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The effects of caffeine ingestion on the reaction time and short-term maximal performance after 36 h of sleep deprivation

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HIGHLIGHTS

- Physical and cognitive performances were reduced after 36 h of total sleep deprivation.
- Physical and cognitive performances were improved after the ingestion of 5 mg·kg^{-1} of caffeine.
- 5 mg·kg^{-1} of caffeine could counteract the negative effect of 36 h of TSD on physical and cognitive performances.

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ABSTRACT

The aim of the present study was to investigate the effects of caffeine ingestion on cognitive and physical performances after 36 h of sleep deprivation. In randomized order, thirteen healthy male physical education students (age: 21.1 ± 1.1 years, body mass: 77.1 ± 7.2 kg, height: 1.77 ± 0.06 m) completed four test sessions at 18:00 h: after placebo or 5 mg·kg^{-1} of caffeine ingestion during a baseline night (RN) (bed time: from 22:30 h to 07:00 h) or a night of 36 h of sleep deprivation (TSD). During each test session, participants performed the squat jump (SJ), the reaction time, and the 30-s Wingate tests (i.e., for the measurement of the peak (PP) and mean (MP) powers and the fatigue index (FI)). The results showed that PP and MP decreased and FI increased during the TSD compared to RN in the placebo condition ($p < 0.001$). The caffeine ingestion improved PP after TSD compared to RN ($p < 0.001$). SJ decreased significantly after the TSD compared to RN after both placebo and caffeine ingestions ($p < 0.001$). However, SJ increased significantly after caffeine ingestion during RN and TSD ($p < 0.001$). The reaction time increased significantly after TSD compared to RN ($p < 0.001$). However, the reaction time decreased significantly after the caffeine ingestion only during the TSD ($p < 0.001$). Therefore, caffeine is an effective strategy to counteract the effect of 36 h of sleep loss on physical and cognitive performances.

1. Introduction

Sleep is commonly viewed as a restorative process that influences the homeostatic regulation of the autonomic, neuroendocrine, and immune systems [1]. The sleep–wake cycle is of fundamental importance to human circadian rhythms, and its disruption can have consequences on both mental and physical performances in various settings [2]. Likewise, sleep disruption decreases metabolic activities in prefrontal regions of the brain governing executive functions [1].

Sleep loss, either total (TSD) or partial, is associated with increased sleepiness and decrements in neurobehavioral [3,4] and physical performances [5–8]. Likewise, sleep loss is a common source of stress both in athletes and non-athletes [1]. TSD has been shown to negatively affect many physiological, cognitive, and behavioral measures within the body [9]. In this context, previous studies showed that TSD or partial sleep loss may decreases short-term maximal performance [5,7].

On the other hand, caffeine may enhance short-term performance and alertness after sleep loss [10]. In this context, Wesensten et al. [11] showed that 600 mg of caffeine ingestion improve cognitive performance after 85 h of sleep deprivation. The caffeine's ergogenic effect could be explained by: (i) a reduction of the sensation of fatigue induced by exercise [12], (ii) an enhancement of the excitation–contraction coupling [13] (iii), and a stimulation of the central nervous system (CNS) [14]. However, the literature presents inconclusive results concerning the effect of caffeine ingestion on short-duration high-intensity maximal exercise. In this context, previous studies failed to observe substantial performance increments following caffeine ingestion [15]. However,

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other studies revealed that caffeine ingestion lead to a significant increase in short-term performance [16–18]. Likewise, previous studies showed that caffeine is an effective strategy to maintain cognitive performance after an overnight period of sleep loss [18].

There are many situations in which sleep is disturbed prior to an athletic event (e.g., jetlag or anxiety). Indeed, sleep loss, either total or partial, may be experienced by athletes who have to get up early in the morning to travel to a competition or who cannot fall asleep because of the psychological stress of a major event [1]. Therefore, the purpose of the present study was to investigate the relative efficacy of caffeine ingestion for restoring the negative effect of 36 h of TSD both physical and cognitive performances.

