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JAINARA LIMA MENEZES

**IMPACTO DA CAFEÍNA SOBRE INDICADORES
NEUROMUSCULARES, ESTRESSE OXIDATIVO E DANO
MUSCULAR NO *POWERLIFTING* PARALÍMPICO**

**SÃO CRISTÓVÃO
JULHO/ 2026**

JAINARA LIMA MENEZES IMPACTO DA CAFEÍNA SOBRE INDICADORES NEUROMUSCULARES,
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Tese apresentada ao Programa de Pós-Graduação em Ciências Fisiológicas da Universidade Federal de Sergipe como requisito à obtenção do grau de Doutora em Ciências Fisiológicas.

Orientador: Prof. Dr. Felipe José Aidar Martins

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“Consagre ao Senhor tudo o que você faz, e os seus planos serão bem-sucedidos.”

— Provérbios 16:3

RESUMO

Impacto da cafeína sobre indicadores neuromusculares, estresse oxidativo e dano muscular no powerlifting paralímpico, Jainara Lima Menezes, São Cristóvão, 2026.

Introdução: O exercício de força melhora o desempenho, mas gera fadiga e dano muscular. A suplementação de cafeína (CA) é usada para mitigar esses efeitos, porém os parâmetros neuromusculares, dano muscular e o estresse oxidativo ainda precisam ser esclarecidos, principalmente no powerlifting paralímpico. **Objetivo:** Avaliar os efeitos da suplementação de CA no desempenho de força (estática e dinâmica), no estresse oxidativo e dano muscular no powerlifting paralímpico. **Métodos:** Este estudo investigou os efeitos agudos da suplementação com CA (9 mg/kg) sobre indicadores de força estática (Taxa de desenvolvimento de força (RFD), Impulso, Pico de torque, força isométrica máxima, força de pico em 1 segundo e variabilidade da força) e força dinâmica (Velocidade Propulsiva Média (VPM), Velocidade Máxima (Vmáx) e Potência foram avaliadas a 45% de 1RM antes, após, 24 e 48h de uma sessão de treinamento e também a 80% de 1RM, sendo 5x5. Marcadores de estresse oxidativo e de atividade antioxidante enzimática (TBARS/MDA, capacidade antioxidante total, grupos sulfidril e ácido úrico) e marcadores de dano muscular (termografia, CK (creatina quinase), LDH (lactato desidrogenase) e creatinina), em 14 atletas paralímpicos de levantamento de peso do sexo masculino. O protocolo incluiu duas condições (CA e PLA (placebo)), 5x5 a 80% de 1RM no supino adaptado. **Resultados:** A força estática CA aumentou significativamente a RFD, o impulso, pico de torque, além da força isométrica máxima, melhorou a força de pico em 1 segundo e reduziu a variabilidade, sugerindo um recrutamento aprimorado de unidades motoras. Em relação a força dinâmica não foram observadas diferenças significativas com 45% de 1RM. No entanto, com 80% de 1RM, o grupo CA demonstrou melhora significativa em comparação com o grupo PL durante a Série 1 e a Série 5. No entanto, a CA também levou a um aumento do estresse oxidativo, evidenciado por níveis elevados de TBARS, capacidade antioxidante reduzida, diminuição dos grupos sulfidril e concentrações mais altas de ácido úrico após o exercício. A termografia mostrou um aumento em todos os músculos avaliados com CA em comparação com a condição PLA. Para CK, LDH e creatinina, não houve diferenças significativas. **Conclusão:** A CA demonstra propriedades ergogênicas promissoras, contribuindo para o aumento da força muscular e permitindo que atletas mantenham a intensidade do treinamento mesmo com cargas mais elevadas. Além disso, não parece aumentar o dano muscular, sugerindo um perfil de uso seguro nesse aspecto. No entanto, embora melhore agudamente o desempenho neuromuscular, a CA não

reduz — e pode até exacerbar — o estresse oxidativo induzido pelo exercício, o que indica que sua utilização em esportes de força deve ser feita com cautela.

Descritores: Treinamentos de força; Powerlifting paralímpico; Suplementação Nutricional; Cafeína; Dano muscular; Estresse oxidativo.

ABSTRACT

Impact of caffeine on neuromuscular indicators, oxidative stress and muscle damage in Paralympic powerlifting, Jainara Lima Menezes, São Cristóvão, 2026.

Introduction: Resistance exercise improves performance but generates fatigue and muscle damage. Caffeine (CA) supplementation is used to mitigate these effects; however, neuromuscular parameters, muscle damage, and oxidative stress still need to be clarified, especially in paralympic powerlifting.

Objective: To evaluate the effects of CA supplementation on strength performance (static and dynamic), oxidative stress, and muscle damage in paralympic powerlifting.

Methods: This study investigated the acute effects of CA supplementation (9 mg/kg) on indicators of static strength (Rate of Force Development (RFD), Impulse, Peak torque, maximum isometric strength, peak strength at 1 second, and force variability) and dynamic strength (Mean Propulsive Velocity (MPV), Maximum Velocity (Vmax), and Power) were evaluated at 45% of 1RM before, after, 24h, and 48h of a training session, and also at 80% of 1RM, performing 5x5). Markers of oxidative stress and enzymatic antioxidant activity (TBARS/MDA, total antioxidant capacity, sulfhydryl groups, and uric acid) and markers of muscle damage (thermography, CK (creatine kinase), LDH (lactate dehydrogenase), and creatinine) were assessed in 14 male Paralympic powerlifting athletes. The protocol included two conditions (CA and PLA (placebo)), 5x5 at 80% of 1RM in the bench press. **Results:** Regarding static strength, CA significantly increased RFD, impulse, peak torque, and maximum isometric strength, while improving peak strength at 1 second and reducing variability, suggesting enhanced motor unit recruitment. Regarding dynamic strength, no significant differences were observed at 45% of 1RM. However, at 80% of 1RM, the CA group demonstrated significant improvement compared to the PLA group during Set 1 and Set 5. Nonetheless, CA also led to an increase in oxidative stress, as evidenced by elevated TBARS levels, reduced antioxidant capacity, decreased sulfhydryl groups, and higher uric acid concentrations post-exercise. Thermography showed an increase in all evaluated muscles with CA compared to the PLA condition. For CK, LDH, and creatinine, there were no significant differences. **Conclusion:** CA demonstrates promising ergogenic properties, contributing to increased muscle strength and allowing athletes to maintain training intensity even with higher loads. Furthermore, it does not appear to increase muscle damage, suggesting a safe usage profile in this regard. However, while it acutely improves neuromuscular performance, CA does not reduce—and

may even exacerbate—exercise-induced oxidative stress, indicating that its use in strength sports should be approached with caution.

Descriptors: Strength training; Paralympic powerlifting; Nutritional supplementation; Caffeine; Muscle damage; Oxidative stress.

RESUMO VOLTADO PARA SOCIEDADE

Impacto da cafeína sobre indicadores neuromusculares, estresse oxidativo e dano muscular no powerlifting paralímpico, Jainara Lima Menezes, São Cristóvão, 2026.

Introdução: O exercício físico é importante para melhorar o desempenho e a saúde, principalmente em esportes de força, como o levantamento de peso. Porém, treinos intensos também podem causar cansaço no corpo e maior “desgaste interno”. Por isso, muitos atletas usam suplementos, como a cafeína, para tentar melhorar o rendimento. Mesmo assim, ainda não se sabe completamente como a cafeína afeta a força, a recuperação e o estado do corpo após o treino, especialmente em atletas paralímpicos de levantamento de peso. **Objetivo:** O estudo teve como objetivo entender se o uso de cafeína ajuda a melhorar a força dos atletas durante o treino, além de avaliar como ela influencia a recuperação do corpo e possíveis sinais de desgaste após o exercício. **Metodologia:** Participaram do estudo 14 atletas homens de levantamento de peso paralímpico. Eles realizaram sessões de treino com e sem o uso de cafeína, em dias diferentes. Em cada sessão, os atletas fizeram um treino de supino adaptado com cargas altas e repetidas séries. Durante o estudo, foram avaliados o desempenho nos exercícios, a capacidade de gerar força, a velocidade dos movimentos e a manutenção do desempenho ao longo do treino. Também foram feitas análises no sangue e outros testes para verificar sinais de cansaço do corpo e recuperação após o exercício. **Resultados:** Os resultados mostraram que a cafeína ajudou os atletas a produzirem mais força e a terem melhor desempenho em exercícios intensos. Eles conseguiram aplicar mais força e manter melhor o rendimento durante o treino pesado. No entanto, em exercícios mais leves, não houve diferença importante com o uso da cafeína. Por outro lado, foi observado que a cafeína aumentou sinais de maior “desgaste interno” do corpo após o exercício, indicando que o organismo ficou mais sobrecarregado. Apesar disso, não foram encontrados sinais de aumento de lesões ou danos diretos aos músculos.

Conclusão: A cafeína pode ajudar atletas de levantamento de peso paralímpico a melhorar a força e o desempenho em treinos mais pesados, sendo uma estratégia útil em situações de alta intensidade. No entanto, como ela pode aumentar o nível de esforço do corpo após o exercício, seu uso deve ser feito com cuidado e orientação profissional, para garantir que os benefícios superem possíveis efeitos negativos.

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LISTA DE ABREVIATURAS E SIGLAS

1RM 1 Repetição Máxima

ANOVA – Análise de variância

ACSM- American College of Sports Medicine

CAAE -Certificado de Apresentação de Apreciação Ética

CA- Cafeína

Ca²⁺- cálcio

CRE- Creatinina

CK- Creatina quinase

CIM -Contração Isométrica Máxima

CNS -Conselho Nacional de Saúde

CONEP -Comissão Nacional de Ética em Pesquisa

DOMS- Dor muscular de início tardio

EO- Estresse oxidativo

FIM- Força Isométrica Máxima

IOC- Comitê Olímpico Internacional

IPC- Comitê Paralímpico Internacional

LDH- Lactato desidrogenase

LHS- Lipase sensível a hormônio

MDA- Malondialdeído

MPV- Velocidade propulsiva média

MIF- Força Estática Máxima

MIF 1s- Força Estática Máxima em 1 segundo

MaxV- Velocidade máxima

PC- Powerlifting convencional

PP- Powerlifting paralímpico

PDE- Fosfodiesterase

PLA- Placebo

PT- Pico de Torque

RFD- Taxa de desenvolvimento de força

ROS- Espécies reativas de oxigênio

RM- Repetição máxima

SNC- Sistema nervoso central

SH- Grupos Sulfidril

TAC- Capacidade Antioxidante Total

TBARS- Substâncias Reativas ao Ácido Tiobarbitúrico

UA- Ácido úrico

TCLE- Termo de consentimento livre e esclarecido

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1 INTRODUÇÃO

A prática de exercícios físicos é uma estratégia não farmacológica fundamental para a promoção da saúde, melhora da aptidão física e otimização do desempenho esportivo. Quando planejado e individualizado, pode induzir adaptações morfológicas e metabólicas, que variam de acordo com o tipo de estímulo, intensidade, volume e especificidade da modalidade esportiva (NATIONAL STRENGTH & CONDITIONING ASSOCIATION, 2021). Em ambientes competitivos, essas adaptações são determinantes para o desempenho esportivo, especificamente em modalidades que demandam altos níveis de força, potência e controle neuromuscular (SCHOENFELD, GRGIC e KRIEGER, 2019; ASLAM, HABYARIMANA e BIN, 2025).

No contexto do esporte de alto rendimento, modalidades que exigem força e potência têm ganhado ênfase científica, devido a demanda mecânica imposta ao sistema musculoesquelético e neural, há o desenvolvimento de danos morfológicos e moleculares, e consequentemente, redução no desempenho (SCHOENFELD *et al.*, 2017). Esse panorama é altamente representativo no powerlifting paralímpico, cujo modelo competitivo se baseia na manifestação de força máxima e intensidade severa (AIDAR *et al.*, 2021). Tais particularidades biológicas e metodológicas tornam os atletas significativamente predispostos ao desenvolvimento de fadiga neuromuscular, lesões na ultraestrutura do músculo e desequilíbrios no sistema antioxidante (AIDAR *et al.*, 2021; AIDAR *et al.*, 2022).

Devido à alta intensidade, volume dos treinos e competições em esportes de alto rendimento, há maior desenvolvimento de fadiga e alterações morfofuncionais e moleculares importantes. A fadiga em atletas de elite tem causas multifatoriais, que envolvem desde a exaustão energética, acúmulo de metabólitos e alterações hormonais, bem como, o aumento de marcadores de dano muscular (SOLER-LÓPEZ *et al.*, 2024). Essas alterações moleculares prejudicam a adaptação muscular, comprometendo a recuperação, podendo desencadear estresse oxidativo, inflamação, comprometimento da função muscular e aumentar o risco de lesão (NOCELLA *et al.*, 2019).

Assim, a utilização de suplementos para atenuar esses efeitos tem se tornado uma estratégia importante no meio esportivo. A prevalência do uso de suplementos entre atletas de alto rendimento é alta, com estimativas variando de 60% a mais de 90% em diferentes modalidades (KNAPIK *et al.*, 2016). Os suplementos mais empregados para atletas com base em dados que comprovam a melhora do desempenho físico, mas podem melhorar a saúde, a adaptação ao exercício, auxiliando os atletas no treino ou na competição com maior eficácia (RAWSON, MILES e LARSON-MEYER, 2018). Esses mesmos autores destacam em seu estudo de revisão a utilização de suplementos por atletas

como proteínas, creatina, cafeína, vitaminas, minerais, antioxidantes, ômega 3 e compostos ergogênicos, com o intuito de aprimorar o desempenho, acelerar a recuperação e reduzir danos musculares (RAWSON, MILES e LARSON-MEYER, 2018).

Adicionalmente, o Comitê Olímpico Internacional (IOC) destacou que apenas alguns suplementos possuem evidências concretas do benefício direto ao desempenho, entre eles estão: a creatina, cafeína e nitrato, sendo a utilização adequada recomendada após avaliação nutricional individualizada (MAUGHAN *et al.*, 2018). Especificamente, a cafeína é utilizada como um dos recursos ergogênicos mais utilizados e estudados no esporte, devido aos seus efeitos estimulantes no sistema nervoso central e sua capacidade de aprimorar o desempenho físico, incluindo resistência aeróbica, força muscular, potência, velocidade de movimento e capacidade de realizar exercícios repetidos de alta intensidade (JIMÉNEZ *et al.*, 2021; WANG *et al.*, 2022). Entretanto, embora há evidências consistentes sobre os efeitos da cafeína em modalidades de endurance e exercícios de força submáxima (STADHEIM *et al.*, 2021; PAKULAK *et al.*, 2022). Ainda não está totalmente esclarecido os efeitos dessa substância no desempenho neuromuscular, dano muscular e o estresse oxidativo, principalmente no Powerlifting paralímpico.

Sendo assim, a presente investigação fundamenta-se na necessidade de preencher uma lacuna crítica na literatura do esporte de alto rendimento, direcionando o foco para o powerlifting paralímpico — uma modalidade cujas respostas fisiológicas à suplementação ergogênica ainda são escassamente documentadas. Sabendo que este esporte impõe sobrecargas neuromusculares extremas, além de elevadas exigências metabólicas e funcionais aos atletas (AIDAR *et al.*, 2021; AIDAR *et al.*, 2022). Nesse sentido, este estudo avalia o impacto da cafeína sobre variáveis determinantes da performance, examinando seus mecanismos de ação direta no rendimento físico, bem como suas repercussões nos marcadores biológicos de estresse oxidativo e dano tecidual microestrutural. Ao cruzar esses dados, a pesquisa não apenas impulsiona o estado da arte nas Ciências Fisiológicas e na fisiologia do esporte adaptado, mas também fornece subsídios empíricos para que comissões técnicas elaborem protocolos individualizados.

