ORIGINAL ARTICLE

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Caffeine Decreases Neuromuscular Fatigue in the Lumbar Muscles: A Randomized Blind Study

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SUMMARY

Background. The purpose was to evaluate a possible ergogenic effect on postural muscles, still unknown through Biering-Sørensen's lumbar extension test.

Methods. A double-blind, controlled placebo, crossover study. 51 healthy, physically inactive male subjects (18-25 years) with average body mass (BMI 18.5-24.9 kg/m^2) were recruited. The subjects received oral caffeine (6 mg/kg) and saline (0.3%) in two cross-evaluations separated by one week.

Results. The primary outcome was the time in the Biering-Sørensen test after 1 hour of treatment. The secondary results were peak lumbar extension force, rating of perceived exertion, electromyography (EMG's) median frequency and muscle recruitment of *multifidus spinae* and transversalis/interne oblique muscles, and cardiovascular variables (heart rate and blood pressure). 27 subjects were blindly treated with caffeine and saline. Caffeine was ergogenic during the Biering-Sørensen test. It increased lumbar extension time ($F = 0.34$, $p < 0.05$), but not peak force. The perception of effort decreased with caffeine ($F = 0.37$, $p < 0.05$). Caffeine increased muscle stimulation frequency ($p < 0.05$) and recruitment ($t_{23} = 0.49$, $p < 0.05$) of *multifidus spinae*. In the transversalis/interne oblique muscles, caffeine increased the median frequency ($t_{23} = 0.13$, $P<0.05$) and the distribution of higher frequencies ($p < 0.05$). Caffeine also increased muscle recruitment in the transversalis/interne oblique muscles ($t_{23} = 0.94$, $p < 0.05$). Tachycardia and increased blood pressure at the lumbar test were higher in the caffeine condition ($p < 0.05$).

Conclusions. Caffeine is ergogenic for postural muscles. Decreased rated perceived effort (RPE) and improved muscle activity suggest central mechanisms of caffeine.

Study registration. The trial was prospectively registered (trial RBR- blinded) in the Brazilian Clinical Trials Register (http://www.ensaiosclinicos.gov.br/).

KEY WORDS

Caffeine; electromyography; fatigue; lumbar spine; Biering-Sørensen test.

INTRODUCTION

Caffeine (1,3,7-trimethylxantine) is one of the most used ergogenic substances by physical activity practitioners and athletes (1). Caffeine is hydrophilic, fast absorbed, and distributed to all body tissues (1, 2). Caffeine is a non-selective competitive adenosine receptor antagonist that increases endurance (1, 2) and resistance (2). The most used caffeine dose is 6 mg/kg, 1 hour before physical activity (1). Ergogenic amounts of caffeine have minor adverse effects, such as angiogenesis, insomnia, and gastrointestinal discomfort (3).

Adenosine has an important inhibitory modulator effect in the central nervous system (CNS) (4). Caffeine decreases the rate of perceived exertion (2), pain (2, 5), and central and mental fatigue during exercise (6, 7). Exercise-induced fatigue increases brain motor area potentiation after transcranial magnetic stimulation (8) and modifies spinal excitability and cortical silence in fatigued muscles (9). Caffeine also improves exercise performance (10), cognitive and executive functions (5), and vigor (9) in exercising subjects. Caffeine does not show an ergogenic effect in a mouse lacking neuronal $A_{2A}R$ (4) and in rats treated with 5'-N-ethylcarboxamidoadenosine (NECA), an adenosine analog (11).

We conducted this study to evaluate the effects of caffeine on endurance (Biering-Sørensen test) and electrical activity (electromyography – EMG) of core muscles, a well-controlled experiment in our laboratory (9). There is ergogenic evidence for upper and lower limb muscles (12), but not for core muscles. Recently, these muscles are part of the sports training routine, as they are essential for performance enhancement (13) and injury prevention (14) in athletes. The possible ergogenic effects of caffeine on core muscles can contribute to better periodization used in the muscle regeneration and treatment of mesocycles for performance, tapering, and competitions.