2. Methods

2.1. Participants

Thirteen healthy male physical education students (age: 21.1 ± 1.1 years; body mass: 77.1 ± 7.2 kg; height: 1.77 ± 0.06 m) volunteered to participate in the present study. They had exactly the same time schedule at the university from sunrise to sunset under the control of the experimental team. Participants had taken part in various recreational low-intensity physical activities such us walking, jogging, or aerobics in our university. During the experimental period, medications, which are expected to affect physical performance, were prohibited. Before participation, all the participants were informed about the experimental procedures, the possible risks, and discomforts associated with the study and signed a written informed consent. The study was conducted according to the Declaration of Helsinki and the protocol was fully approved by the University Ethics Committee. Participants were selected according to their usual consumption of caffeine and on the basis of their answers to the Horne and Ösberg Self-Assessment Questionnaire [18] (i.e., to have a group without "extreme type" (i.e., participants were selected as "neither type")). This second criterion resulted in a sample of participants who shared the same timing in terms of rising times (06:30 ± 00:30 h) and bedtimes (23:00 ± 00:30 h). Participants reported no sleep disorder, are non-smokers, and do not consume caffeine or any alcoholic beverages.

2.2. Experimental design

After four consecutive nights of sleep in the laboratory (sleep adaptation: between 22:30 and 07:00 h), in a randomized order, participants performed four test sessions: after placebo or 5 $mg \cdot kg^{-1}$ of caffeine ingestion during a baseline night (RN) or a night of 36 h of TSD. During the RN, participants were synchronized with a nocturnal sleep from 22:30 to 07:00 h. During the TSD, they were not allowed to sleep and were kept awake by passive means such us watching TV.

During each test session, after 10 min of rest in a sitting position, participants ingested the caffeine or the placebo dose; then they remain in a sitting position for 60 min. After the 60 min, they performed the reaction time, the squat jump (SJ), and the Wingate tests at 18:00 h with 15 min of recovery between the SJ and the Wingate test. To give the desired amount (5 $mg \cdot kg^{-1}$) of caffeine, the quantity was measured using an electronic weighing (BOECOTE; accuracy ± 1 mg).

Oral temperature was measured during the two sleep conditions at 08:00 and 18:00 h using a digital clinical thermometer (Omron, Paris, France; accuracy ± 0.1 °C) inserted sublingually for at least 3 min. All test sessions took place in similar conditions of temperature and relative humidity (27–28 °C and 63–66%, respectively). Instructions about sleep and diet were given to the participants prior to the commencement of the study [19,20]. They were prescribed with standard isocaloric meals to consume, with breakfast at 07:30 h, lunch at 12:00 h, and dinner at 20:00 h [21]. Only water was allowed ad libitum between meals. They were requested to maintain their habitual physical activity throughout the experimental period and to avoid strenuous activity before each test session [22]. The overall daily energy intake goal was set at 10.5 MJ (2500 kcal) per capita/day. Values of the daily nutritional intake were calculated using the software NUTRISOFT-BILNUT® (Ver. 4, Paris, France).

2.3. Simple and choice reaction time

The reaction time was used as an index of individuals' motor performance [23]. The assessment of simple reaction time was performed by the slant of the software "React" [23]. The test was consisted to answer rapidly to a visual stimulus while pushing on a key of a microcomputer [23]. During the test, participants were instructed to press on a key of the microcomputer when a visual stimulus appeared.

2.4. Squat jump test

The subjects were asked to perform a SJ without any load on an infrared jump system (Optojump, Italy) interfaced with a microcomputer [24]. The subjects stood between two 1-m infrared sensor bars to perform the SJ. In the SJ, the subjects lower themselves into a squat position and after a brief pause, jump upward as quickly and as high as possible. No downward motion is allowed immediately before jumping upward.

2.5. Wingate test

The Wingate test was conducted on a friction-loaded cycle ergometer interfaced with a microcomputer [19,25]. The seat height and handlebars were adjusted appropriately for each subject. The Wingate test consisted of a 30 s maximal sprint against a constant resistance related to body mass (0.087 $kg \cdot kg^{-1}$ body mass) [22]. The Wingate test began from a rolling start, at 60 rpm against minimal resistance. When a constant pedal rate of 60 rpm was achieved, a countdown of "3–2–1–go!" was given, and the test resistance was applied. Subjects were verbally encouraged throughout the test to avoid pacing and to sustain a maximal effort throughout the test. Every second, power output was calculated by the computer and stored. The highest power output over 1 s (PP) and the mean power (MP), corresponding to the ratio between total work done and time to do it (i.e., 30 s), were recorded. The fatigue index (FI) (i.e., the percentage of power output decrement) was calculated by the difference between PP and the lowest power (Plow) divided by PP:

$$\text{Fatigue Index (FI)} = (\text{PP} - \text{Plow})/\text{PP}.$$

2.6. Profile of mood states (POMS)

The POMS consists of 65 adjectival items (e.g., tense, scared) developed to measure 7 aspects of mood (anxiety/tension, depression/dejection, anger/hostility, confusion/bewilderment, vigor/activity, fatigue/inertia, and friendship). As described by McNair et al. [26] the responses to each item range from 0 to 4, with higher scores indicating more negative mood (0 indicates "not at all," and 4 indicates "extremely").