2. REVISÃO DA LITERATURA

2.1 Powerlifting e Powerlifting paralímpico: características, demandas fisiológicas e implicações biológicas

O powerlifting convencional (PC) é uma modalidade esportiva que tem como principal objetivo a avaliação da força máxima absoluta, sendo composta por três levantamentos realizados com barra e pesos livres: agachamento, supino e levantamento terra. Em contexto competitivo, o atleta deve executar uma única repetição máxima (1RM) em cada exercício, e o desempenho final é determinado pela soma das maiores cargas levantadas nos três movimentos (FERLAND e COMTOIS, 2019). As competições são organizadas por categorias de sexo e massa corporal, o que reforça o caráter comparativo da força absoluta entre atletas com características antropométricas semelhantes. Essas particularidades conferem ao *powerlifting* uma predominância de esforços de curta duração, altíssima intensidade e baixíssimo número de repetições, com mínima contribuição de fatores temporais ou de velocidade, e elevada exigência neuromuscular (BISHOP et al., 2018; HOEK et al., 2024).

No âmbito do treinamento e da competição, o *powerlifting* prioriza adaptações neuromusculares específicas, como o recrutamento máximo de unidades motoras, a sincronização motora e o aumento da eficiência neural, em detrimento de adaptações predominantemente metabólicas (OLIVEIRA et al., 2024). O foco da modalidade está na maximização da produção de força em movimentos multiarticulares realizados sob rigoroso controle técnico e biomecânico, nos quais a capacidade de gerar altos níveis de tensão muscular é determinante para o desempenho (KOMPF e ARANDJELOVIĆ, 2017). Diferentemente do *weightlifting* e do halterofilismo olímpico, modalidades caracterizadas pela elevada demanda de potência, velocidade e coordenação intermuscular nos movimentos de arranco e arremesso, o *powerlifting* não estabelece relação direta entre desempenho e expressão de força em função do tempo, enfatizando exclusivamente a magnitude da carga deslocada (EVANGELISTA et al., 2015; ASLAM; HABYARIMANA; YONGBIN, 2025).

Essas mesmas características estruturais e fisiológicas estão presentes no *powerlifting paralímpico* (PP), modalidade que preserva os princípios fundamentais do powerlifting convencional (PC), sendo uma versão adaptada onde o único exercício realizado é o supino. Este esporte é projetado para indivíduos com deficiências físicas que afetam os membros inferiores, como paralisia cerebral, nanismo e outras condições físicas (PRATA et al. 2025). Os atletas são classificados por sexo e peso corporal, independentemente do tipo de deficiência, desde que atendam aos critérios de elegibilidade

definidos pelo Comitê Paralímpico Internacional (RUM et al., 2024). Esse mesmo comitê estabeleceu regras para a participação na modalidade, restrita a atletas com deficiências nos membros inferiores ou quadris que se enquadrem nos critérios mínimos estabelecidos para o esporte (WORLD PARAPOWERLIFTING, 2026).

Assim, o movimento realizado é feito com os membros inferiores estendidos sobre o banco e pode ser fixado por cintas para deficiências aplicáveis (LEITE JR. Et al., 2025), sendo considerado válido apenas se a barra tocar o peito e for erguida até a extensão completa dos cotovelos, sem assimetrias ou movimentos irregulares (RUM et al., 2024) (Figura 1).



Figura 1: Realização de supino por um para-atleta. Adaptado por RUM et al. (2022).

Nesse contexto, evidências recentes têm demonstrado que a presença de deficiência física não limita necessariamente a capacidade de produção de força máxima no supino. O estudo conduzido por Hoek et al. (2023) comparou o desempenho de recordistas mundiais no supino entre atletas do PP e do PC revelando que os atletas do PP apresentaram valores superiores de força absoluta e relativa em relação aos atletas do PC. Esses achados indicam que, apesar das limitações funcionais associadas às deficiências, atletas do PP são capazes de alcançar níveis elevados de desempenho neuromuscular. Como consequência desse reconhecimento científico e esportivo, observa-se um aumento expressivo no número de estudos envolvendo o PP, com destaque para os Estados Unidos (EUA) como o país com maior produção científica na área, seguido pelo Brasil, que se consolida como uma das principais nações na investigação do levantamento de peso paralímpico (SILVERTHORNE et al., 2025) (Figura 2).

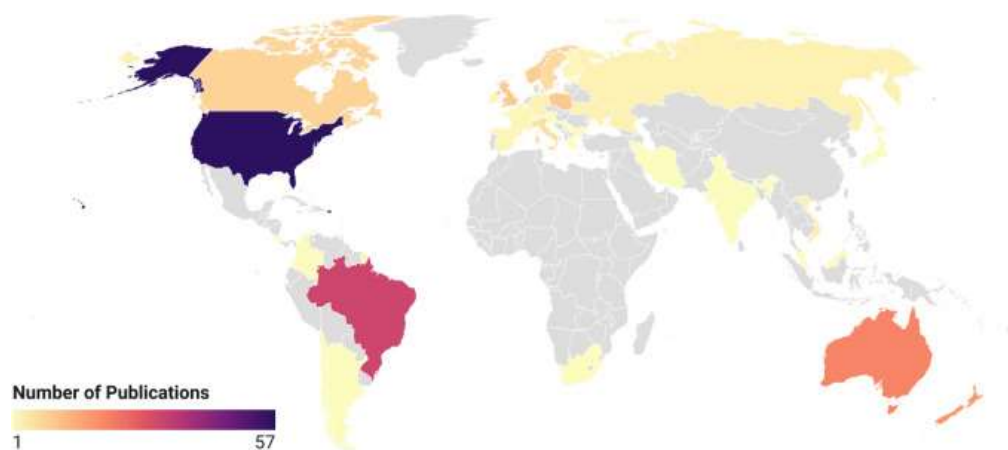


Figura 2: Artigos originários de cada país sobre a investigação do levantamento de peso paralímpico. Adaptado por SILVERTHORNE et al. (2025).

Apesar do elevado nível de desempenho apresentado por atletas do powerlifting paralímpico, as características inerentes à modalidade impõem demandas fisiológicas significativas ao sistema musculoesquelético. A execução repetida de esforços máximos ou supramáximos no supino, associada à elevada tensão mecânica, ao alto recrutamento de unidades motoras e à predominância de contrações excêntricas e isométricas em diferentes fases do movimento, pode aumentar substancialmente o risco de sobrecarga muscular e de lesões, especialmente em uma população que frequentemente apresenta assimetrias funcionais, alterações biomecânicas e padrões compensatórios de movimento (PINHEIRO et al., 2024).

Sob essas condições, a elevada sobrecarga mecânica promove microlesões nas fibras musculares e no tecido conjuntivo, desencadeando uma resposta inflamatória acompanhada pelo aumento da produção de espécies reativas de oxigênio (ERO). Quando a geração dessas espécies excede a capacidade dos sistemas antioxidantes, instala-se um estado de desequilíbrio redox, capaz de comprometer proteínas contráteis, lipídios de membrana e componentes envolvidos no acoplamento excitação-contração, reduzindo a eficiência da produção de força muscular. Paralelamente, a elevada demanda neural necessária para o recrutamento de unidades motoras de alto limiar favorece o desenvolvimento da fadiga neuromuscular, tanto por mecanismos periféricos quanto centrais, culminando na redução do desempenho físico e no aumento da suscetibilidade a lesões (SOLER-LÓPEZ et al., 2024).

Dessa forma, torna-se fundamental investigar de maneira sistemática os marcadores de dano muscular e estresse oxidativo nessa modalidade, a fim de compreender as respostas fisiológicas e subsidiar estratégias de treinamento e intervenção, promovendo melhorias no desempenho.

2.1.1 Dano muscular e desequilíbrio redox em esporte de alto rendimento

Exercícios de alta intensidade desencadeiam respostas metabólicas que incluem aumento significativo da produção de espécies reativas de oxigênio (ROS), causando danos musculares e estresse oxidativo (EO). A sobrecarga mecânica desencadeia danos estruturais nas fibras musculares, com alterações na permeabilidade da membrana, desorganização do sarcômero e desenvolvimento de processos inflamatórios, evidenciados por elevação de marcadores como creatina quinase (CK), lactato desidrogenase (LDH) e mioglobina, além de alterações funcionais como redução temporária da capacidade de produção de força, aumento da rigidez muscular, diminuição da amplitude de movimento, edema local e dor muscular de início tardio (DOMS) (STOŽER, VODOPIVC e BOMBEEK, 2020).

O dano muscular induzido pelo exercício ocorre nas primeiras 24 horas, atingindo o pico entre 24 e 72 horas, e podem durar de cinco a sete dias após a sessão de exercício (LEITE et al., 2023). Esses processos fazem parte das adaptações ao treinamento, entretanto, quando excessivos podem comprometer o desempenho e aumentar o risco de lesões musculoesqueléticas nos atletas (Figura 3).

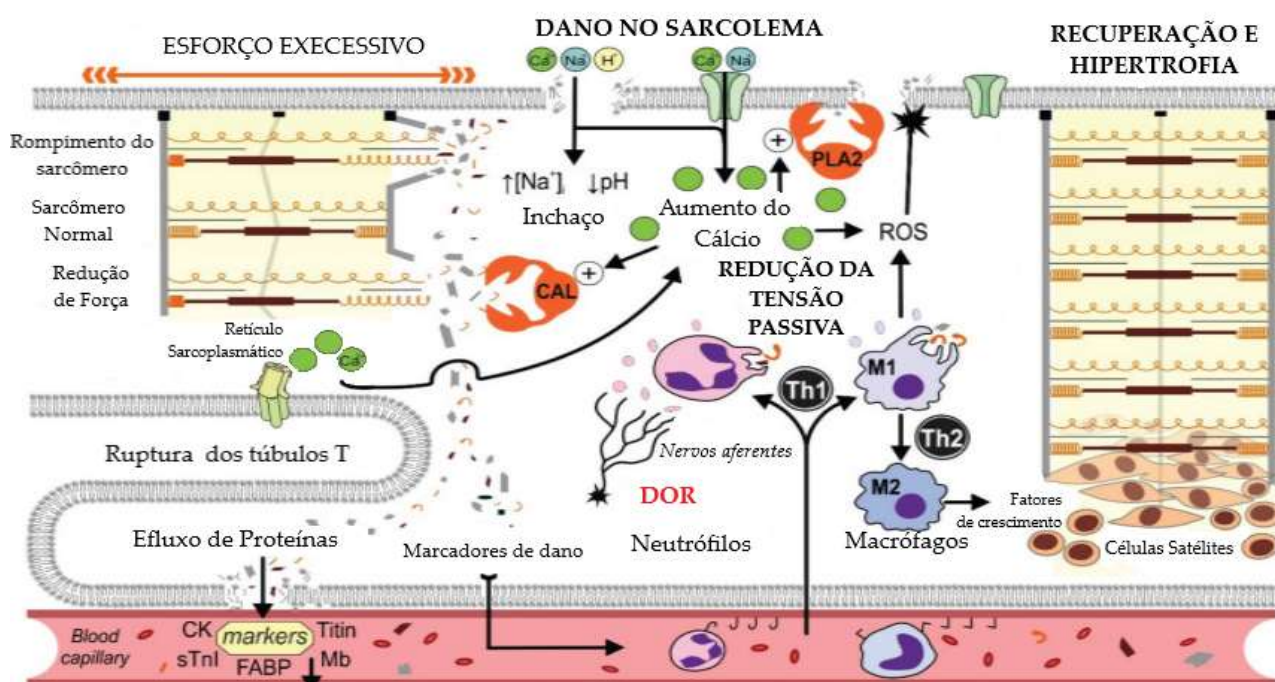


Figura 3: Mecanismos moleculares cruciais propostos para os sintomas e o processo de reparação da lesão muscular induzida pelo exercício. Adaptado por STOŽER, VODOPIVC e BOMBEEK (2020).

O estresse oxidativo caracteriza-se pelo desequilíbrio entre a produção de espécies reativas de oxigênio (EROs) e de nitrogênio (ERNs) e a capacidade do sistema antioxidante em neutralizá-las,

condição frequentemente exacerbada durante exercícios de alta intensidade (NOCELLA et al., 2019). Durante o exercício, o aumento da demanda energética eleva o consumo de oxigênio e acelera o metabolismo celular, favorecendo a formação dessas espécies reativas. Embora sejam frequentemente associadas a danos celulares, as EROs desempenham funções fisiológicas essenciais, atuando como moléculas sinalizadoras envolvidas na regulação da expressão gênica, na adaptação ao treinamento, na vasorregulação, na resposta imunológica e na indução da síntese de enzimas antioxidantes (GOMES; SILVA; OLIVEIRA, 2012; PISOSCHI; POP, 2015).

Em condições fisiológicas, a produção de EROs é rigorosamente controlada pelos sistemas antioxidantes enzimáticos e não enzimáticos, responsáveis por manter a homeostase redox e limitar o dano oxidativo (GOMES; SILVA; OLIVEIRA, 2012; HALLIWELL, 2014). Entretanto, quando a geração dessas espécies excede a capacidade antioxidante do organismo, estabelece-se um estado de estresse oxidativo. Nessa condição, as EROs passam a oxidar proteínas, lipídios e ácidos nucleicos, promovendo alterações estruturais e funcionais que comprometem a integridade celular e a função tecidual (JACKSON; VASILAKI; MCARDLE, 2016; PISOSCHI; POP, 2015). Dentre alguns tipos de espécies reativas podemos visualizar através da tabela adaptada de Zhang *et al* (2019):

Tabela 1: Características químicas das principais EROS

Espécies	Reatividade	Alvo preferido
Radical livre:		
Óxido nítrico ($\text{NO}^\bullet$)	+	Metais
Anión de radical superóxido ($\text{O}_2^{\bullet-}$)	++	Cluster Fe – S, $\text{NO}^\bullet$
Radicais hidroxila ($\text{OH}^\bullet$)	++++++	Qualquer macromolécula
Anión de radical carbonato ($\text{CO}_3^{\bullet-}$)	+++++	Tyr, Trp, tióis, guanina
Dióxido de nitrogênio ($\text{NO}_2^\bullet$)	+++	Thiols, Tyr
Radical alcoxila ($\text{RO}^\bullet$)	++++	Compostos orgânicos
Radical peroxil ($\text{ROO}^\bullet$)	+++	Compostos orgânicos
Não radicais:		
Peróxido de hidrogênio (H_2O_2)	++	Tióis, metais

Peroxinitrito (ONOO ⁻ / ONOOH)	++++	CO ₂ , Cys, Trp, Met, Metais
Ácido hipocloroso (HOCl)	++++	Amino, tióis
Oxigênio singlete (¹ O ₂)	++	Guanina, lipídios não saturados, aminoácidos

Legenda: A tabela apresenta reatividade e alvo preferido das principais EROS. A reatividade é definida de acordo com um potencial de redução de elétrons (E°) em pH7 ou a taxa de reação com glutatona (GSH) para radicais e não radicais, e é fornecida com uma descrição relativa de + e ++ (baixo), +++ (mediar) e ≥ ++++ (alto). **Abreviação:** Cys, Cysteine; Fe-S, sulfeto de ferro (II); Met, Metionina; EROS, espécies reativas de oxigênio; Tyr, tirosina; Trp, triptofano; Elétron não emparelhado indicado por um ponto sobrescrito à direita que precede o símbolo de carga.