METHODS

Study design and subjects

We conducted a randomized, double-blind, placebo-control, crossover study, where both examiner and subjects were unaware of ingested substances. Recruitment was carried out through social networks in the community within the micro-region of Araranguá (south latitude 28º56'05 "and west longitude 49º29'09"), southern Brazil. Subjects also collaborated in the recruitment of others, according to the public involvement statement. We called the volunteer to clarify the study design and the inclusion and exclusion criteria. The study followed the Consolidated Standards of Reporting Trials (CONSORT).

We recruited healthy, physically inactive male subjects with average body mass (BMI 18.5-24.9 kg/m^2) aged 18-25 years. Participants were only male due to hormonal factors presented by women that interfere with the caffeine effect. The screening included a semi-structured interview about medical regarding medical history, coffee consumption (15), and the International Physical Activity Questionnaire (IPAQ) – short form – Brazilian version (16, 17). Exclusion criteria were: smoking habit; hypertension; diabetes; lumbar and orthopedic diseases; neurological, cardiac and gastric diseases; continued use of antidepressants and medications or supplements containing caffeine; and engaging in physical activities more than two times/wk.

The study respected the declaration of Helsinki. Moreover, the Brazilian National Health Council also requires compliance with ethical principles of Resolution No. 466 of December 12, 2012. All subjects signed a consent form before inclusion in the study. The trial was approved by the Brazilian Ethics Committee (http://plataformabrasil.saude.gov.br) with file number CAEE blinded. The Universal Trial Number (UTN) is blinded.

Randomization and blinding

All eligible subjects were evaluated when treated with vehicle or caffeine, blinded to the researchers through tubes reporting substance 1 or A, respectively. Subjects were randomized in a 1:1. One independent researcher (MCS) carried out simple randomization with a coin toss to start the test with a blinded substance. Other researchers blindly (LC, TN and MG) performed the treatment and assessments. The statistician was unaware of treatment allocation before completion of data collection and data cleaning.

Intervention

Caffeine and sodium chloride (NaCl) with 98% purity was purchased from Sigma-Aldrich® (Brazil). The subjects' body mass (BMI) was measured, a concentrated caffeine solution (3 mg/mL) was prepared freshly, and the individual volume adjusted by multiplying body mass by two. The vehicle solution consisted of 0.3% NaCl in water. Subjects drank identical volumes of caffeine (6 mg/kg) or vehicle, 1-hour before the Biering-Sørensen test, the time necessary to reach plasma peaks. A researcher (LC) followed the subjects for 24 hours to monitor the development of adverse effects of caffeine that they could report.

To perform the Sørensen test (18), the subject was positioned on the stretcher in ventral decubitus with arms extended along the body and head in a neutral position. The Sørensen test consists of using the subjects' own body weight to create postural resistance. The isometric resistance time, defined as the time to maintain the proposed posture until exhaus-

tion, was recorded. To control the horizontality of the trunk during the test, we used a stadiometer as a static reference. When contact was lost, the examiner asked the participant to correct the position. The individual's exhaustion was defined by the impossibility of maintaining the standardized posture for more than three seconds, that is, the loss of contact with the stadiometer, by voluntary lowering of the trunk or when the maximum time of 240 seconds was reached.

Measurements

The analyzes were performed in the Evaluation and Rehabilitation of the Locomotor System Lab (Laboratório de Avaliação e Reabilitação do Aparelho Locomotor – LARAL, UFSC, Araranguá (SC), Brazil). Participants were advised to continue habitual eating throughout the study. Physical activity, alcohol consumption, and caffeine (food and beverages) were avoided in the 48 hours before the tests. Each test was performed on two separate days, with 1-week intervals, during the morning or afternoon, according to the subjects' agenda availability. The researchers sought all subjects for evaluations in the laboratory.

