supplementation was blinded) suggests that this is unlikely to be the main driver of the impairment in performance following sleep loss. Secondly, the PSD trials in the present study deprived sleep during the early phase of the night. Previous research has shown that sleep partially deprived in either the early or late phase of the night might lead to different endurance exercise performance effects (Mejri et al., 2016; Mougin et al. 2001). Thus, the effects of caffeine might be different under different PSD conditions. We also did not perform muscle biopsies, so whether the impact of sleep loss on muscle glycogen availability (Skein et al., 2011) played a mechanistic role in this study is unknown and should be examined in the future. Finally, due to logistical constraints, this study exclusively included male participants. As the impact of sleep loss on psychophysiological responses and exercise performance may vary between sexes (Ołpińska-Lischka et al., 2021; Romdhani, Hammouda et al., 2021), future research should investigate the influence of biological sex on caffeine's efficacy under both early and late-night sleep deprivation.

Conclusion

We conclude that 10-km time trial running performance is impaired by a single night of partial sleep deprivation, and that acute caffeine supplementation (6 mg/kg body mass) approximately 45 min prior to exercise can counteract the negative impact of sleep deprivation on 10-km running performance.

Conflicts of interest

The authors declare that they have no conflicts of interest with the contents of this article.

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Authors contributions

Y.-S. TSAI and H.-S. WANG conceived and designed the research; Y.-S. TSAI performed the experiments, data collection and data analysis; T.-T. CHEN, Y.-C. CHAN and C.-C. HUANG assisted with experiments; T.-F. LAI and Y. LIAO supplied sleep-measuring equipment and assisted with data collection; Y.-S. TSAI, Y.-C. CHEN, R. S. METCALFE and H.-S. WANG interpreted results of experiments then drafted, edited and revised the manuscript. All authors approved the final version of manuscript.

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