No músculo esquelético, o estresse oxidativo exerce papel relevante na redução da capacidade contrátil. A oxidação de proteínas miofibrilares, enzimas metabólicas e componentes envolvidos no acoplamento excitação-contração prejudica a produção de força, reduz a eficiência metabólica e favorece o desenvolvimento da fadiga muscular (JACKSON; VASILAKI; MCARDLE, 2016). Além disso, o excesso de EROs potencializa a resposta inflamatória decorrente do dano muscular induzido pelo exercício, retardando os processos de recuperação e aumentando a suscetibilidade a novas lesões.

Embora a mitocôndria represente uma das principais fontes de produção de EROs durante o exercício, essas espécies também são geradas por outras vias enzimáticas, como a NADPH oxidase e a xantina oxidase, além de células do sistema imunológico recrutadas para o tecido lesionado (NOCELLA et al., 2019). Após exercícios que promovem dano muscular, especialmente aqueles com elevada componente excêntrica, ocorre infiltração de neutrófilos e macrófagos no tecido muscular. Essas células são fundamentais para a remoção de detritos celulares e para o processo de regeneração, porém liberam quantidades adicionais de EROs durante a resposta inflamatória, ampliando temporariamente o estresse oxidativo local (POWERS; NELSON; HUDSON, 2011).

Em atletas submetidos a elevadas cargas de treinamento, a manutenção prolongada desse ambiente pró-oxidante pode comprometer as adaptações ao exercício e favorecer o desenvolvimento de estados de fadiga persistente e overtraining. Nesse contexto, Tanskanen, Atalay e Uusitalo (2010) demonstraram que o aumento do estresse oxidativo participa da fisiopatologia da síndrome do overtraining, contribuindo para a redução da capacidade de desempenho. De forma semelhante, Magherini et al. (2019) observaram que o excesso de estresse oxidativo altera a secreção de hormônios relacionados ao metabolismo energético, como leptina, adiponectina e grelina, comprometendo o controle do balanço energético e os processos de recuperação. Adicionalmente, Cheng et al. (2020) demonstraram que a interação entre estresse oxidativo e inflamação constitui um

dos principais mecanismos responsáveis pelas alterações morfológicas e funcionais observadas no músculo esquelético após exercícios extenuantes.

Diante dessas evidências, o estresse oxidativo deve ser compreendido não apenas como consequência do exercício intenso, mas como um importante modulador das respostas adaptativas e da recuperação muscular. Em modalidades caracterizadas por elevada exigência neuromuscular, como o powerlifting paralímpico, nas quais esforços máximos são realizados repetidamente, a manutenção do equilíbrio redox torna-se fundamental para preservar a função muscular, otimizar o desempenho e reduzir os efeitos deletérios associados ao dano muscular e à fadiga. Nesse contexto, torna-se relevante investigar estratégias capazes de modular o estado redox e favorecer a recuperação fisiológica de atletas submetidos a elevadas cargas de treinamento.

2.2 Suplementação de Cafeína

A cafeína é quimicamente conhecida como 1,3,7-trimetilxantina, um composto orgânico classificado como uma xantina, de natureza anfipática e com rápida absorção gastrointestinal (REDDY *et al.*, 2024; SILVA *et al.*, 2020). É reconhecida como o suplemento estimulante mais popular globalmente, principalmente por atletas. Sua popularidade no meio esportivo decorre de propriedades ergogênicas associadas a melhora do desempenho físico, bem como de seu custo relativamente baixo e ampla acessibilidade (SAMPAIO-JORGE *et al.*, 2021; WILK *et al.*, 2019a; SILVA *et al.*, 2020). Os efeitos incluem o aumento do estado de alerta e da vigília, retardamento da fadiga e a melhora do humor (BARCELOS *et al.*, 2020; SILVA *et al.*, 2020).

A cafeína anidra em cápsulas é considerada uma forma de suplementação eficiente, sendo a eficácia atribuída à facilidade de ingestão (SOKMEN *et al.*, 2008), evitando a interferência de outras substâncias que poderiam modular seu efeito ergogênico (SILVESTRE *et al.*, 2018). A dose recomendada varia entre 3 a 9 miligrama por quilograma (mg/kg) de peso corporal, doses superiores a 10 mg/kg podem ser tóxicas, manifestando-se através de diversos sintomas, como: tremores, insônia, nervosismo, irritabilidade, ansiedade, náuseas e desconforto gastrointestinal (PALLARÉS *et al.*, 2013; WILK *et al.*, 2019a).

Nesta perspectiva, a dose ideal deve ser ajustada de acordo com o tipo de modalidade, intensidade e volume do exercício, apresentando variações de acordo com fatores como sexo e idade do indivíduo (WILK *et al.*, 2019a). A literatura científica aponta uma variabilidade na eficácia da cafeína, especialmente em indivíduos habituados ao consumo. Wilk *et al.* (2019b) observaram que

altas doses agudas (9 e 11 mg/kg) não resultaram em melhorias na força ou resistência muscular em atletas já habituados à substância. Em outro estudo, Wilk et al. (2019a) também indicaram que doses de 3, 6, e 9 mg/kg antes do exercício não tiveram efeito significativo na potência ou na velocidade da barra em usuários habituais de cafeína.

Em contraste, Pallarés et al. (2013) observaram que a dose de 9 mg/kg melhorou a velocidade e a potência da barra durante exercícios de supino e agachamento com cargas progressivas (25% a 90% de 1RM). Além disso, a ingestão aguda de altas doses (9 e 12 mg/kg) aumentou a velocidade média e máxima da barra em atletas com ingestão habitual moderada a alta (FILIP-STACHNIK et al., 2021). Todavia, a dose de 12 mg/kg aumentou significativamente a frequência de efeitos colaterais negativos (FILIP-STACHNIK et al., 2021). Durkalec-Michalski et al. (2019) observou que doses de 6 e 9 mg/kg de cafeína otimizaram o desempenho físico, sendo a dose de 9 mg/kg também aumentou a atividade intermitente de alta intensidade. A dose de 3 mg/kg não produziu efeito positivo, sugerindo que doses mais elevadas tendem a ser mais eficazes. Dessa maneira, a dose de 9 mg/kg foi considerada um protocolo eficaz e seguro para indivíduos habituados à cafeína (GUEST et al., 2021). Apesar de várias evidências que sustentam o potencial ergogênico da cafeína, os resultados apresentados na literatura permanecem heterogêneos, principalmente em exercícios de força e potência. Essa variação de resultados pode ser influenciada por vários fatores, como dose ingerida, tipo de exercício, nível de treinamento do atleta, características biológicas individuais e o grau de habituação ao consumo de cafeína. Nesse contexto, compreender os mecanismos fisiológicos e neurobiológicos subjacentes à ação da cafeína torna-se fundamental para elucidar seus efeitos sobre o desempenho físico.

2.2.1 Mecanismos de ação da cafeína

A cafeína apresenta uma farmacocinética bem estabelecida, caracterizada por rápida absorção gastrointestinal, metabolismo hepático e ampla distribuição tecidual. Sua biodisponibilidade é próxima a 100%, com a concentração plasmática máxima atingida entre 30 e 120 minutos após a ingestão, geralmente em torno de 60 minutos, dependendo da forma de consumo e de variáveis individuais (NEHLIG, 2018; SILVESTRE *et al.*, 2018; BARCELOS *et al.*, 2020). A ingestão juntamente com alimentos ou carboidratos pode retardar a absorção e atenuar o pico de concentração plasmática.

Devido à sua natureza anfipática, a cafeína distribui-se amplamente pelos tecidos, atravessando as membranas celulares, a barreira hematoencefálica e a placenta. A excreção ocorre predominantemente pela urina, com meia-vida variável entre 2 e 12 horas, influenciada por fatores fisiológicos, ambientais e genéticos, incluindo alterações no metabolismo hepático (RUIZ et al., 2011; FRANÇA et al., 2015; NEHLIG, 2018).

O principal efeito ergogênico da cafeína está associado à sua ação no sistema nervoso central, atuando como antagonista competitivo dos receptores de adenosina (A_1 e A_2). Ao bloquear a ação inibitória da adenosina, a cafeína aumenta a excitabilidade neuronal, resultando em maior estado de alerta, redução da percepção de esforço e atenuação da fadiga central (BARCELOS et al., 2020; BENJAMIM et al., 2021).

No fígado, a cafeína é metabolizada principalmente pela enzima citocromo P450 1A2 (CYP1A2), originando três metabólitos biologicamente ativos: paraxantina (~84%), teobromina (~12%) e teofilina (~4%) (Figura 4). Essas moléculas apresentam efeitos fisiológicos distintos, incluindo estímulo à lipólise, vasodilatação e broncodilatação, respectivamente (NEHLIG, 2018; REDDY et al., 2024).

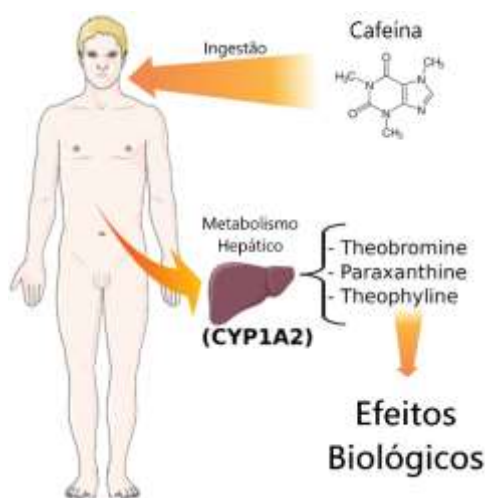


Figura 4: Metabolização da cafeína pelo fígado. Adaptado por BARCELOS et al., (2020).

No músculo esquelético, a cafeína pode influenciar diretamente o acoplamento excitação contração ao aumentar a liberação de cálcio (Ca^{2+}) a partir do retículo sarcoplasmático por meio da interação com os receptores de rianodina. Esse aumento da disponibilidade de Ca^{2+} contribui para maior recrutamento de unidades motoras, aumento da tensão muscular e potencial otimização da produção de força, mecanismos particularmente relevantes em exercícios de alta intensidade e curta duração (MATERKO et al., 2011; BARCELOS et al., 2020).

Embora a cafeína também atue no metabolismo energético, através do estímulo da lipólise e da inibição da fosfodiesterase (PDE), promovendo elevação do AMPc e ativação da lipase sensível a hormônio (LHS), esses efeitos parecem ter maior relevância em exercícios de longa duração. Em modalidades caracterizadas por esforços máximos e curta duração, tais mecanismos metabólicos provavelmente exercem papel secundário quando comparados aos efeitos neurais e neuromusculares da cafeína (SAMPAIO-JORGE et al., 2021; TAVARES; SAKATA, 2012).

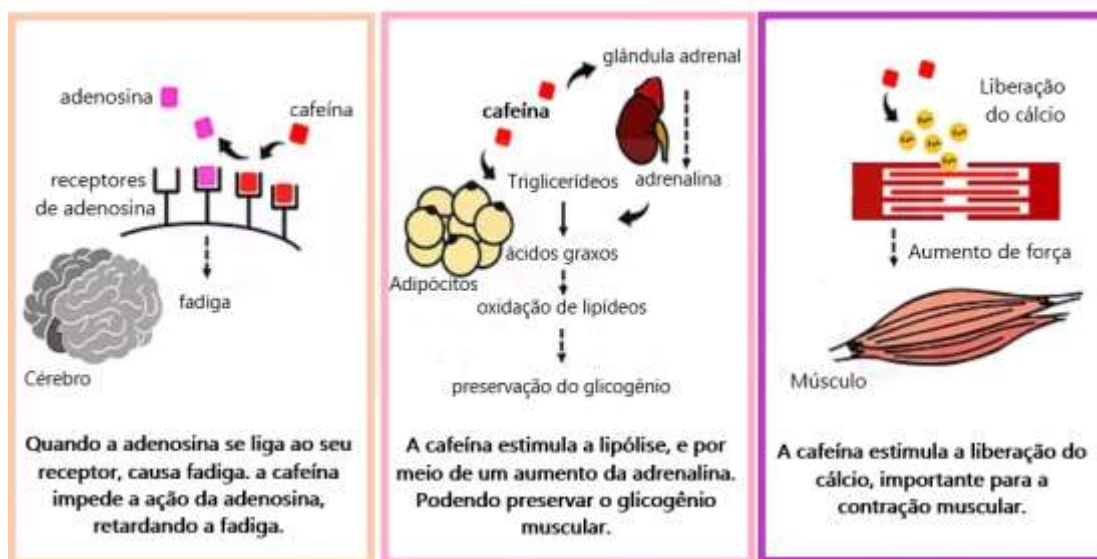


Figura 5: Efeitos da cafeína no SNC e metabolismo. Adaptado de Mysportscience (2022).

Diante dos mecanismos centrais e periféricos atribuídos à cafeína, percebe-se que seus efeitos envolvem uma complexa interação entre modulação neural, excitabilidade muscular e respostas metabólicas. Todavia, a relevância desses mecanismos varia conforme o objetivo do seu uso, como o tipo de modalidade esportiva, a intensidade e a duração do esforço, bem como as características individuais do atleta. Nesse sentido, compreender como a cafeína tem sido utilizada no esporte e de como esses mecanismos otimizam o desempenho físico torna-se essencial, especialmente em modalidades de força máxima, nas quais os efeitos ergogênicos parecem estar mais associados à tolerância ao esforço, à eficiência neuromuscular e à atenuação da fadiga central e ao metabolismo energético.

2.2.2 Benefícios da Cafeína no desempenho esportivo

A utilização de suplementação para otimizar o desempenho, tem ganhado destaque em pesquisas na área na nutrição esportiva. Estratégias suplementares são investigadas para a preparação da competição (como a alimentação pré-exercício), desenvolvimento da performance durante a competição e a recuperação pós-competição (FECHAR et al., 2016). Além disso, pesquisadores investigam o uso de recursos ergogênicos que podem melhorar o desempenho no exercício, principalmente no esporte de alto rendimento, com objetivo de reduzir a fadiga, modulando alterações neuromusculares, minimizando danos musculares e controlado equilíbrio redox (NAVARRO et al, 2025). Especificamente, a cafeína é reconhecida pela American College of Sports Medicine (ACSM), pois potencializa o desempenho esportivo por meio de ações centrais e periféricas no metabolismo corporal, sendo um dos recursos ergogênicos mais estudados e utilizados em atletas de alto rendimento, por aumentar o estado de alerta e potencializar a performance em múltiplas modalidades (NUTRITION AND ATHLETIC PERFORMANCE, 2016).

De acordo com Lima e Silva et al. (2021), a cafeína apresenta efeitos em várias modalidades esportivas e exercícios físicos. Os autores destacam que o efeito estimulante da cafeína no sistema nervoso central (SNC) é o mecanismo mais aceito para explicar a melhora no desempenho físico durante exercícios de alta intensidade que envolvem o corpo todo. Mas, devido a sua rápida ação e absorção no organismo, outros sistemas fisiológicos, também podem ser ativados ao máximo. Sabe-se que a cafeína promove benefícios agudos em diversos aspectos do desempenho esportivo, incluindo resistência aeróbica, força muscular, potência, velocidade de movimento e capacidade de realizar exercícios repetidos de alta intensidade (GRGIC et al., 2020).