After 1 hour of oral treatment, the endurance of *multifidus spinae* was assessed by peak extensor force (14), and the Biering-Sørensen test (19). At the same time, electromyography (EMG) assessed the median frequency and muscle recruitment (Root mean Square of EMG signal – RMS) of *multifidus spinae* and transversalis/interne oblique muscles (19), Borg Rating of Perceived Exertion – RPE – Brazilian version (30-s intervals) (20) were performed. Heart Rate (HR) was assessed during rest and at 15-s intervals during the test.

At the end of the test, we evaluated lactate capillary blood (earlobe) levels (21) and collected antecubital venous blood samples for plasma separation and storage (-80 °C). Plasma caffeine concentrations were assessed using High-Performance Liquid Chromatography – HPLC (22). HPLC system consisted in a quaternary pump with a degasser, and it was coupled to an autosampler and DAD-UV detector (Agilent 1200 series) (Agilent Technologies Mexico, S. de R.L. de C.V.). Separation was performed on a reverse-phase column Zorbax® SB-Aq narrow bore RR (2.1 × 100 mm, 3.5 µm) (Agilent Technologies). The column oven was maintained at 40 °C, while the autosampler was set at room temperature. Fifteen microliters of processed sample was injected into the HPLC system.

Outcomes and sample size

We designed this trial to detect changes in the Biering-Sørensen test time (primary outcome) and peak force (secondary outcome), RPE (secondary outcome), and EMG's median frequency and muscle recruitment (secondary outcome). The study was carried out with 27 subjects to have 85% power with alpha = 0.05.

Statistics

A blinded researcher (ASAJr) performed the statistical analysis according to an intention-to-treat principle. The results were described on mean ± SD. We assessed the normal distribution of results before statistical comparisons. Paired Student's t-test analyzed Biering-Sørensen time and peak force, blood lactate levels, RPE and HR (area under the curve – AUC), and final 10% of EMG median frequency. The final 10% of the RMS was assessed through Wilcoxon matched-pairs signed-rank test.

This analysis was performed using GraphPad Prism (v. 5.0, GraphPad Software). The STATISTICA (v. 8.0, StatSoft, Inc.) was used to compare ANOVA of EMG median frequency and RMS, chi-square test of EMG median frequencies, and Mann Whitney test of HR. P-value < 0.05 was considered significant. Effect sizes (Cohen's d) were calculated for between-group changes in mean differences for all outcome measures, where a Cohen's d = 0.2 represents a "small" effect size, 0.5 represents a "medium" effect size and 0.8 a "large" effect size (7). Cohen's test was used for repeated measurements, defined as 0.02 small, 0.13 medium, and 0.26 large (7).

Public involvement statement

There were no funds or time allocated for public involvement, so we were unable to involve subjects. We have invited subjects to help us develop our dissemination strategy.

RESULTS

Subjects and plasma caffeine levels

Fifty-one subjects were recruited between 7/15/2018 and 12/15/2018, interrupted on 5/11/2018 when the sample size was completed. **Figure 1** shows the protocol per the Consolidated Reporting Trials Standards (23). There were 27 undergraduate students, single, with ideal body mass index, irregularly active and light (25.9%), moderate (48.1%), and high (25.9%) coffee consumers. **Table I** shows the subjects' demographic and baseline data.

Subjects consumed 404.9 ± 55.3 mg of caffeine (95%CI 382-427.7) (**table II**) before the test, rising to plasma 0.086 ± 0.04 nmol/L (95%CI 0.06-0.11) after 1 hour. Even after 48 h of withdrawal of caffeinated foods and beverages, plasma levels were 0.011 ± 0.03 nmol/L (95%CI 0-0.04) when treated with a vehicle. Subjects for 24 hours and reported no adverse effects such as tachycardia, angiogenesis, insomnia or gastrointestinal discomfort.