2.7. Statistical analysis

Statistical tests were processed using STATISTICA Software (StatSoft, France). Data were reported as mean ± SD. Once the assumption of normality was confirmed using the Shapiro–Wilk W-test, parametric tests were performed. Data were analyzed using a two-way ANOVA analysis (2 [Sleep] × 2 [Caffeine]) with repeated measures on both factors. Core temperature values were analyzed using a three-way ANOVA analysis (2 [Time-of-day] × 2 [Sleep] × 2 [Caffeine]) with repeated measures. When the ANOVA indicated significant Sleep or Caffeine effects or

significant interaction Sleep × Caffeine, significant differences between means were tested using the Tukey post-hoc test. The level of statistical significance was set at $p < 0.05$.

3. Results

3.1. Temperature

A significant main effect for Time-of-day ($F = 27.82$, $p < 0.001$) was observed demonstrating that the oral temperature improved significantly from morning to afternoon ($p < 0.001$, Fig. 1). However, there were no significant main Sleep ($F = 0.14$, $p > 0.05$) and Caffeine effects ($F = 0.52$, $p > 0.05$).

3.2. Profile of mood state

POMS results after the RN and the TSD with placebo or caffeine ingestions are presented in Table 1. The statistical analysis revealed that depression, confusion, fatigue, and anxiety increased and vigor decreased after the TSD in comparison with the RN ($p < 0.001$). However, depression, confusion, fatigue, and anxiety decreased and vigor increased after the ingestion of caffeine in comparison with the placebo during TSD ($p < 0.001$).

3.3. Wingate test

Mechanical parameters recorded during the Wingate test during the RN and the TSD after placebo and caffeine ingestions are presented in Table 2.

3.3.1. Peak power (PP)

There was a significant main effect for Sleep ($F = 31.79$; $p < 0.001$) indicating that PP was significantly lower during TSD in comparison with RN ($12 \pm 0.3\%$; $p < 0.001$) after the ingestion of the placebo. Likewise, a significant effect for Caffeine ($F = 33.34$; $p < 0.001$) and a significant interaction Sleep × Caffeine ($F = 31.79$; $p < 0.001$) were observed indicating that PP increased significantly during the TSD after caffeine in comparison with placebo ingestion ($p < 0.001$). However, during RN, no significant difference was observed between placebo and caffeine ingestions.

3.3.2. Mean power (MP)

There was a significant main Sleep effect ($F = 32.82$; $p < 0.001$) indicating that MP was significantly lower during TSD in comparison with RN after the placebo ingestion ($p < 0.001$). Likewise, a significant Caffeine effect ($F = 13.68$; $p < 0.001$) and a significant Sleep × Caffeine interaction ($F = 13.81$; $p < 0.001$) were observed. During the TSD, a significant increase of MP was observed after the caffeine ingestion in comparison with the placebo ingestion. However, no significant difference was observed between the placebo and the caffeine ingestions during RN.

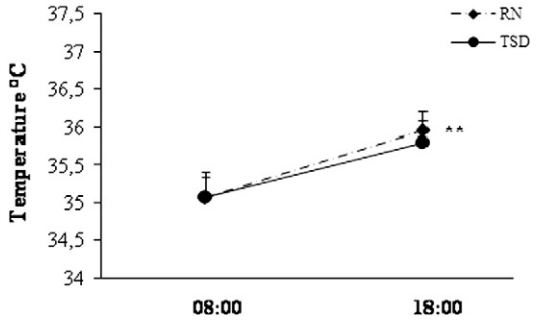

Fig. 1. The diurnal variation of oral temperature in the reference night (RN) and the partial sleep deprivation (TSD) conditions. Values are mean ± SD. **Significant differences between the time points.