Um estudo realizado por Stadheim et al. (2021) mostrou que a cafeína aumentou o tempo de exaustão após a ingestão, elevou o $\text{VO}_{2\text{máx}}$ em atletas de elite, o que contribuiu para a melhora no desempenho de resistência de alta intensidade. Além disso, Grgic et al. (2018) mostraram em uma meta-análise que a cafeína é eficiente no ganho de força muscular máxima da parte superior do corpo e na potência muscular em homens. Ferreira, Silva e Bueno (2021) demonstraram efeito significativo na resistência muscular e na força máxima no exercício de supino. Em contraste, Ruiz-Fernández et al. (2023) mostrou que a ingestão aguda de cafeína melhorou a força muscular, a potência e resistência, revelando-se mais eficiente em cargas mais altas ($\geq 75\%$ 1RM) e em exercícios para membros inferiores do que em membros superiores, evidenciando resultados de acordo com o tamanho do grupo muscular. Recentemente, Montalvo-Alonso et al. (2024) mostraram que a ingestão

aguda de cafeína apresentou efeito semelhante aos estudos anteriores para exercícios que enfatizam tanto membros superiores quanto os inferiores, em participantes do sexo masculino e feminino treinados em resistência. Esse conjunto de dados demonstra que a cafeína desencadeia respostas diferentes, dependendo da dosagem, sexo e condicionamento do indivíduo, sendo ainda contraditórios seus resultados em grupos musculares específicos.

Essas melhorias no desempenho físico estão associadas a mecanismos moleculares centrais e periféricos bem estabelecidos. No SNC, a cafeína atua como antagonista dos receptores de adenosina A_1 e A_2A , reduzindo a inibição neural, aumentando a excitabilidade cortical e a ativação motora voluntária. Esses efeitos resultam em menor percepção de fadiga e dor, maior recrutamento de unidades motoras, além de aumento da motivação e do estado de alerta, favorecendo a produção de força e potência muscular durante o exercício (AGUIAR JR. et al., 2020; MESQUITA et al., 2020; GRGIC, 2021). No nível periférico, a cafeína aumenta a liberação de Ca^{2+} pelo retículo sarcoplasmático nas fibras musculares, facilitando o acoplamento excitação–contração e, conseqüentemente, elevando a contratilidade muscular (FUKUTANI, KUNIMATSU e ISAKA, 2022).

Além disso, pode aumentar a disponibilidade de ácidos graxos como substrato energético por meio da estimulação da lipólise e modular a atividade da bomba Na^+/K^+ , contribuindo para a melhora da condução do impulso elétrico muscular (MAGKOS, STAVROS e KAVOURAS, 2005; Wu et al., 2024). Embora esses efeitos centrais e periféricos expliquem, de forma ampla as melhorias no desempenho físico associadas à cafeína, o aumento da ativação neural e da contração muscular pode também ter influência no estresse mecânico imposto ao músculo esquelético durante o exercício. Isso também levanta questionamentos acerca do impacto da cafeína sobre o dano muscular e as respostas relacionadas ao estresse oxidativo induzido pelo exercício.

Os efeitos da cafeína na prevenção do dano muscular e do estresse oxidativo em atletas são limitados e, na grande maioria, não há evidências robustas de que a cafeína possa prevenir o dano muscular induzido pelo exercício ou atenuar marcadores clássicos de estresse oxidativo em protocolos de alta intensidade. Estudo de Ribeiro et al. (2016) evidenciou que a ingestão imediata de cafeína (6 mg/kg de peso corporal) reduziu o nível de fadiga muscular e preservou a potência, sem alterações em marcadores de dano muscular. Filip-Stachnik et al. (2023) mostrou que em uma concentração menor de cafeína (3 mg/kg de peso corporal) não houve aumento da resistência muscular e nem alterações significantes no equilíbrio pró-oxidante-antioxidante e no dano muscular em homem treinados com exercício de resistência.

Além disso, Chen et al. (2019) mostrou que a cafeína na dose de 6 mg/kg parece facilitar a recuperação da potência muscular anaeróbica e atenuar a dor muscular tardia após exercício de alta intensidade em ambos os sexos. Caldas et al. (2022) evidenciou através de uma revisão sistemática que em quatro estudos com a administração repetida de cafeína entre 24 e 72 horas após o dano muscular pode atenuar a percepção da dor em magnitudes que variam de 3,9% a 26%. A dose única de cafeína antes ou após o exercício não alterou os níveis sanguíneos circulantes de creatina quinase (CK). A suplementação com cafeína parece atenuar a percepção da dor, mas isso não parece estar relacionado a uma atenuação da lesão muscular induzida pelo exercício. Grgic et al (2019) indica que doses na faixa de 3 a 9 mg/kg parecem ser adequadas para produzir respostas metabólicas quando administradas 60 minutos antes do exercício.

Em relação ao estresse oxidativo, a cafeína apresenta propriedades antioxidantes *in vitro* e em modelos animais (CECHELLA et al., 2014; BARCELOS et al., 2020), mas em humanos, a suplementação aguda (3-6 mg/kg) não reduz de forma significativa os marcadores de estresse oxidativo após exercício intenso (FILIP-STACHNIK et al., 2023). Em atletas treinados, a cafeína pode aumentar a concentração de glutathiona reduzida (GSH) após o exercício, proporcionando um nível de efeito antioxidante, mas sem impacto relevante em outros marcadores de dano oxidativo (FILIP-STACHNIK et al., 2023). Em suma, a cafeína pode desencadear respostas na contratilidade do músculo esquelético e mobilizar substratos energéticos, mas não há evidências suficientes que indiquem que esses mecanismos previnam dano estrutural muscular ou previnam o estresse oxidativo em atletas de elite.

3. PROBLEMA DO ESTUDO

Apesar da cafeína ser amplamente utilizada como recurso ergogênico por atletas, a maioria das evidências enfatizam modalidades de endurance ou em exercícios de força submáxima, havendo lacunas relevantes quanto aos seus efeitos em esportes caracterizados por esforços máximos, como o Powerlifting. Além disso, embora os mecanismos centrais e periféricos da cafeína sejam relativamente bem descritos, ainda não está claro como esses efeitos se traduzem simultaneamente em alterações no desempenho de força dinâmica e estática, bem como em respostas fisiológicas relacionadas ao dano muscular e ao estresse oxidativo em atletas paralímpicos. Diante desse cenário, podemos questionar: Quais os efeitos da suplementação de cafeína durante o treinamento sobre o desempenho de força dinâmica e estática, e suas implicações sobre o estresse oxidativo e o dano muscular em atletas paralímpicos?

O estudo será apresentado no modelo escandinavo com os capítulos em forma de artigo com a produção de três manuscritos, com base nos objetivos específicos, contendo a formatação de título, resumo, metodologia, resultados, discussão, conclusão e referências. Os artigos responderão as questões citadas acima.

4. OBJETIVOS

4.1 GERAL

Avaliar o efeito da cafeína sobre indicadores neuromusculares, estresse oxidativo e dano muscular no Powerlifting Paralímpico

4.2 ESPECÍFICOS

- Avaliar os efeitos da suplementação de cafeína na fadiga antes, durante e após sessões de treinamento em atletas de levantamento de peso paralímpico;
- Avaliar o efeito da suplementação de cafeína sobre os indicadores estáticos da força após o treinamento a 80% de 1RM em atletas de powerlifting paralímpico;
- Avaliar o efeito da suplementação de cafeína sobre os indicadores de estresse oxidativo, danos musculares e a temperatura corporal após o treinamento a 80% de 1RM de atletas de powerlifting paralímpico.

5. ESTUDO 1: EFEITO DA SUPLEMENTAÇÃO AGUDA DE CAFEÍNA SOBRE OS INDICADORES DE FORÇA DINÂMICA EM DIFERENTES MOMENTOS DE TREINAMENTO NO LEVANTAMENTO DE PESO PARALÍMPICO.

Menezes JL, Aidar FJ, Fischetti F, Badicu G, Getirana-Mota M, González-Valero G, et al. Effect of Acute Caffeine Supplementation on Dynamic Force Indicators at Different Training Moments in Paralympic Powerlifting. **Journal of Biological Regulators and Homeostatic Agents**, 2023, **37(9):4571-4579**. doi.org/10.23812/j.biol.regul.homeost.agents.20233709.446

EFFECT OF ACUTE CAFFEINE SUPPLEMENTATION ON DYNAMIC FORCE INDICATORS AT DIFFERENT TRAINING MOMENTS IN PARALYMPIC POWERLIFTING

Background: Several reports have highlighted the beneficial impacts of caffeine on performance in various disciplines of Paralympic Powerlifting (PP), a sport renowned for its emphasis on maximal strength. Moreover, caffeine consumption within the context of PP has been found to be safe. **Objective:** To examine the effects of caffeine intake before, during, and after PP training sessions at national level in Brazil. **Methods:** Thirteen male PP athletes competing at national level (31.31 ± 10.13 years, 80.77 ± 22.66 kg) participated in the study. They were provided with either 9.0 mg/kg of Caffeine Anhydrous (CA) or Placebo (PL) and were evaluated using 45% of their one-repetition maximum (1RM) before and after training sessions, as well as 24 and 48 hours after sessions. Additionally, they performed five sets of five repetitions maximum (5x5), with assessments carried out during the first and fifth sets for all five repetitions. Evaluations focused on Mean Propulsive Velocity (MPV), Maximum Velocity (MaxV), and Power. **Results:** No significant differences were observed with 45% 1RM. However, at 80% 1RM, CA demonstrated significant improvement compared to PL during Set 1 and Set 5 ($p < 0.05$). **Conclusions:** CA exhibits promising ergogenic properties, enabling athletes to sustain training intensity throughout the session, even when working with heavier PP loads.

Keywords: caffeine; strength training; Paralympic Powerlifting

INTRODUCTION

The performance-enhancing benefits of caffeine have been documented across various modalities, including resistance training, intermittent exercise, and strength activities [1–3]. Research has examined caffeine supplementation and its impact on subjective exertion perceptions. It has been found that Caffeine Anhydrous (CA) affects both the central nervous system and peripheral levels [4,5]. By reducing fatigue and effort during competitions, CA has the potential to enhance performance and boost overall vigor [4].

Consequently, an increasing number of athletes are consuming caffeine as a way to enhance their performance [6,7]. Nevertheless, the effectiveness of caffeine supplementation can be influenced by various factors such as training level, timing of ingestion, frequency of use, and the specific type of exercise performed [4,8]. Importantly, it has been determined that caffeine is safe for use in strength training exercises like Bench Press, including those involving Paralympic athletes and related sports [9,10].

When considering strength exercises, particularly in the context of Paralympic Powerlifting characterized by heavy loads and one-repetition maximum (1RM) [11,12], several studies have indicated that the use of ergogenic aids tends to enhance performance and aid in post-training recovery [13,14].

In contrast to the perceived benefits of supplements like caffeine to enhance performance, research findings have indicated divergence in effects, including potential negative outcomes associated with caffeine consumption [9,15]. Some athletes may not experience performance improvements or even perceive a decline in performance [16–18]. Moreover, the results of various studies have demonstrated contradictory findings concerning the impact of caffeine on different types of exercises [19,20] and the influence of training level [19–22]. This suggests that while caffeine may positively contribute to improving performance in some cases, it may also have no discernible effect [20,23,24].

Therefore, the objective of this study was to assess the effects of caffeine supplementation before, during, and after training sessions in national-level Paralympic powerlifting athletes. Based on the research hypothesis, it could be concluded that caffeine would enhance performance in this specific group of athletes and alleviate post-training fatigue.

Materials and Methods

Study Design

During the first week, familiarization, body assessments, consent form signing, and the 1RM evaluation were conducted. In the subsequent weeks (second and third), participants received either Caffeine Anhydrous (CA) or Placebo (PL) supplementation, based on random allocation, and then proceeded with exercise sessions. These sessions consisted of five sets of five repetitions maximum (5x5) at 80% of their one-repetition maximum, with rest periods of three to five minutes between sets [10,25]. Following the training sessions, athletes resumed their daily activities. A washout week was not included in the study design, as CA has maximum half-life of 10 hours, providing sufficient time for the substance to be eliminated from participants' bodies [26] (Fig. 1).

Participants

The study included a sample of 13 male Paralympic Powerlifting (PP) athletes who held top-10 national rankings in their respective weight categories. All participants met the eligibility criteria established by the Brazilian Paralympic Committee for competing in the sport [27]. Athletes had various disabilities, including one with left leg atrophy, one with below-the-knee amputation on the left leg, two with below-knee amputation on the right leg, one with right leg injury resulting in loss of function and muscle tone, one with spinal cord injury due to schistosomiasis infection, one with above-knee amputation on the right leg, three with poliomyelitis sequelae affecting the lower limbs, and three with arthrogryposis.

Individuals with cardiorespiratory or cardiac diseases or those using any form of ergogenic aids were excluded from the sample. Women did not participate in the study due to potential hormonal changes and the potential interaction of hormone supplementation [10]. All participants voluntarily agreed to participate and provided informed consent. The study was approved by the Research Ethics Committee of the Federal University of Sergipe (ID-CAAE: 71549517.0.0000.5546, technical advice: 2.637.882, approval date: May 7, 2018), and is in compliance with the ethical principles outlined in the Declaration of Helsinki (1964, revised in 2013) by the World Medical Association, as well as with Resolution 466/2012 of the National Research Ethics Committee - CONEP of the National Health Council. A detailed description of sample characteristics can be found in Table 1 (Ref. [28]).

Table 1: Characteristics of the study participants (n = 13).

Variable	(Mean $\pm$ standard deviation)
Age (years)	31.31 $\pm$ 10.13
Body mass (kg)	80.77 $\pm$ 22.66
Experience (years)	4.27 $\pm$ 1.20
Systolic blood pressure (mmHg)	125.3 $\pm$ 14.7
Diastolic blood pressure (mmHg)	74.9 $\pm$ 11.7
1RM test (bench press) (kg)	147.31 $\pm$ 41.86
1RM test/body mass (kg)	1.87 $\pm$ 0.40

1RM: one-repetition maximum. All athletes with loads that keep them in the top 10 of their categories nationwide. Athletes with values above 1.4 in the Bench Press (1-RM/Body Weight) would be considered elite athletes, according to Ball & Weidman [28].

Instruments and Procedures

Athletes' body mass was measured using a digital electronic platform scale with wheels (Micheletti, São Paulo, Brazil), which had maximum capacity of 300 kg. To conduct evaluations, an official 2.10 m Bench Press (Eleiko, Halmstad, Sweden) and a 20 kg Olympic barbell (total length 220 cm) were used, both of which were approved by IPC [25,29]. Additionally, a Vitruve linear position encoder was used. Force Measurement System (Vitruve, Mostoles, Madrid, Spain) [30] was used to assess dynamic strength indicators such as Maximum Velocity (MaxV), Mean Propulsive Velocity (MPV), and Power. This encoder precisely measured the vertical displacement velocity, capturing data from the beginning of the movement until full arm extension [31], which has demonstrated reliability when used in bench press evaluations [32]. For the 1RM test, load increments were adjusted based on the athletes' estimation of their maximum lift effort. In the event of failure, the load was reduced by 2.5%. The test protocol consisted of a maximum of five attempts, with a 5-minute rest interval between each attempt [33,34].