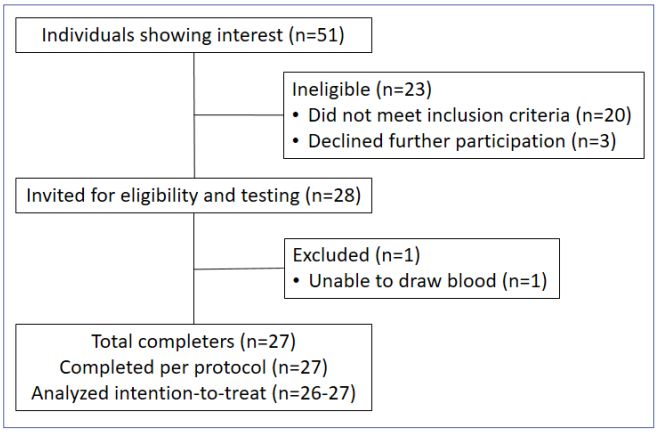

Figure 1. CONSORT flow diagram. CONSORT, Consolidated Standards of Reporting Trials.

Table I. Baseline participant characteristics.

Variables	
Demographics	
Male, number, %	Male, 27 (100%)
Age, years	20.3 (1.9)
Education, %	Undergraduate (100%)
Job status, %	Student (100%)
Civil status, %	Single (100%)
Anthropometrics	
Body mass, kg	68.8 (11.4)
Height, m	1.77 (0.09)
Body mass index, kg/m^2	21.9 (2.3)
Resting physiology	
Systolic blood pressure, mmHg	123.5 (11)
Diastolic blood pressure, mmHg	75.1 (10.9)
Heart rate, bpm	78 (11.3)
International Physical Activity Questionnaire (IPAQ) *(Craig et al. 2003; Ipaq 2005; S. Matsudo, T.Araújo, V. Matsudo, D. Andrade, E. Andrade, L.C. Oliveira 2012)*	
Irregularly active A, number (%)	14 (51.9%)
Irregularly active B, number (%)	13 (48.1%)
Coffee consumption *(Mitchell et al. 2014)*	
Low, number (%)	7 (25.9%)
Moderate, number (%)	13 (48.1%)
High, number (%)	7 (25.9%)

Caffeine increases fatigue resistance of spinal muscles

Primary outcome

Caffeine had a small effect on increasing 13.8 ± 6.3% Biering-Sørensen endurance, but not peak force **figure 1A** and **table II**).

Secondary outcomes

Caffeine induced a moderate effect size on increased blood lactate production (**table II**), a biomarker of exercise intensity. Biering-Sørensen test rated perceived effort (RPE) intensities classified as moderate to intense ($F_{6,258} = 64$, $\eta^2 = 0.6$, b = 1.0, $p < 0.05$, **figure 2B**). Moreover, caffeine had moderate effect on decreased RPE (**table II**). HR increased during the test ($F_{13,637} = 6$, $\eta^2 = 0.11$,$\beta = 1.0$, $p < 0.05$, **figure 2C**), and caffeine demonstrated a great positive chronotropic effect during the physical test ($p < 0.05$, **figure 2D**, **table II**).

Caffeine stimulates spimusclecles activity

Figure 3A illustrates the raw EMG values for the *multifidus spinae*, where we see the endurance effect of caffeine. The median frequency of *multifidus spinae* decreased over Biering-Sørensen test ($F_{29,1131} = 127$, $\eta^2 = 0.76$, $p < 0.05$, **figure 3B**), without caffeine effect during ($F_{1,39} = 0.2$, $\beta^2 = 0.005$, $p < 0.05$, **figure 3B**) and at the end of the test ($t_{20} = 0.021$, $p = 0.9$, **figure 3C**). However, the distribution of median frequencies revealed higher frequencies on caffeine-treated subjects ($\chi^2_1 = 144$, $p < 0.05$, **figure 3D**). Caffeine still had

Table II. Effects of caffeine on primary and secondary outcomes.