Table 1
Mean (±SD) values for POMS subscale recorded after the reference (RN) and sleep deprivation (TSD) nights with placebo and caffeine ingestions.

	RN		TSD	
	Placebo	Caffeine	Placebo	Caffeine
Depression	14 ± 4.12	13.85 ± 4.04	29.61 ± 2.96*	15.15 ± 4.26£
Confusion	11.23 ± 2.62	12.54 ± 2.44	16.92 ± 2.6*	11.69 ± 2.01£
Fatigue	10.46 ± 2.5	9.61 ± 3.2	18.85 ± 3.1*	12.46 ± 2.07£
Vigor	15.08 ± 2.69	16.69 ± 2.17	10.15 ± 2.64*	15.77 ± 2.59£
Anxiety	11.08 ± 2.78	13.23 ± 3.29	15.54 ± 3.8*	12.23 ± 2.77£

* Significant difference in comparison with RN.
£ Significant difference in comparison with placebo at the same sleep condition.

was observed between the placebo and the caffeine ingestions during RN.

3.3.3. Fatigue index

For the FI, the main Sleep ($F = 30.75$; $p < 0.001$) and Caffeine ($F = 30.70$; $p < 0.001$) effects and the interaction Sleep × Caffeine ($F = 63.93$; $p < 0.001$) were significant. The post hoc showed that the FI increased significantly after TSD in comparison with RN during the placebo condition. Likewise, the FI increased after the caffeine in comparison with the placebo ingestion during TSD.

3.4. Jump performance (SJ)

There was a significant main Sleep effect ($F = 30.75$; $p < 0.001$) indicating that SJ was significantly lower during TSD in comparison with RN during the placebo condition ($p < 0.001$, Fig. 2). Likewise, a significant Caffeine effect ($F = 30.70$; $p < 0.001$) and a significant interaction Sleep × Caffeine ($F = 63.93$; $p < 0.001$) were observed. During the TSD, a significant increase of SJ was observed after the caffeine in comparison with the placebo ingestions (Fig. 2). However, no significant difference was observed between the placebo and the caffeine ingestions during RN.

3.5. Simple and choice reaction time tasks

The ANOVA revealed a significant main Sleep ($F = 67.49$ and $F = 35.09$ for the simple and the choice reaction time respectively; $p < 0.001$) and Caffeine ($F = 18.27$ and $F = 16.44$ for the simple and the choice reaction time respectively; $p < 0.05$). Likewise, the Sleep × Caffeine interaction was significant for both the simple and the choice reaction time ($F = 30.25$ and $F = 35.06$ for the simple and the choice reaction time respectively; $p < 0.001$). The post hoc analysis revealed a significant increase for both simple and choice reaction time after TSD in comparison with RN ($p < 0.001$) during the placebo test session. Likewise, a significant decrease for both simple and choice reaction time ($p < 0.001$) was observed after caffeine ingestion in comparison with placebo during TSD test session (Fig. 3).

Table 2
Mean (±SD) values for peak (PP) and mean (PM) powers and the fatigue index (IF) recorded the reference (RN) and sleep deprivation (TSD) nights with placebo and caffeine ingestions.

	RN		TSD	
	Placebo	Caffeine	Placebo	Caffeine
PP ($W \cdot kg^{-1}$)	11.2 ± 0.1	11.3 ± 0.9	9.8 ± 0.5*	11.1 ± 0.8£
MP ($W \cdot kg^{-1}$)	8.9 ± 0.7	8.4 ± 0.7	7.4 ± 0.9*	8.2 ± 0.2£
FI (%)	51.83 ± 12.65	52.11 ± 10.99	54.08 ± 13.1*	51.14 ± 11.13£

* Significant difference in comparison with RN.
£ Significant difference in comparison with placebo at the same sleep condition.

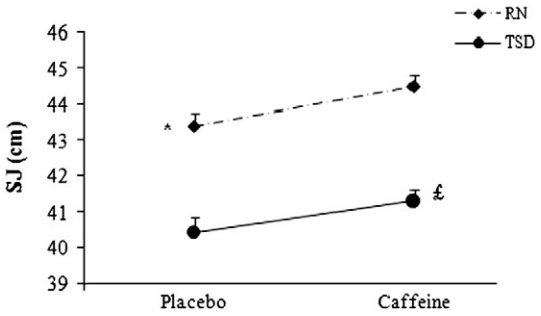

Fig. 2. Squat jump (SJ) performances (mean ± SD) recorded after the reference night (RN) and the total sleep deprivation (TSD) conditions with placebo and caffeine ingestions. *Significant differences in comparison with RN. £Significant differences in comparison with placebo at the same sleep condition.