Weeks		Procedures and tests		
Week 1 (Familiarization and 1RM test)	→	Body assessment and ASS → Warm up	TEST  (1RM test)	→ Rest 
Week 2 (supplementation 50% Caffeine or 50% Placebo)	→	Pre Test Supplement (MPV, MaxV and Power with 45% 1RM) → Warm up	Training  5X5, 80%1RM	→ Post-test, 24 and 48 Hours (MPV, MV and Power with 45% 1RM)
Week 3 (supplementation)	→	Pre Test →	Training	→ Post-test, 24 and 48 Hours

50% Caffeine or 50% Placebo)	Supplement (MPV, MaxV and Power with 45% 1RM)	Warm up	 5X5, 80%1RM	(MPV, MV and Power with 45% 1RM)
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Fig. 1. Experimental study design. Notes: ASS, consent form signature; 5x5, five sets of five repetitions maximum; 1RM, onerepetition maximum; MPV, Mean Propulsive Velocity; MaxV, Maximum Velocity.

Caffeine Anhydrous (CA) and Placebo (PL)

Caffeine Anhydrous (CA) and Placebo (PL) capsules were prepared at a compounding pharmacy using capsules that were identical in color, size, and quantity. CA capsules were carefully measured to contain the dosage of 9.0 mg/kg of body weight [35,36], while PL capsules were filled with an appropriate amount of pharmaceutical talc to match the number of CA capsules. CA or PL allocation was determined through a random drawing conducted immediately after the athletes' arrival at the collection room. A standardized interval of 60 minutes was observed between supplement's ingestion and the tests' conduction.

Assessments and Training

The protocol used VPM, MaxV, and Power indicators to assess velocity loss using load equivalent to 45% of 1RM [37]. Athletes' performance was evaluated through three repetitions, both before and immediately after training, as well as 24 and 48 hours later. Training sessions consisted of 5x5, with load set at 80% of 1RM, and a minimum of three minutes of rest between sets for the adapted bench press exercise [10,25]. MPV, MaxV, and Power indicators were measured during the 5x5 training, specifically in series "1" and "5", encompassing all five repetitions, under both caffeine supplementation and placebo (PL) conditions.

Statistical Analysis

Descriptive statistics and central tendency, mean ($\bar{X}$) $\pm$ standard deviation (SD), and 95% confidence interval (95% CI) measures were used. The Statistical Package for Social Sciences (SPSS), version 22.0 (IBM, Armonk, NY, USA) software was used to carry out the study statistics. The Shapiro-Wilk test was used to verify the normality of variables, taking into account the sample size, and analysis of variance (ANOVA) (Two-Way, 2x2, Moment and Condition) and post hoc Bonferroni repeated measures tests to assess performance between groups. Significance level of $p < 0.05$ was adopted. The effect size was determined by the "partial eta squared" (η^2_p) values,

considering low effect (≤ 0.05), medium effect (> 0.05 to 0.25), large effect (> 0.25 to 0.50), and very large effect values (> 0.50) [38,39].

RESULTS

No significant difference was found for training with Caffeine and Placebo, with 45% 1RM, in any of the evaluations, as shown in Figs. 2,3,4. In Fig. 2, no significant differences were observed in relation to conditions (Placebo and Caffeine), $F = 0.066$, $p = 0.801$; and moments (Before and After), $F = 2.498$, $p = 0.077$; and also in relation to interactions (Condition and Moment), $F = 1.832$, $p = 0.163$. In Fig. 3, no significant differences were observed in relation to conditions (Placebo and Caffeine), $F = 0.051$, $p = 0.829$; and moments (Before and After), $F = 3.244$, $p = 0.099$; and also relation to interactions (Condition and Moment), $F = 1.512$, $p = 0.230$. In Fig. 4, no significant differences were observed in relation to conditions (Placebo and Caffeine), $F = 0.510$, $p = 0.490$; and moments (Before and After), $F = 0.906$, $p = 0.449$; and also in relation to interactions (Condition and Moment), $F = 1.759$, $p = 0.174$. Table 2 and Fig. 5 show the MPV, MaxV, and Power results, evaluated in the first and last series of repetitions of training with 80% 1RM, with caffeine and placebo. The table 2 shows absolute and statistical values, while the Fig. 5 shows the kinetic indicators of variables.

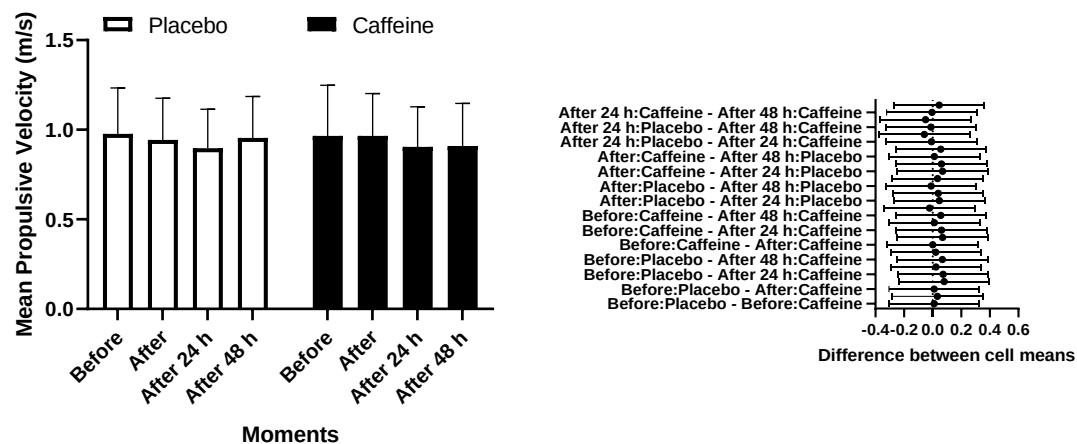


Fig. 2: Analysis of the Mean Propulsive Velocity (MPV) at a load of 45% of the 1 RM between pre, post, 24 and 48 hours post intervention, Placebo and with Caffeine with Mean Propulsive Velocity (MPV).

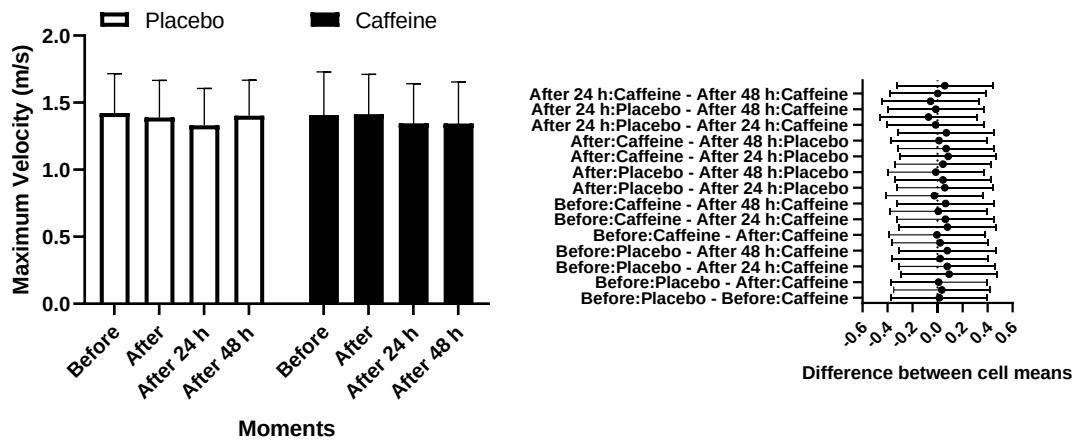


Fig. 3: Analysis of the Maximum Velocity (MaxV) at a load of 45% of the 1 RM between the pre, post, 24 and 48 hours post intervention, Placebo and Caffeine.

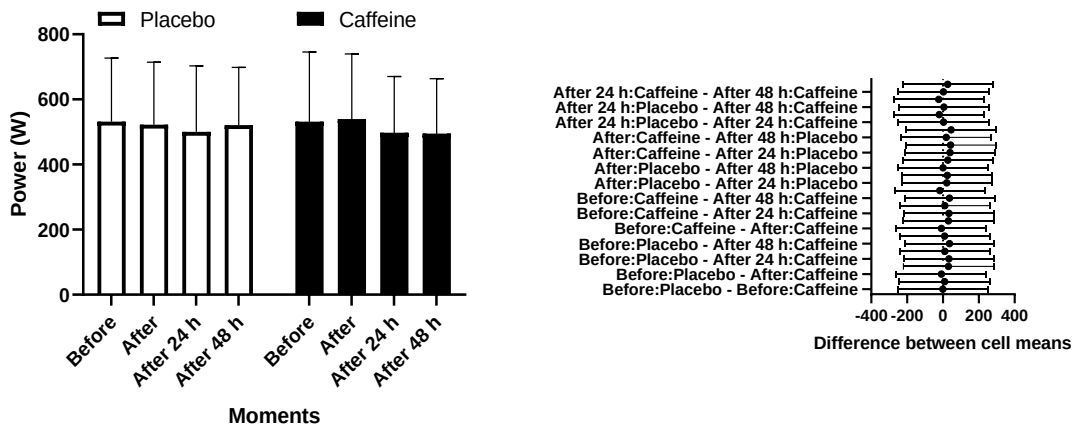
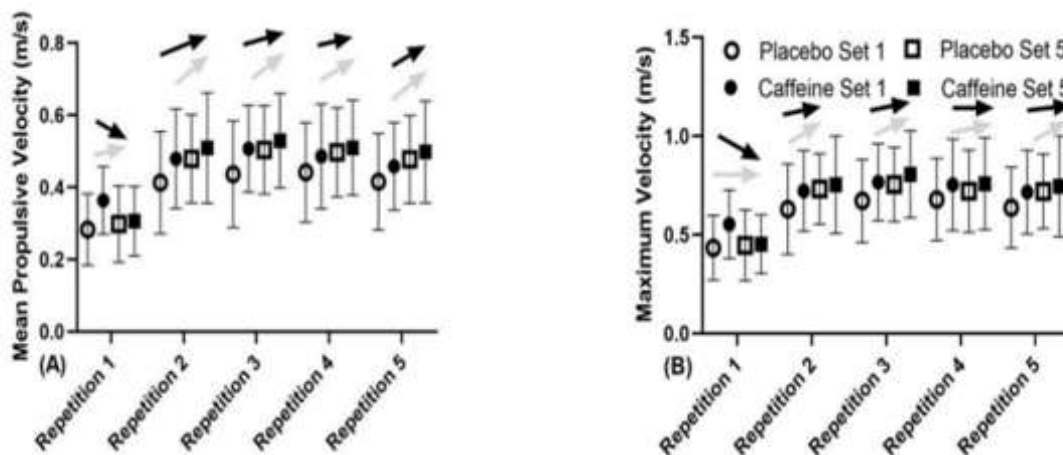


Fig. 4. Analysis of power at load of 45% of the 1RM between pre, post, 24, and 48 hours after intervention, with Placebo and Caffeine.



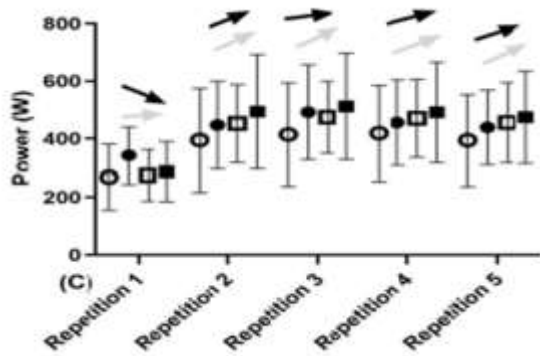


Fig. 5. Kinetics of the repetitions in Sep 1 and 5, with placebo and Caffeine. Absolute and statistical results are shown in Table 2. Arrows indicate the kinetics and the trend of values in each repetition, of (A) MPV, (B) MaxV, and (C) Power. Arrows represent the direction in recovery, gray arrows in relation to placebo and the black arrows in relation to caffeine. Although there were no significant differences, in terms of absolute values, the use of caffeine showed higher values.

Table 2. Mean propulsive Velocity, Maximum Velocity and Power, in the repetitions of the Sep 1, and Sep 5, in relation to the supplement, Placebo and Caffeine with 80% of 1RM (Mean $\pm$ SD, and CI 95%).

	MPV (m/s)	MV (m/s)	Power (W)
Set 1 / Rep 1	0.28 $\pm$ 0.10a,b,c,d,#	0.43 $\pm$ 0.16a,b,c,d,#	268.00 $\pm$ 113.12a,b,c,d,#
Placebo	(0.22-0.34)	(0.33-0.53)	(199.64 to 336.36)
Sep 1 / Rep 1	0.36 $\pm$ 0.09	0.55 $\pm$ 0.17e,f,g,h	342.00 $\pm$ 97.66e,f,g,h
Caffeine	(0.31-0.42)e,f,g,h	(0.45-0.66)	(282.98-401.02)
Sep 1 / Rep 2	0.41 $\pm$ 0.14a,##	0.63 $\pm$ 0.23a,##	395.15 $\pm$ 179.63y,##
Placebo	(0.33-0.50)	(0.49-0.77)	(286.60-503.71)
Sep 1 / Rep 2	0.48 $\pm$ 0.14e	0.72 $\pm$ 0.20e	447.08 $\pm$ 151.75e
Caffeine	(0.40-0.56)	(0.60-0.85)	(355.38 to 538.78)
Sep 1 / Rep 3	0.44 $\pm$ 0.15b,###	0.67 $\pm$ 0.21b,###	414.62 $\pm$ 178.29b,###
Placebo	(0.35-0.53)	(0.54-0.80)	(306.87-522.36)
Sep 1 / Rep 3	0.51 $\pm$ 0.12f	0.77 $\pm$ 0.20f	491.85 $\pm$ 164.39f
Caffeine	(0.43-0.58)	(0.65-0.88)	(392.51 to 591.18)
Sep 1 / Rep 4	0.44 $\pm$ 0.14*,c	0.68 $\pm$ 0.21c	419.23 $\pm$ 165.76c
Placebo	(0.36-0.52)	(0.55-0.80)	(319.06-519.40)
Sep 1 / Rep 4	0.49 $\pm$ 0.14*,g	0.75 $\pm$ 0.23g	455.85 $\pm$ 147.71g
Caffeine	(0.40-0.57)	(0.61-0.89)	(366.59-545.10)
Sep 1 / Rep 5	0.42 $\pm$ 0.13**,d,####	0.64 $\pm$ 0.21d	394.46 $\pm$ 158.46d,####
Placebo	(0.33-0.50)	(0.51-0.76)	(298.70-490.22)
Sep 1 / Rep 5	0.46 $\pm$ 0.12**,h	0.72 $\pm$ 0.21h	439.15 $\pm$ 129.27h
Caffeine	(0.39-0.53)	(0.59-0.84)	(361.04-517.27)
Sep 2 / Rep 1	0.30 $\pm$ 0.11*,i,j,k,l,#	0.45 $\pm$ 0.18*,i,j,k,l	274.23 $\pm$ 87.85i,j,k,l,#
Placebo	(0.23-0.36)	(0.34-0.55)	(221.14-327.32)
Sep 2 / Rep 1	0.31 $\pm$ 0.10***,m,n,o,p	0.45 $\pm$ 0.15*,m,n,o,p,#	287.31 $\pm$ 102.25m,n,o,p
Caffeine	(0.25-0.36)	(0.36-0.54)	(225.52-349.09)
Sep 2 / Rep 2	0.48 $\pm$ 0.12***,I,##	0.73 $\pm$ 0.18i,##	452.46 $\pm$ 135.50i,##
Placebo	(0.41-0.55)	(0.62-0.84)	(370.58 to 534.34)
Sep 2 / Rep 2	0.51 $\pm$ 0.15m	0.75 $\pm$ 0.25m	493.77 $\pm$ 198.40m
Caffeine	(0.42-0.60)	(0.60-0.90)	(373.88-613.66)
Sep 2 / Rep 3	0.50 $\pm$ 0.12j,###	0.75 $\pm$ 0.19j,###	474.46 $\pm$ 124.15j,###