Variable		Mean (SD)		95% Confidence Interval		Effect size	Power (β)	p
		Washout	Caffeine	Placebo	Caffeine			
Biering-Sørensen	time (sec)	143.9 (42)	159 (46.3)	126.1-161.6	139.5-178.6	0.34	0.75	$t_{23} = 1.7$, $p = 0.047$*
	peak force (kgf)	44.8 (16.7)	46.1 (17.8)	37.4-52.3	38.2 - 54	0.07	0.12	$t_{23} = 0.4$, $p = 0.32$
Rated perceived effort, Borg (AUC)		660 (339.4)	538 (315.3)	509.5-810.5	398.2-677.7	0.37	0.81	$t_{21} = 2.1$, $p = 0.045$*
Blood lactate, mmol/L		5.6 (2.4)	7.1 (3.2)	4.6-6.6	5.7-8.4	0.53	0.97	$t_{23} = 1.7$, $p = 0.045$*
Hear rate, bpm (AUC)		12.5 (4.8)	16.3 (6.2)	10.5-14.5	13.7-18.9	0.69	0.99	$t_{23} = 3.4$, $p = 0.002$*

AUC: Area Under Curve; *$p < 0.05$ paired Student t-test.

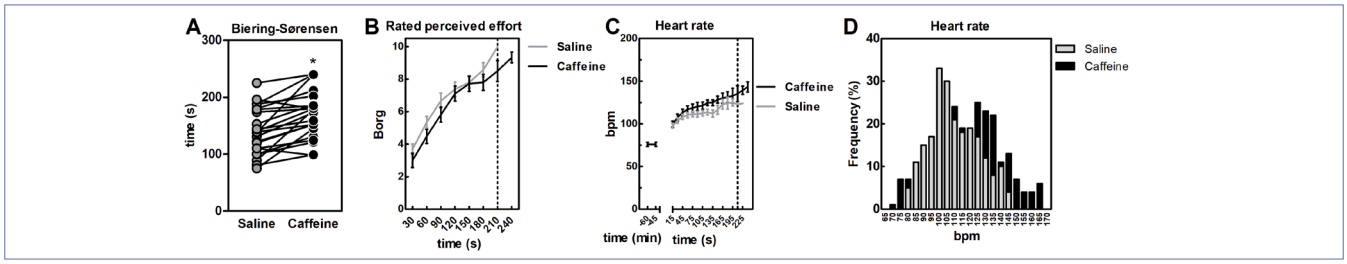

Figure 2. Effects of caffeine on endurance **(A)**, rated perceived effort **(B)** and heart rate **(B-C)** during the Biering-Sørensen test.

Panels A-C are described with mean ± SD, D as relative frequency. n = 25-27 for 14 independent experiments. *p < 0.05 (Paired Student t-test).

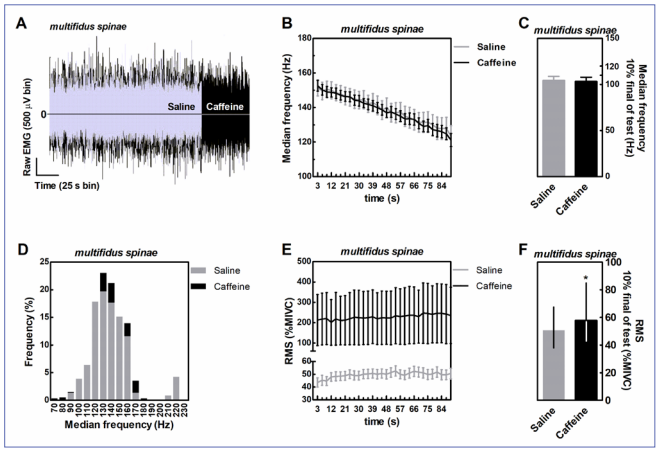

Figure 3. Effects of caffeine on the raw EMG signal **(A)**, median frequency **(A-C)** and recruitment **(D-E)** of the *multifidus spinae* muscle during the first 90 seconds and 10% final of the Biering-Sørensen test.