4. Discussion

The aim of the present study was to determine the effect of caffeine ingestion on simple and choice reaction time and short-term maximal performances measured in the afternoon after 36 h of TSD. Our results showed that simple and choice reaction time, PP, MP, and FI during the Wingate test, and the SJ performance were adversely affected by 36 h of TSD. However, the caffeine ingestion may improve both cognitive and physical performances after TSD.

Consistent with previous reports [27,28], our results showed a significant decrease in simple reaction time 60 min after the caffeine ingestion. To explain this effect, previous studies have suggested that the stimulating effect of caffeine on the CNS through antagonism of adenosine [34] may cause an increase in adrenaline and dopamine that could improve the subjects' attention. Concerning the short-term maximal performances, during RN, the present study's results showed that PP, MP, and FI were unaffected by caffeine ingestion in the afternoon. These results are in agreement with previous works that showed no significant variation in physical performance in trained [29] and untrained subjects [30] after caffeine ingestion. The mechanisms by which caffeine would have an effect on the energy system contribution during short-term performance remain unclear. Several mechanisms of caffeine demonstrated in vitro studies that used higher levels of this substance include a caffeine-induced Ca^{2+} release from the sarcoplasmic reticulum, a direct effect on myofibrils, an inhibition of phosphodiesterase, and alterations in neuromuscular transmission [31]. Therefore, a higher dose of caffeine may be needed to elicit an ergogenic effect on physical performance. Moreover, the absence of a significant effect of caffeine on physical performance observed in the present study could be explained by the level of trainability of the subjects. In this context, Davis and Green [32] reported that the ergogenic effect of caffeine on anaerobic exercises occurs especially in trained subjects. Previous reports revealed an ergogenic effect of caffeine ingestion in trained subjects using the same doses as in the present study [33]. However, the present study's results are at odds with those of Kang et al. [34] that showed a significant increase in PP and MP during a Wingate test after the ingestion of 5 $mg \cdot kg^{-1}$ of caffeine. More recently, Lara et al. [35] showed that energy drink with 3 $mg \cdot kg^{-1}$ of caffeine might be an effective ergogenic aid to improve physical performance in female soccer players. Abian-Vicen et al. [36] showed that the intake of a caffeine-containing energy drink increased jump performance; but didn't affect basketball shooting precision but. The reason for these discrepancies may be due to the utilization of various exercise modes, frequencies, intensities and durations, as well as different evaluation procedures [36].

The present study results showed that TSD affect the cognitive performance (i.e., an increase in the reaction time). It has been reported that both TSD and partial sleep deprivation impair the ability to maintain wakefulness, increase the subjective sleepiness, reduce the motivation, and, perhaps most critically, degrade the cognitive performances [1]. In line with the present study's findings, Philip et al. [37] showed that 30 to 64 h of sleep deprivation lead to an alteration of reaction time. Likewise, previous studies showed that the disruption of sleep affect cognitive performances (i.e., decrease of alertness and increase of reaction time) [38–40]. The decrease of attention and accuracy after TSD previously reported [38–40] could explain in part our findings. Likewise, in agreement with previous reports, our results showed that PP and MP, attention, and alertness were affected by TSD [41,42]. Likewise, these results are consistent with those of Souissi et al. [43] and Abedelmalek et al. [7] who showed that one night of partial sleep deprivation affect short-term maximal performance (i.e., PP and MP) in the afternoon of the following day. The decreased performances in the afternoon after the sleep deprivation condition could be due to a higher level of fatigue at this time of day after longer time of being awake [8]. Indeed, since fatigue has been shown to be higher in the afternoon than the morning [44–47], this fatigue could be increased after the sleep loss condition leading to a reduced capacity to produce or to maintain performances.