Placebo	(0.43-0.58)		(0.64-0.87)		(399.44 to 549.49)	
Sep 2 / Rep 3	0.53±0.13n		0.81±0.22n		511.92±184.08n	
Caffeine	(0.45-0.61)		(0.67-0.94)		(400.68-623.16)	
Sep 2 / Rep 4	0.50±0.12k		0.72±0.21k		470.08±134.69k	
Placebo	(0.42-0.57)		(0.59-0.85)		(388.68-551.47)	
Sep 2 / Rep 4	0.51±0.13°		0.76±0.23°		491.38±174.60o	
Caffeine	(0.43-0.59)		(0.62-0.90)		(385.88-596.89)	
Sep 2 / Rep 5	0.48±0.12l,####		0.72±0.19l		455.38±138.88l,####	
Placebo	(0.40-0.55)		(0.61-0.83)		(371.46 to 539.31)	
Sep 2 / Rep 5	0.50±0.14p		0.75±0.26p		474.54±160.09p	
Caffeine	(0.41-0.58 m)		(0.59-0.90)		(377.80-571.28)	
P	*0.047	i 0.003	* 0.008	j 0.001	to 0.003	k 0.015
	**0.043	j<0.001	to 0.005	k 0.009	b 0.001	l 0.004
	***0.003	k,l 0.004	b,c<0.001	l 0.001	c<0.001	m 0.002
	a,b0.001	m,n<0.001	d 0.002	m,n<0.001	d 0.002	n,o,p<0.001
	c<0.001	o,p<0.001	e,f<0.001	o,p<0.001	e,f,g<0.001	# 0.005
	d 0.003	# 0.007	g 0.004	# 0.011	h 0.004	## 0.047
	e,f<0.001	##0.046	h 0.008	## 0.010	i 0.015	### 0.002
	g<0.001	###0.004	i 0.021	### 0.006	j<0.001	#### 0.007
	h 0.005	####0.006				
η ² p	*0.462		* 0.364		Letters 0.775	
	Letters 0.814		Letters 0.783		#0.271	
	#0.277		#0.248			

*p < 0.05 (ANOVA). η²p = partial eta square (small effect ≤0.05, medium effect 0.05 to 0.25, high effect 0.25 to 0.50, and very high effect >0.50). (A) Intraclass (Moment); (B) Intraclass (Condition); and (C) Interclass differences. Symbols *, # (intraclass), or Letters (interclass), refer to moments, conditions, or interactions where differences occurred. Homologous symbols where differences occurred, symbols indicate the conditions (e.g., # - #, ## - ##..., on intraclass differences) or moments when they occurred (e.g., * - *, ** - **..., on intraclass differences). Homologous letters a - a; b - b..., indicate interclass differences. SD, standard deviation; MPV, Mean Propulsive Velocity; Maximum Velocity (MaxV) and Power; ANOVA, analysis of variance.

Discussion

This study aimed to assess the effects of pre-workout with caffeine supplementation on powerlifting athletes before, during, and after training sessions. Results revealed no significant differences in MPV, MaxV, and Power parameters at intensity of 45% of 1RM. These findings suggest that caffeine supplementation did not impact post-training fatigue, as indicated by velocity indicators at 45% of 1RM, which corresponds to MPV of approximately 1.0 m/s. It is important to note that MPV is commonly used as indicator of training control and fatigue [40], as it is associated with the accumulation of metabolites in the bloodstream, such as lactate and ammonia. The use of MPV as a reliable indicator for assessing power has been extensively described in literature since 2010 [40].

Although strength training for this population typically involves submaximal to maximal loads ranging from 80% to 100% of 1RM, with sets consisting of 3 to 5 repetitions [41], it appears that velocity during Bench Press exercises starts to decline when approaching one-third of the total number of repetitions at load from 60% to 75% of 1RM [42]. Therefore, for higher loads, such as those applied in the training system at 80% of 1RM, the fatigue process might have been altered, taking into account the rest periods allowed between sets. Aidar and Fraga [13] found that training session (5x5) at 80% to 90% of 1RM in Paralympic Powerlifting athletes did not induce significant oxidative stress or fatigue. Conversely, another study assessing the use of ibuprofen at different levels of Paralympic weightlifting training reported that this type of training tends to be intense and therefore promotes fatigue. The results indicated that training intensity can influence the immune system, and when combined with the use of ibuprofen, it contributes to recovery and improves the immune system response, particularly in highly trained athletes [14]. Moreover, it has been suggested that caffeine intake can lead to increase of approximately 3–4% in maximum strength. This might explain why variations observed in this study were not substantial.

In this study, evaluations of athletes during training sessions were conducted, which involved performing 5 sets of 5 repetitions (5x5). This study specifically focused on assessing the dynamic strength indicators during the first and fifth series of repetitions. Results revealed that in both series 1 and 5, athletes who received caffeine supplementation demonstrated higher MPV, MaxV, and Power values compared to those who received placebo. These findings suggest that caffeine supplementation tends to enhance training intensity when compared to training with placebo.

Our findings are consistent with previous studies that have examined the effects of caffeine supplementation on muscle strength. For instance, a study evaluating upper body strength (bench press) with caffeine dose of $8.0 \text{ mg} \cdot \text{kg} \text{ bw}^{-1}$ found that plasma calcium release was significantly higher under the caffeine condition ($100.1 \pm 1.9 \text{ kg}$) compared to the placebo condition ($94.2 \pm 2.5 \text{ kg}$, $p < 0.001$). The researchers concluded that caffeine supplementation enhanced plasma calcium release and optimized bench press muscle strength [42].

Our results are also consistent with research indicating that the effective caffeine dose for performance enhancement may be dependent on the load magnitude. Specifically, doses of 3.0 mg/kg have been shown to positively impact velocity during low-load exercises, while dose of 9.0 mg/kg , the same dose applied in this study, tends to have a positive effect on velocity during high-load exercises [43].

Despite the observed improvements associated with caffeine use, a study has suggested that the moment of ingestion may play a crucial role. In this particular study, which examined caffeine supplementation in the morning and afternoon, 12 trained men, similar to our sample, were evaluated. The assessment focused on the bench press exercise, with incremental loads of 25%, 50%, 75%, and 90% of 1RM, specifically measuring maximum velocity. Participants received either 6.0 mg/kg of caffeine or placebo.

Interestingly, this study revealed that tympanic temperature was lower in the morning when participants received placebo compared to caffeine. Consequently, caffeine supplementation facilitated muscle contraction speed maintenance in the morning across all load intensities. However, when caffeine supplementation occurred outside the morning period, its impact on performance was found to be minimal, with the possibility of experiencing side effects at non-optimal supplementation moments [44].

Furthermore, reinforcing the notion that caffeine may not have a positive impact on the performance of Paralympic athletes, a study took an intriguing approach by providing placebo supplementation to athletes while informing them that it was caffeine. Surprisingly, this deceptive placebo intervention appeared to enhance the responses in explosive movements among para-powerlifting athletes, particularly when performed at lower intensities. These findings suggest that factors beyond the actual physiological effects of caffeine, such as psychological and motivational factors, might contribute to the observed performance improvements in this population [45].

Regarding power, this study showed results and kinetics with caffeine supplementation consistent with those of MPV and MaxV. However, in contrast to our findings, other research indicated that acute caffeine doses administered before exercise did not lead to power output and bar velocity improvements, especially in athletes who regularly consume caffeine. These conflicting results highlight the complexity of the relationship between caffeine intake and power performance, suggesting that individual factors and habitual caffeine use may play a role in determining the ergogenic effects of caffeine in this context [36].

In line with our findings, a study investigating the effectiveness of caffeine doses of 3.0 and 6.0 mg/kg on barbell velocity during the bench press exercise, specifically in individuals who regularly consume caffeine, reported that 6.0 mg/kg of caffeine tended to enhance power output [46]. Additionally, another study demonstrated improvements in acute caffeine supplementation prior to the bench press exercise, showing an increase in average power and velocity among participants with limited caffeine supplementation history [47]. Conversely, the use of 3 mg/kg/BM of caffeine before

exercise did not have an effect on 1RM in a four-week bench press strength training program. Interestingly, the placebo group showed improvements in maximum velocity at 60% and 70% of 1RM, while the caffeine group demonstrated enhanced maximum velocity across intensities ranging from 10% to 30–100% of 1RM ($p < 0.05$) [48].

It is important to acknowledge that further research is necessary to fully understand both the positive and negative effects of caffeine supplementation in Paralympic athletes. This is particularly relevant as this population requires a more comprehensive examination of not only performance aspects but also the potential impact on their specific impairments. However, a previous study has indicated that caffeine supplementation is a safe option from the hemodynamic perspective [10].

Moreover, despite the results obtained in this study, there are some limitations that should be highlighted. Firstly, there was lack of control over participants' dietary intake and sleep patterns during the recovery period. This could have potentially influenced recovery and had an impact on training outcomes. Additionally, the study did not monitor the athletes' habitual caffeine consumption or investigate other factors that might have affected the results. Furthermore, this study focused on a broad range of eligible athletes for the sport without differentiating them based on the type of disability, which could have influenced the outcomes.

Conclusions

In conclusion, this study found that caffeine supplementation at a dose of 9.0 mg/kg did not have an impact on velocity and recovery when assessed at velocity of 1.0 m/s and 45% of 1RM. Therefore, the results suggest that caffeine supplementation does not interfere with the performance and recovery of para-powerlifting athletes during a training session. However, when examining the 5x5 training session and evaluating the first and last series, where the execution velocity was monitored for five repetitions in each series, with loads at 80% of 1RM, caffeine demonstrated significant benefit in maintaining velocity and power when compared to placebo.

Regarding the practical implications, the results suggest that supplementation with 9.0 mg/kg of caffeine can be a beneficial ergogenic strategy, enabling athletes to sustain training intensity throughout the session, even when using heavier loads, as observed in the training of parapowerlifting athletes.

Author Contributions

Conceptualization, JLM and FJA; methodology, FF, and GB; validation, MGM, and GGV; formal analysis, FJA, and FMC; investigation, JLM, FJA, and MGM; data curation, SC, and GG; writing—original draft preparation, JLM; writing—review and editing, FJA, GGV, FMC, SC, GG, FF, MGM, and GB; visualization, GB, and FMC; supervision, SC and GG; project administration, FJA. All authors read and approved the final manuscript. All authors have read and agreed to the published version of the manuscript.

6. ESTUDO 2: EFEITOS DA SUPLEMENTAÇÃO AGUDA DE CAFEÍNA NO TREINAMENTO PARALÍMPICO DE LEVANTAMENTO DE PESO SOBRE A FORÇA ESTÁTICA E OS PARÂMETROS DE ESTRESSE OXIDATIVO

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Effects of acute caffeine supplementation in Paralympic powerlifting training on static strength and oxidative stress parameters

Abstract

Paralympic Powerlifting is a maximal strength sport that requires high training intensity and efficient recovery to maintain performance and prevent injury. Intense training can induce oxidative stress, potentially compromising performance and physiological adaptation. Caffeine (CA) is a widely used ergogenic aid with potential to enhance muscle strength and reduce oxidative damage. This study investigated the acute effects of CA supplementation (9 mg/kg) on static strength indicators and oxidative stress markers in 14 male Paralympic powerlifting athletes. The protocol included two conditions (CA and placebo), with athletes performing 5 sets of 5 repetitions at 80% of 1RM in the adapted bench press. Strength variables (maximal isometric force, 1-second peak force, and force variability) and biomarkers (TBARS/MDA, total antioxidant capacity, sulfhydryl groups, and uric acid) were analyzed. CA supplementation significantly increased maximal isometric strength, improved 1-second peak force, and reduced variability, suggesting enhanced motor unit recruitment. However, CA also led to increased oxidative stress, as evidenced by elevated TBARS levels, reduced antioxidant capacity, decreased sulfhydryl groups, and higher uric acid concentrations post-exercise. In conclusion, while CA acutely improves neuromuscular performance, it does not attenuate—and may even exacerbate—exercise-induced oxidative stress. Therefore, its use in strength sports should be approached with caution.

Keywords: Caffeine, Oxidative stress, Strength training, Paralympic weightlifting

INTRODUCTION

Paralympic Powerlifting (PP) is a maximal strength sport characterized by high-intensity training [1–3]. Due to the demands placed on physical performance and training intensity, optimal recovery and strength improvement are essential components [4,5]. In this context, the use of ergogenic aids to enhance strength and facilitate recovery has been increasingly explored. Inadequate recovery can compromise subsequent training sessions by reducing training volume and intensity, and may also increase the risk of injury [5–8]. Therefore, improving both strength and post-training recovery is crucial, as it can support performance enhancement and progression, even under conditions of intense and prolonged training, particularly among elite athletes [9]. Intense physical exercise induces oxidative stress, as high-intensity activity stimulates the production of reactive oxygen species (ROS) [10–12]. When ROS levels become unbalanced, they can cause tissue damage and negatively affect cellular structures such as organelles, nucleic acids, lipids, and proteins, posing significant health risks [6,7,10].

In this regard, caffeine (CA) may attenuate exercise-induced oxidative stress by neutralizing reactive oxygen species (ROS) and scavenging free radicals [13,14], thereby contributing to the prevention or reduction of muscle damage [15]. CA administration has been shown to reduce malondialdehyde (MDA) levels and improve liver function in a dose-dependent manner [16]. Furthermore, caffeine supplementation can increase neuronal strength and excitability by modulating intramuscular calcium dynamics, stimulating calcium (Ca^{2+}) release from the sarcoplasmic reticulum and increasing motor unit recruitment [17,18]. Physiologically, this occurs via binding to ryanodine receptors, antagonism of adenosine receptors and modulation of voltage-dependent calcium channels (VOCCs) [18].

Additionally, it may promote lipolysis, contributing to the sparing of muscle glycogen [16,19]. However, the effects of caffeine on oxidative stress markers and strength following resistance exercise in athletes remain conflicting [14,20–24]. On one hand, some studies have reported improved performance without affecting oxidative stress levels [20,22,24]. On the other hand, minimal reductions in oxidative stress have been observed, with increases only in glutathione levels, without significant changes in other ROS-related parameters [21]. Other studies have reported a more pronounced reduction in oxidative stress following caffeine supplementation [14,23].

Considering the lack of consensus on the effects of CA in strength training, the aim of this study was to evaluate the acute supplementation of 9 mg/g of CA during Paralympic

weightlifting training on indicators of static strength and post-exercise oxidative stress, after training at 80% of one repetition maximum (1RM). Since low doses (3 mg/kg) improve speed at light loads (25-50% of 1RM), while high loads (such as 90% of 1RM) and high-intensity activities require higher doses (6-9 mg/kg) to optimize power[25]. The 9 mg/kg protocol stands out as a safe strategy to maximize performance in adapted strength sports[26]. Therefore, our hypothesis was that CA supplementation would increase muscle strength and reduce post-exercise oxidative stress.

MATERIALS AND METHODS

Study design

This study, outlined in Figure 1, was conducted over a period of three weeks. In the first week, participants were familiarized with the adapted bench press, followed by the one-repetition maximum (1RM) test. During the second and third weeks, participants received either anhydrous caffeine (CA) at a dose of 9 mg/kg or a placebo (PLA), administered one hour prior to training[25–28]. The order of conditions (CA or PLA) was randomized, with 50% of participants allocated to each condition in a crossover design.