Panels A, C and D are described with mean ± SD, B as relative frequency, and E median + interquartile range. n = 25-27 for 14 independent experiments. *p < 0.05 *vs* saline (Wilcoxon matched-pairs signed-rank test).

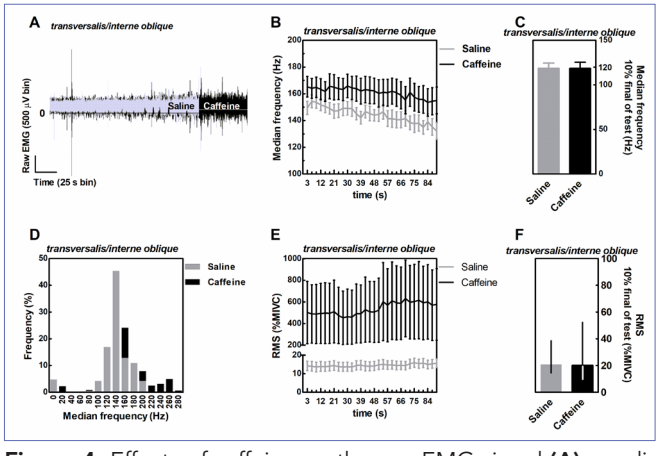

Figure 4. Effects of caffeine on the raw EMG signal **(A),** median frequency **(A-C)** and recruitment **(D-E)** of the transversalis/internal oblique muscles during the first 90 seconds and 10% final of the Biering-Sørensen test.

Panels A, C and D are described with mean ± SD, B as relative frequency, and E median + interquartile range. n = 25-27 for 14 independent experiments.

a significant effect size ($h^2 = 0.49$) on increasing multifidus spinae RMS values throughout the Biering-Sørensen test, from the beginning ($F_{1,540} = 53$, $p < 0.05$, **figure 3E**) to the end of the test ($Z = 1.77$, $p < 0.05$, **figure 3F**).

The raw EMG data already demonstrate the ergogenic effects transversalis/interne oblique when increasing the time in the Biering-Sørensen test (**figure 4A**). Caffeine ergogenic effect size was medium ($\eta^2 = 0.13$) on increased median frequency ($F_{29,107}$ transversalis, **figure 4B**) of in transversalis/interne oblique. The distribution of median frequencies shifted to the right for higher frequencies ($\chi^2_1 = 2{,}900$, $p < 0.05$, **figure 4D**) transversalis/interne oblique. The effect size of caffeine-increased muscle recruitment was also large ($\eta^2 = 0.94$, $F_{1,540} = 87$, $p < 0.05$, **figure 4E**). These ergogenic effects disappeared in the final 10% of the Biering-Sørensen test (**figure 4C**,**F**).

DISCUSSION

We first demonstrated the ergogenic effects of caffeine on postural muscles - *multifidus spinae* and transversalis/interne oblique - components of core muscles. Core muscles are essential for physical performance and injury prevention, the goals of sports medicine. All participants in our study were men, which we can consider a limitation of our study. The increase in Biering-Sørensen endurance (or fatigue resistance) had a small effect size, as well established for other muscle groups and activities (24, 25). The Biering-Sørensen test is the most robust predictor for low back pain and trunk stability (26), with implications from health to elite sport. Strengthening core muscles increases Biering-Sørensen test performance and decreases low back pain (14, 26). There is not much evidence in the sports sciences. Water polo, soccer, and rowing athletes performed well in the Biering-Sørensen test (163.6 ± 50.7 sec) (19). Muscle activity of abdominal and spine extensors of long-distance runners and triathletes was higher in the Biering-Sørensen test, which contributes to running performance, such as more excellent absorption by the trunk muscles of disrupting torques generated by the lower limbs (19).