On the others hand, the present study's results demonstrated that the ingestion of caffeine could improve cognitive and physical performances after a TSD night. These finding are in line with Reyner and Horne [48] who reported an increase in physical performance after sleep deprivation following the administration of caffeine. In the same context, Smith [49] showed that caffeine ingestion counteracts the decrease of performance, reaction time, and alertness typically observed after sleep deprivation. In the same way, Bonnet and Arand [50] showed that caffeine ingestion increase alertness and mental performance after sleep deprivation. As a result, caffeine is routinely used to minimize performance and alertness impairment after either TSD or partial sleep

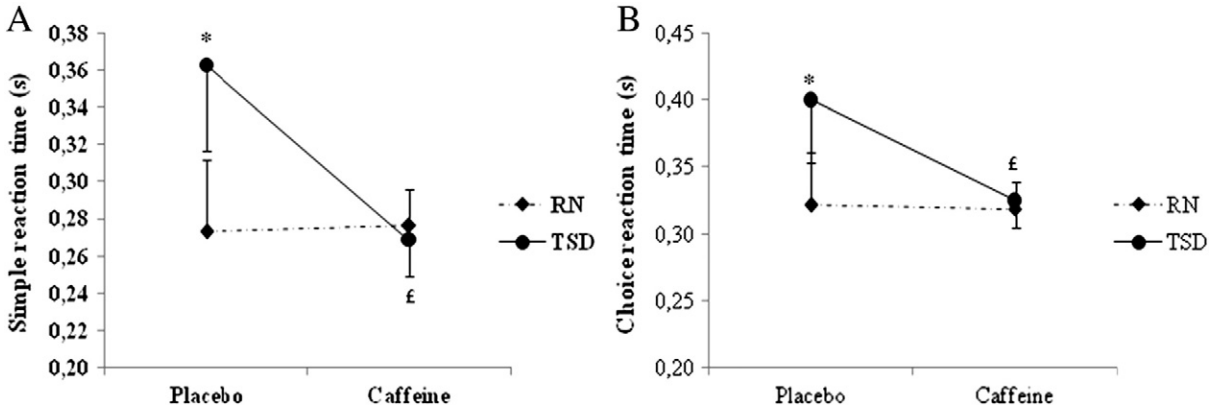

Fig. 3. Performances (mean ± SD) during the simple [A] and the choice [B] reaction time recorded at the reference night (RN) and the total sleep deprivation (TSD) conditions with placebo and caffeine ingestions. *Significant differences in comparison with RN. £Significant differences in comparison with placebo at the same sleep condition.

deprivation [48]. It is possible that the antagonism of adenosine receptors is the responsible of the improvement of performance [51] after the ingestion of caffeine. In this context, Porkka-Heiskanen [52] has identified adenosine as a moderator of prolonged wakefulness.

Our findings show that, consequently to TSD, negative mood has been raised. These results agree with Caldwell et al. [45], Kautz [38], and Penetar et al. [39] who reported that sleep deprivation affects mood states assessed with the standard POMS scale and the visual analogical Scale. Furthermore, the present study's findings showed that caffeine ingestion counteract the effect of TSD on mood states. In agreement, Kautz [38] showed that caffeine administration may positively affect mood states evaluated by the POMS scale after a sleep deprivation condition. Additionally, using visual analogical scales, Kautz [38] showed that caffeine consumption leads to the decrease of sleepiness and the increase of awareness, the concentration capabilities, the self confidence, the anxiety, the agitation and the neurosis. Lieberman et al. [53] showed that caffeine administration reduce the negative mood states induced by the sleep deprivation. Likewise, Penetar et al. [39] showed that the administration of caffeine (i.e., 150, 300, and 600 mg) decrease the fatigue and increased the vigor after a night of sleep deprivation. Urry and Landolt [54] showed that caffeine cannot substitute for sleep. The available evidence suggests that adenosinergic mechanisms contribute to waking-induced impairments of intentional processes.

4.1. Study limitations

The present study has some limitations. First, we didn't record the quality of sleep during both RN and TSD conditions. Moreover, participants weren't well-trained and this could limit the ergogenic effect of caffeine on physical performances.

5. Conclusion

The present study's results revealed an increase in simple and choice reaction time after TSD. Likewise, SJ, PP, MP, and FI during the Wingate test were affected by 36 h of sleep deprivation. In addition, caffeine ingestion improves cognitive and physical performances (i.e., increased PP and MP during the Wingate test, vertical jump height during the SJ test, and reaction time) after TSD. Therefore, caffeine could counteract the negative effect of sleep loss on both cognitive and physical performances. However, experimental studies should be conducted to determine the effect of caffeine ingestion on the diurnal variation of short-term maximal performance after sleep loss.

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