In the third week, the supplementation was switched: participants who had received CA in the second week were given PLA, and those who had received PLA were given CA. These sessions consisted of five sets of five repetition maximum (5X5) at 80% of one repetition maximum (1-RM), with three to five minutes of rest between sets described by Austin and Mann [29]. During this period, data were collected on static strength: Maximum Static Strength (MIF), Isometric Strength Variability (Variability), Maximum Force in One Second (MIF 1s), and markers of oxidative stress, such as Thiobarbituric Acid Reactive Substances (TBARS)/Malondialdehyde (MDA), Total Antioxidant Capacity (TAC), Sulfhydryl Groups (SH), and Uric Acid (UA).

All collections were performed between 9:00 am and 12:00 pm. Blood collection and all measurements occurred 60 minutes before training, immediately after, 24 hours after, and 48 hours after the session. To ensure the impartiality of the results, the athletes underwent a restriction period during the three weeks of testing. During this time, the consumption of alcohol, tobacco, medications, and any supplements or ergogenic substances was prohibited. The control included the strict exclusion of caffeine sources, such as coffee, teas, cola soft drinks, chocolate, and energy drinks, a condition that was individually validated through interviews before the start of the interventions. Before starting the training protocol, the athletes performed an upper-limb

warm-up consisting of three exercises: (elbow extension on the pulley, dumbbell shoulder rotations, and dumbbell shoulder abduction. Three sets of 10 to 20 reps (1-RM) were performed, totaling approximately 10 minutes of warm-up [5–7]. Next, a specific warm-up was performed with the bench press exercise, using 30% of 1-RM. This warm-up consisted of 10 slow repetitions (3-second eccentric phase and 1-second concentric phase) and 10 fast repetitions (1-second eccentric and concentric phases). During this phase, the athletes were verbally encouraged to perform to their fullest.

Week 1 Familiarization and testing		Warm-up	Test 1 RM	rest	
					
Week 2 and 3 50% Placebo or 50% caffeine supplementation	Before training: MIF, Variability, MIF 1s, TBARS/MDA, TAC, SH, AU	Warm-up	Training 80% 1RM  5x5	Immediately after training, 24h and 48h: MIF, Variability, MIF 1s, TBARS/MDA, TAC, SH, AU	rest

Figure 1: Experimental design of the study.

Legend: MIF (Maximum static force), Variability (variability of isometric force), MIF 1s (maximum force in one second), TBARS (Thiobarbituric Acid Reactive Substances)/ MDA (Malondialdehyde), TAC (Total Antioxidant Capacity), SH (Sulphydryl Groups) and AU (Uric acid).

Sample

The sample consisted of 14 male Powerlifting (PP) athletes who occupied the top 10 national rankings in their respective weight classes. The athletes presented the following physical disabilities: two had left leg atrophy, three had left leg amputations, two had right leg amputations, one had a right leg injury resulting in loss of muscle function and tone, and one had a spinal cord injury caused by schistosomiasis. Finally, three athletes had polio sequelae in the lower limbs, and two others were diagnosed with arthrogryposis.

The inclusion criteria were: being clinically stable, having at least 18 months of experience in the sport, actively participating in sports competitions, and being eligible for Paralympic sports.

The exclusion criteria were: individuals with cardiorespiratory diseases, pre-existing cardiac conditions, or using any type of ergogenic aid. Women were also excluded from participation due to the potential interference of hormonal fluctuations and the possibility of interaction with hormonal supplements, variables that could compromise the reliability of the effects attributed to the intervention.

The sample size was determined using the open-source software G*Power® (version 3.0; Berlin, Germany), adopting an “F family statistic (ANOVA)” with a standard $\alpha < 0.05$, $\beta = 0.80$, for the rate of force development (RFD) in Powerlifting athletes. This allowed for an estimated sample power of 0.80, with a minimum of eight individuals per group, suggesting that the sample is adequate. The observed power in blood indicators and static strength indicators was greater than 0.80. Corroborating this, other studies that worked with similar indicators and with a population of Paralympic athletes used similar samples [26,27,30,31]. All participants participated in the study voluntarily. The ethical integrity of the study was ensured by approval from the Research Ethics Committee of the Federal University of Sergipe under CAAE number 67953622.7.0000.5546 and opinion number 6,523,247, dated November 22, 2023. The study was conducted in accordance with the ethical principles described in the Declaration of Helsinki (1964, revised in 2013) of the World Medical Association, as well as Resolution 466/2012 of the National Research Ethics Committee (CONEP) of the National Health Council. The sample characteristics are shown in Table 1.

Table 1: Characteristics of the study participants (n = 14)

Features	(Mean $\pm$ SD)
Age (years)	32,4 $\pm$ 8,5
Body mass (kg)	81,7 $\pm$ 21,2
Experience (years)	3,1 $\pm$ 1,0
Systolic blood pressure (mmHg)	126,4 $\pm$ 15,2
Diastolic blood pressure (mmHg)	75,5 $\pm$ 11,9
1RM test (bench press) (kg)	126,9 $\pm$ 41,2
1RM test/body mass (kg)	1,6 $\pm$ 0,4

1RM: one repetition maximum.

Instruments

A Michetti brand digital wheelchair-safe electronic scale, MicWelchair (Michetti, São Paulo, SP, Brazil), was used to measure participants' body mass.

The exercise was performed on an official bench (2.10 m long, Eleiko, Halmstad, Sweden) and Olympic barbell (220 cm total length, 20 kg weight) approved by the International Paralympic Committee (IPC) (15,16).

Upper Limb Muscle Strength

Static strength, maximal isometric strength (MIF), variability, and maximal average force in one second (FMI1S) were measured using a Chronojump force sensor (Chronojump®, BoscoSystem, Madrid, Spain). It was attached to a bench press using Spider HMS carabiners (Simond, Chamonix, France), which had a breaking load of 21 kN and are certified for climbing by the Union International des Associations d'Alpinisme (UIAA). A steel chain with a breaking load of 2,300 kg was used to attach the load cell to the bench. During the assessment, the volunteers were instructed to perform a single elbow extension movement with maximum force and speed, followed by relaxation.

Anhydrous Caffeine (CA) and Placebo (PLA)

The CA (9.0 mg/kg) and PLA capsules were compounded in a specialized pharmacy to ensure physical uniformity (color, size, and volume). This rigorous standardization guaranteed the effectiveness of the blinding, preventing athletes from identifying the ingested substance through sensory perception.

Biochemical analysis

Blood samples (10 mL) were collected from the antecubital vein of the participants' forearm, a procedure performed by a properly qualified nursing technician. Immediately after collection, the blood was transferred to tubes containing the anticoagulant, ethylenediaminetetraacetic acid (EDTA). The samples were initially centrifuged at 3000×g for 10 minutes. After this process, the plasma and serum were separated and aliquoted, then kept refrigerated at 4°C for subsequent analysis. An additional fraction of the blood was processed by centrifugation at 800×g for 15 minutes at 4°C. The serum obtained was then stored in an ultrafreezer at -80°C for preservation for future analysis.

Tissue Reactive Species (TBARS) Marker Analysis

To quantify lipid oxidation, we determined TBARS, following the method of Lapenna et al. (17). First, 200 μL of each blood sample was mixed with 400 μL of a solution containing 15% trichloroacetic acid (TCA), 0.25 N HCl, 0.375% thiobarbituric acid (TBA), 2.5 mM butylated hydroxytoluene (BHT), and 40 μL of 8.1% sodium dodecyl sulfate (SDS). The mixture was then heated at 95 °C for 30 minutes in a dry bath incubator. To prevent lipid peroxidation during heating, the pH of the solution was adjusted to 0.9 using concentrated HCl, and BHT was added as an antioxidant. After cooling the solution to room temperature, 4 mL of butanol was added, and the samples were centrifuged at $800 \times g$ for 15 minutes at approximately 4 °C. Finally, the absorbance of the supernatant was measured at 532 nm, using a molar extinction coefficient of $1.54 \times 10^5 \text{ M}^{-1} \cdot \text{cm}^{-1}$. TBARS results were expressed as malondialdehyde equivalents (MDA), in nmol MDAeq/mL of plasma.

Total Sulfhydryl (SH) Analysis

Plasma antioxidant activity was assessed by quantifying sulfhydryl groups (SH), following the method described by Faure and Lafond (18). Briefly, 50 μL aliquots of plasma samples were mixed with 1 mL of Tris-EDTA buffer (pH 8.2). The first absorbance (A1) was measured at 412 nm. Subsequently, the samples were transferred to test tubes containing 20 μL of DTNB (10 mM), diluted in methanol (4 mg/mL), and kept in the dark at room temperature. After 15 minutes, a second absorbance (A2) was measured at 412 nm. The concentration of sulfhydryl groups was calculated using the following equation: $(A2 - A1 - B) \times 1.57 \text{ mM} \times 1000$, and the results were expressed as nmol/mg.

Analysis of Enzymatic Antioxidant Activity

Total antioxidant capacity (TAC) quantification was performed using a commercial kit (Labtest®, Minas Gerais, Brazil), strictly following the manufacturer's instructions. To construct the calibration curve, an aqueous standard solution of Trolox (2.0 mmol/L) was used, prepared at different concentrations: 0.1; 0.25; 0.5; 1.0 and 1.5 mmol/L. Aliquots of 20 μL of the standard solutions were added to 800 μL of reagent R1. After a stabilization period of 3 minutes at room

temperature (RT), 80 μ L of reagent R2 were added. The mixture was incubated for 5 minutes, also at RT, and then the absorbance at 620 nm was read. Based on the readings obtained for the different concentrations of Trolox, a standard curve was generated, from which the TAC results were expressed in millimoles equivalent of Trolox per liter (mmol Trolox eq/L).

Uric Acid Analysis

Uric acid (UA) quantification was performed by an enzymatic method (UV, uricase-peroxidase), using a commercial kit (Labtest®, Minas Gerais, Brazil). Aliquots of 20 μ L of plasma from each athlete were homogenized with specific reagents, under a controlled temperature of $37 \pm 0.2^\circ\text{C}$. Absorbances were determined in a spectrophotometer, with reading at 540 nm.

Statistical Analysis

Descriptive statistics were reported as mean ($\bar{X}$) $\pm$ standard deviation (SD), along with 95% confidence intervals (95% CI). The normality of the data was assessed using the Shapiro–Wilk test, considering the sample size.

To compare performance across different four time points (Pre $\times$ Post $\times$ Post 24h $\times$ Post 48h) and two conditions (Placebo and Caffeine), the assumptions for applying a two-way ANOVA for repeated measures. When significant main or interaction effects were found, Bonferroni post hoc tests were used to identify specific differences between time points. Effect size for the ANOVA was evaluated using partial eta squared (η^2_p), with the following thresholds: small effect (≤ 0.05), medium effect (0.05 to 0.25), large effect (0.25 to 0.50), very large effect (> 0.50) [36]. All statistical analyses were conducted using SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA), adopting a significance level of $p < 0.05$.

RESULTS

Table 2 shows the results of Oxidative Stress, and Table 3 shows the results of static strength.

Table 2: Oxidative Stress (Mean $\pm$ SD; 95% CI) at different time points with Placebo and Caffeine supplementation in Paralympic Powerlifting

MDA/TBARS (nmol)	TAC (nmol)	SH (nmol)	UA (mg/dL)
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Before Placebo	182.96±62.49 (146.88-219.04)	103.09±5.23 a (100.07-106.11)	158.91±110.04 a (95.37-222.44)	5.34±.98 a (4.77-5.90)
Caffeine Before	182.01±62.30 (146.04-217.98)	103.14±5.28 b,c (100.09-106.19)	158.03±110.16 (94.42-221.63)	5.33±1.16 b,c,d (4.66-6.00)
After Placebo	167.07±47.66 a (139.55-194.58)	101.78±5.73 d (98.47-105.09)	433.66±318.76 a,b,c (249.61-617.70)	5.78±1.28 e (5.04-6.51)
After Caffeine	215.77±60.90 a (180.61-250.93)	94.26±7.08 b,d (90.18-98.35)	230.23±108.10 c (167.81-292.64)	6.95±1.58 b,e (6.04-7.86)
24h Placebo	180.15±65.68 (142.23-218.07)	99.63±4.47 (97.05-102.21)	230.68±129.27 (156.04-305.32)	5.88±1.17 (5.21-6.56)
24h Caffeine	194.71±87.60 (144.13-245.29)	97.54±5.91 (94.13-100.95)	185.37±187.07 (77.36-293.38)	6.75±1.59 c (5.83-7.67)
48h Placebo	175.51±66.58 (137.07-213.95)	96.78±5.02 a (93.89-99.68)	182.12±105.30 b (121.32-242.92)	6.40±1.04 a (5.80-7.00)
48h Caffeine	175.51±37.22 (154.02-197.01)	95.42±3.20 c (93.57-97.26)	166.76±108.43 (104.15-229.36)	7.13±1.69d (6.15-8.10)
<i>P</i>	"a" <i>p</i> =0.003#	"a" <i>p</i> =0.040* "b" <i>p</i> =0.009* "c" <i>p</i> <0.001* "d" <i>p</i> =0.001#	"a" <i>p</i> =0.034* "b" <i>p</i> =0.033* "c" <i>p</i> =0.020#	"a" <i>p</i> =0.002* "b" <i>p</i> =0.001* "c" <i>p</i> =0.011* "d" <i>p</i> =0.013* "e" <i>p</i> <0.001#
<i>F</i>	4.773 #	5.591* 19.617 #	8.072* 8.155#	7.841* 15.012#
η^2p	0.269#	0.301* 0.601#	0.383* 0.385#	0.376* 0.536#

$p \leq 0.05$ (two way ANOVA and Bonferroni Post Hoc). η^2p = partial eta square (small effect ≤ 0.05 , medium effect 0.05 to 0.25, high effect 0.25 to 0.50, and very high effect > 0.50). The repeated letters (a-a, b-b), represent where the statistical differences are.

A difference was observed between the PLA and CA groups for TBARS ($p=0.003$).

For TAC, differences were observed between the time points: PLA before training and PLA 48 hours later ($p=0.040$), CA before training and CA after training ($p=0.009$), CA before training and 48 hours after ($p<0.001$), and PLA after training and CA after training ($p=0.001$).

In the sulfhydryl group analysis, differences were identified in the PLA time points before and after training ($p=0.034$). There were differences in the PLA time points after training and 48 hours later ($p=0.033$), PLA after and CA after training ($p=0.020$).

For Uric Acid, differences were observed in the following time points: PLA before training and 48 hours later ($p=0.002$), before training with CA and after training with CA ($p=0.001$), before training with CA and 24 hours later ($p=0.011$), before training with CA and 48 hours later ($p=0.013$), and PLA after training and CA after training ($p<0.001$).