The Biering-Sørensen test performance influences athletes' motivation and self-efficacy (27). Athletes are more resistant to high blood lactate concentrations, pain, and fatigue (2, 3). Psychological factors are associated with this performance, such as improved motivation and experience . Moderate doses of caffeine (3-6 mg/kg) decrease RPE in athletes (2, 3). We observed this same effect of caffeine (6 mg/kg) in this spine muscle stress test. Effort perception is a brain function; neurophysiology involves central fatigue and diminished cortical arousal (28), increased cortical activity during movement in the primary motor and supplementary movement areas (29), and increased activation of the temporal and insular cortex (30). This evidence retrieves the CNS's role in the development of central fatigue (10, 14, 30-32). Here, the effect of caffeine on RPE and EMG reinforces this idea.

Caffeine modified the EMG response in the Biering-Sørensen test. EMG activation is the preliminary condition for any force development. Here, caffeine induced a substantial increase in muscle recruitment, a moderate increase in the high frequency of stimulation, and decreased fatigue (EMG/force ratio) of *multifidus spinae* and transversal/internal oblique muscles. There is no evidence about the effects of caffeine on these muscles, but other muscle groups have a similar EMG response to caffeine. Caffeine (5 mg/kg) improved performance in the cycling time trial and increased Maximal voluntary strength (MVC), power output, and muscle recruitment of *vastus lateralis and medialis* (12). Caffeine (6 mg/kg) increased the elbow's maximum flexor torque, muscle recruitment, and fiber conduction velocity (12, 33). Muscle strength was higher in electrically stimulated *adductor pollicis* and ankle dorsiflexors muscles of caffeine-supplemented subjects (500 mg and 6 mg/kg). Caffeine decreased low-frequency fatigue (20, 30, and 40 Hz stimulation) in these muscles, reducing expected muscle force (fatigue) due to an impairment of excitation-contraction coupling. Caffeine did not modify muscle strength at higher stimulation frequencies (> 40 Hz) (33), such as those achieved voluntarily in this study.

This evidence reinforces the role of caffeine in central fatigue, which also modifies cortical silent period (CSP) and spinal excitability (34). CSP refers to an interruption of voluntary contraction by electrical or magnetic stimulation of the motor cortex. A transcranial magnetic stimulation (TMS) fatigued abductor digit minimi muscle and caffeine reduced CSP (8). Caffeine also increased spinal excitability by increasing the slope of the H-reflex recruitment curve normalized to the M wave (H_{slp}/M_{slp}), which provides an alternative measure of monosynaptic reflex gain. These are evidence of the neurophysiological components of the ergogenic effects of caffeine. Moreover, our experimental design removed caffeine for 48 hours from subjects, moderate coffee consumers, which may have enhanced our ergogenic results (4, 27, 34).

However, the mechanisms of action of caffeine's ergogenic effects are intriguing. For a long time, literature insisted on the mobilization of intracellular Ca^{+2} and inhibition of phosphodiesterase (1). These biological effects are achieved *in vitro* (in millimolar concentrations, 10^{-3}), but toxic and lethal *in vivo* (4). Recent studies discuss CNS's antagonism of the adenosinergic system as a possible candidate mechanism for caffeine (34). Electrophysiological studies support the positive effect of caffeine on vigilance, attention, speed of reaction, information processing, and arousal (34).

Caffeine acts on the CNS decrease the perception of effort and modifies the motor drive. A1 receptor (A_1R) and $A_{2A}R$ are primarily responsible for the central effects of adenosine (28), also the main target of nontoxic psychostimulant doses of caffeine in micromolar levels (10^{-6}) (29). Generally, blocking presynaptic A_1R increases the probability of neurotransmitter release, including dopamine and serotonin, whereas blocking presynaptic $A_{2A}R$ decreases neurotransmitter release (28). A1 blockade could reinforce Meeusen's untested hypothesis about central fatigue (35), an exercise-induced increase in extracellular serotonin stimulating sleep, lethargy, and drowsiness, and loss of motivation. Adenosine is a neuromodulator responsible for these latter effects (30). Caffeine decreases CSP in fatigued muscles (34). Experimental manipulation of $GABA_B$ inhibitory presynaptic receptors modulate CSP. In this context, caffeine might interfere with GABAergic neurotransmission differently (36).

Moreover, basal forebrain cholinergic neurons are under tonic inhibitory control of endogenous adenosine. $A_{2A}R$ is highly expressed in the basal ganglia, emphasizing the ventral striatopallidal GABA pathway, where they form functional heteromeric receptor complexes ($A_{2A}R$-D_2) with inhibitory presynaptic dopaminergic D_2-like receptors. D2-like receptors decrease adenylyl cyclase activity through $G_{i/o}$ proteins (37). Caffeine and selective $A_{2A}R$ antagonists produce psychostimulant effects by competing with adenosine for its binding to the A2AR and exerting a negative allosteric modulation within the $A_{2A}R$ of $A_{2A}R$-D_2 heterotetramer (37). Under normal conditions, employing the negative heteromeric allosteric modulation, endogenous adenosine tonically inhibits psychomotor activation mediated by tonic activation of D_2R by endogenous dopamine. Caffeine, through negative allosteric modulation, counteracts the effect of adenosine and produces psychomotor activation (38). Caffeine (a non-selective A_1 and $A_{2A}R$ antagonist) and SCH 58261 (potent and selective $A_{2A}R$ antagonist) are psychostimulants (4). Davis (11) demonstrated that NECA (A_1R and $A_{2A}R$ agonist) inhibits the ergogenic effects of caffeine in rats. We demonstrated that SCH 58261 is ergo-

genic in mice and that the ergogenic effect of caffeine disappears in mice knocked out to neuronal $A_{2A}R$ in the CNS (4). In the present study, we demonstrated that caffeine decreases the perception of effort and increases muscle excitation in humans, increasing the excitation-contraction efficiency of spine muscles. This evidence jointly reinforces the role of caffeine in mitigating central fatigue during exercise.

Caffeine supplementation did not impact the subjects' general health. Monitoring the subjects for 24 hours did not reveal any known adverse effects of caffeine, such as tachycardia, angiogenesis, insomnia, gastrointestinal discomfort, or other symptoms. Dietary consumption and experimental treatment offered the subjects approximately 400 mg of caffeine daily, a dose considered safe and free of adverse effects in moderate consumers (3).

We have identified two limitations of this study. We did not perform a blinding assessment and identified residual caffeine in the plasma of some subjects. This last point shows flaws in the withdrawal protocol. However, blood caffeine concentrations are not a standard measure in most studies focusing on ergogenics. At the same time, blood caffeine is a strong study point. This study is a simple and reproducible experimental design. Increasing the sample size to include patients and athletes of different sexes and ages is necessary for the generalization and applicability of these results.

CONCLUSIONS

In summary, our results show that caffeine is ergogenic for postural muscles. As an ergogenic resource, caffeine may benefit patients with back pain. The results also reinforce the ergogenic role in sports. Decreased RPE and improved muscle activity suggest central mechanisms of caffeine. That is, caffeine attenuates central fatigue during acute exercise.

FUNDINGS

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DATA AVAILABILITY

Data are available under reasonable request to the corresponding author.

CONTRIBUTIONS

ASA Jr: funding, conceptualization, data analysis, manuscript writing. LC, TN, MG, MCS, DLS: study execution. LC, HY, AES manuscript reviewing.

CONFLICT OF INTERESTS

The authors declare that they have no conflict of interests.

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