Table 3. Assessment of static strength (maximal isometric force (MIF), Variability and 1s MIF) (Mean $\pm$ SD; 95% CI) at different times with Placebo and Caffeine supplementation in Paralympic Powerlifting

Warm-up	MIF (N) X $\pm$ DP (IC 95%)	Variability (%) X $\pm$ DP (IC 95%)	AverageMaxF 1s (N) X $\pm$ DP (IC 95%)
Before Placebo	826.61 $\pm$ 252.46 a (680.85-972.38)	0.84 $\pm$ 0.28 a (0.68-1.00)	787.04 $\pm$ 247.00 a (644.42-929.65)
Caffeine Before	830.79 $\pm$ 268.51 (675.76-985.82)	0.81 $\pm$ 0.50 c (0.52-1.10)	805.80 $\pm$ 276.13 (646.37-965.23)
After Placebo	661.39 $\pm$ 263.26 a,b,d (509.39-813.39)	0.93 $\pm$ 0.44 b,f (0.68-1.19)	637.03 $\pm$ 256.48 a,b,d (488.94-785.12)
After Caffeine	758.59 $\pm$ 227.56 d (627.20-889.97)	0.70 $\pm$ 0.35 d,e,f (0.50-0.91)	728.36 $\pm$ 222.39 d (599.95-856.76)
24 Horas Placebo	693.86 $\pm$ 247.43 c (551.00-836.72)	1.30 $\pm$ 0.61 (0.95-1.65)	663.80 $\pm$ 244.65 c (522.54-805.06)
24 Horas Caffeine	749.94 $\pm$ 202.79 (632.85-867.03)	1.15 $\pm$ 0.56 c,d (0.83-1.47)	706.42 $\pm$ 197.83 (592.20-820.65)
48 Horas Placebo	802.28 $\pm$ 234.61 b,c (666.82-937.74)	1.38 $\pm$ 0.67 a,b (1.00-1.77)	770.42 $\pm$ 228.46 b,c (638.51-902.33)
48 Horas Caffeine	763.35 $\pm$ 265.75 (609.91-916.79)	1.29 $\pm$ 0.37 e (1.08-1.51)	734.74 $\pm$ 240.63 (595.80-873.67)
P	“a” $p=0.015^*$ “b” $p=0.008^*$ “c” $p=0.002^*$ “d” $p=0.005^{\#}$	“a” $p=0.039^*$ “b” $p=0.021^*$ “c” $p=0.016^*$ “d” $p=0.003^*$ “e” $p=0.004^*$ “f” $p=0.014^{\#}$	“a” $p=0.025^*$ “b” $p=0.009^*$ “c” $p=0.001^*$ “d” $p=0.011^{\#}$
η^2p	0.424* 0.212 $\#$	0.645* 0.096 $\#$	0.414 0.181 $\#$

$p < 0.05$ (two-way ANOVA, and Bonferroni post hoc, ES η^2p). A vs B; A vs C and B vs C (Cohen t and ES test “d”). η^2p = partial eta square (small effect ≤ 0.05 , medium effect 0.05 to 0.25, high effect 0.25 to 0.50, and very high effect > 0.50).
 * Intraclass, # Interclass. The re-peated letters (a-a, b-b), represent where the statistical differences are.

The results of the static strength evaluation for MIF showed a difference for the PLA condition before and after training ($p=0.015$), PLA after training and 48 hours after ($p=0.008$), PLA 24 hours after training and 48 hours after training ($p=0.002$), PLA after training and CA after training ($p=0.005$). For variability, there was a difference for the conditions: PLA before training and 48 hours after training ($p=0.039$), CA before training and 24 hours after training ($p=0.021$), PLA after training and 48 hours after training ($p=0.016$), CA after training and 24 hours after training ($p=0.003$), CA after training and 48 hours after training ($p=0.004$), and PLA after training and CA after training ($p=0.014$).

For maximum strength in one second, there were differences in the conditions PLA before training and after training ($p=0.025$), PLA after training and 48 hours after training ($p=0.025$), PLA 24 hours after training and 48 hours after training ($p=0.009$), PLA 24 hours after training and 48 hours after training ($p=0.001$), PLA after training and CA after training ($p=0.011$)

DISCUSSION

This study aimed to evaluate the effects of acute caffeine supplementation during Paralympic powerlifting training on static strength indicators and oxidative stress following exercise performed at 80% of one-repetition maximum (1RM). Although the participants were unaware of the substance administered and were not prompted to identify the contents of the supplement, our findings demonstrated significantly higher levels of MDA/TBARS immediately after exercise with CA supplementation compared to PLA. This increase in lipid peroxidation indicates that exercise-induced oxidative stress was elevated under the influence of CA. In this context, the ergogenic action of caffeine on the central nervous system should be considered, which, by improving neuromuscular therapy and muscle contractility, can intensify oxidative stress due to the increased flow of electrons in the mitochondrial chain during exertion[19,37].

These results are consistent with the findings of Olcina et al. [19], who reported an increase in plasma MDA levels following caffeine supplementation during a cycling exercise protocol. However, Mahdavi et al. [24] found no significant effect of CA supplementation on MDA levels after a supramaximal test in basketball players. Oxidative stress is known to represent an imbalance in redox homeostasis, resulting either from an excessive increase in reactive oxygen species ROS

production or a reduction in the body's endogenous antioxidant capacity [5–7]. Malondialdehyde, one of the primary byproducts generated during lipid peroxidation induced by ROS, is widely used as a biomarker for assessing oxidative damage to cellular lipid membranes [38].

The elevated MDA levels observed may be associated with CA role as an adenosine receptor antagonist, which results in greater activation of the sympathetic nervous system and increased catecholamine release [39]. Additionally, CA can enhance Ca^{2+} release via ryanodine receptors and the sarcoplasmic reticulum [17,40], while also inhibiting its reuptake [40], and intensifying energy turnover [39]. These physiological mechanisms—particularly under conditions of high metabolic demand—may lead to increased ROS production, thereby contributing to lipid peroxidation [39]. This supports the notion that intense physical exercise induces oxidative stress, primarily due to the elevated generation of ROS in the human body [5–7]. The literature suggests that higher doses of CA may have a significant effect on MDA production [24], with some studies reporting a dose-dependent reduction in MDA levels [16]. However, previous studies using a moderate dose of 5 mg/kg of CA have shown conflicting results: while some have reported an increase in MDA levels post-exercise—indicating intensified lipid peroxidation [41]—others found no significant changes, possibly due to the lower dosage used [24].

The increase in cytosolic Ca^{2+} concentration promotes its uptake by the mitochondrial matrix, stimulating metabolism and ATP synthesis. Although this mechanism optimizes muscle contractility, it also increases the production of reactive oxygen species ROS. It is important to note that these effects are dose-dependent, with only higher doses of caffeine shown to significantly increase Ca^{2+} release [17,20,40]. In our study, total antioxidant capacity (TAC) was lower immediately after exercise in the CA condition compared to PLA, indicating a reduction in antioxidant defense following caffeine supplementation. This decrease in TAC was observed at pre-exercise, immediate post-exercise, and 48 hours post-exercise time points in the caffeine condition. Caffeine supplementation can enhance the production of ROS during exercise, an effect mediated by its ergogenic properties, tissue mechanical stress, and the high muscle tension generated. This metabolic scenario demands greater efficiency from endogenous antioxidant systems to mitigate excess free radicals and maintain redox homeostasis [41].

When analyzing plasma sulfhydryl group concentrations, a reduction was observed in the CA post-exercise group compared to the PLA post-exercise group, suggesting that high-dose CA supplementation contributed to exercise-induced oxidative stress. Thiols, compounds containing SH groups in their structure, play a crucial role as antioxidant agents, primarily by donating electrons to

neutralize ROS, thereby helping to maintain redox homeostasis in the intracellular environment [42]. A decrease in total thiol levels has been associated with increased lipid peroxidation, reflecting a disruption in the cellular antioxidant system [42]. These findings align with the elevated MDA levels observed in our study following exercise with high-dose CA, and support the conclusion that this oxidative stress response was reflected in the lower plasma SH levels.

Uric acid, a non-enzymatic antioxidant, was the only marker that showed a significant increase after exercise with CA supplementation, compared to PLA post-exercise. The effects of caffeine on UA levels remain uncertain. In a review by Ósz et al. [16], studies and meta-analyses were reported with mixed results, ranging from reductions to no impact on UA levels. These discrepancies may be influenced by various factors, including the source of caffeine, genetic variability, sex, and ethnicity. High-intensity exercise tends to promote a greater release of UA compared to lower-intensity exercise [43]. This increase is related to energy catabolism, the degradation of adenosine triphosphate (ATP), and the subsequent production of purines, which are metabolized into UA through a sequential enzymatic pathway [44]. Initially, ATP is hydrolyzed into adenosine diphosphate (ADP) and adenosine monophosphate (AMP) by nucleoside triphosphate diphosphohydrolases. AMP is then converted into adenosine by the action of 5'-nucleotidase, which is subsequently deaminated into inosine by adenosine deaminase. Inosine is transformed into hypoxanthine by purine nucleoside phosphorylase, which is further oxidized to xanthine and, finally, to uric acid by the enzyme xanthine oxidase [44]. Furthermore, exercise-induced muscle microdamage also contributes to the increased production of UA [43]. Caffeine consumption may potentiate this process, as it is associated with enhanced muscular strength [17,20], which can intensify the mechanical and metabolic load during exercise, thereby promoting greater UA release.

The increase in UA concentrations observed in our study may be interpreted not only as a marker of metabolic stress, but also as part of a physiological defense mechanism, acting as an antioxidant to neutralize the excess free radicals generated during high-intensity exercise [43]. Another piece of evidence supporting the elevation of UA in our study lies in the metabolism of caffeine. As an alkaloid belonging to the purine group, caffeine is primarily metabolized in the liver by the cytochrome P450 enzymatic system (CYP1A2), resulting in the formation of three major dimethylxanthines: paraxanthine, theophylline, and theobromine. These metabolites are subsequently converted into uric acid derivatives [44].

Regarding maximal isometric force, our findings demonstrated an increase in the CA group compared to PLA. Consistent with our results, high doses of caffeine have been shown to significantly

enhance muscle strength in men, likely due to increased Ca^{2+} release into the plasma [17]. However, Wilk et al. [45] reported contradictory results. When administering 9 to 11 mg/kg of caffeine, they observed no improvements in strength or muscular endurance during the bench press exercise in trained athletes, and noted adverse side effects from the supplementation. Lower caffeine doses appear effective in enhancing high-speed, low-intensity muscle actions. However, for high-intensity exercise, higher doses (e.g., 9 mg/kg) may be required [25]. In a previous study involving powerlifters, supplementation with 9 mg/kg of caffeine promoted the maintenance of velocity and power output compared to placebo [27], and did not induce adverse effects, supporting its safety in strength-based sports [26].

Caffeine may enhance muscular strength through several mechanisms. At high doses, it increases Ca^{2+} release from the sarcoplasmic reticulum, thereby stimulating the contraction of skeletal striated muscles [16]. Additionally, CA can stimulate lipolysis by increasing cyclic AMP (cAMP) levels, which raises the availability of free fatty acids that are subsequently oxidized for energy production, thus sparing muscle glycogen [16,41]. An increase in plasma Ca^{2+} concentrations was observed in studies using 8 mg/kg of caffeine, with elevated levels maintained throughout the entire testing period [17,20]. This elevation was associated with improved strength performance and muscle contractility, attributed to the enhanced availability of Ca^{2+} [17,20]. When comparing the PLA and CA groups, a reduction in force variability was observed in both PLA post-exercise and CA post-exercise conditions; however, the reduction was more pronounced with CA, suggesting greater force stability following CA ingestion. This indicates that muscle function was better maintained with CA use. Similarly, Filip-Stachnik et al. [46] reported an increase in both mean and peak bar velocity during the bench press throw following ingestion of high doses of caffeine.

Maximum force in one second was higher in the CA group compared to PLA, suggesting an acute improvement in motor unit recruitment and explosive force generation. These findings reinforce the positive influence of nutritional ergogenic strategies in mitigating fatigue and optimizing performance during strength-based exercise. In the context of supplementation, a previous study involving a similar population used ibuprofen (IBU) and reported reduced fatigue in Paralympic powerlifters following IBU administration [6]. These results also highlight that this type of resistance training induces significant muscular fatigue [5,6]. Muscle force can decline as a result of muscle fatigue, which may be either central or peripheral in origin [5]. Muscle fatigue is a key factor in the reduction of force-generating capacity and can impair motor control during movement execution [6]. Caffeine exerts both central and peripheral actions: centrally, by increasing catecholamine release

[47]; and peripherally, by enhancing Ca^{2+} release and muscle contraction duration [17,40]. Moreover, caffeine inhibits phosphodiesterase, leading to elevated cAMP levels, which in turn stimulates lipolysis and spares muscle glycogen [47]. Caffeine has been shown to significantly prolong time to exhaustion, enhance movement velocity, and amplify force output during submaximal exercise intensities [47].

LIMITATIONS

Despite the relevant findings, this study has some limitations. There was no strict control over external individual behavioral variables, such as diet and sleep quality, which may have influenced the physiological outcomes assessed. These factors are recognized as potential confounding variables that can influence systemic redox balance. However, the randomized, crossover, blinded, and counterbalanced experimental design, in which each participant acted as their own control, allowed the athletes to serve as their own control, minimizing interindividual biological variability. Furthermore, all participants were instructed to maintain their usual routines and avoid strenuous exercise in the 24 hours preceding data collection, seeking to mitigate acute interferences in the analyzed markers. Future studies with multidimensional monitoring (food diaries) are recommended to refine the understanding of these interactions.

Moreover, the intervention was acute in nature, which limits the extrapolation of results to long-term contexts, where adaptive responses may differ. Another point relates to TBARS; although it is a well-established method for assessing lipid peroxidation, we recognize its limitations regarding specificity, since other non-lipid substances can react with thiobarbituric acid. However, MDA serves here as a useful indicator of the overall redox state after caffeine supplementation. Future studies should incorporate more specific markers, such as F2-isoprostanes, or evaluate the enzymatic defense system (e.g., SOD and GPx) to confirm the antioxidant potential of caffeine in athletes. Finally, the findings are specific to trained Paralympic powerlifting athletes and may not be generalizable to non-athletic populations, who could exhibit different physiological responses to the same stimuli.

CONCLUSION

Delayed fatigue, increased muscle strength, and efficient post-exercise recovery are critical factors, especially for high-performance athletes. These elements not only contribute to enhanced

physical performance, but also support physiological adaptation to training and allow for the continuity of intense and prolonged training programs without compromising performance.

In the present study, acute caffeine supplementation at a dose of 9 mg/kg demonstrated positive effects on maximal isometric force, peak force in one second, and reduced force variability during Paralympic powerlifting exercise performed at 80% of 1RM. However, despite the neuromuscular performance benefits, significant changes were observed in oxidative stress markers, including increased TBARS levels, reduced total antioxidant capacity, decreased sulfhydryl groups, and elevated uric acid concentrations.

Thus, it can be concluded that although caffeine promoted an acute improvement in strength performance, its supplementation was not effective in attenuating exercise-induced oxidative stress in Paralympic powerlifting athletes.

PRACTICAL APPLICATIONS

From a practical point of view, the results suggest that the use of 9mg/kg of CA may be a strategy to increase strength after training in weightlifters.

AUTHORS' CONTRIBUTIONS

Conceptualization, J.L.M. and F.J.A.; methodology, F.J.A.; material preparation, data collection and analysis, J.L.M.; F.J.A.; J.U.d.O.; J.L.d.S. and A.C.M.; software, J.L.d.S.; investigation, J.L.M.; and F.J.A.; data curation, F.J.A.; writing - original draft preparation, J.L.M.; writing - review & editing, all authors; visualization, J.L.d.S.; project administration, F.J.A. All authors read and agreed to the published version of the manuscript.

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INSTITUCIONAL INSTITUTIONAL REVIEW BOARD STATEMENT

This study was carried out in accordance with Resolution 466/2012 of the National Commission for Research Ethics (CONEP) of the National Health Council and following the ethical principles of the Declaration of Helsinki (1964, revised in 2013) of the World Medical Association. This study was approved by the Research Ethics Committee of the Federal University of Sergipe (ID-CAAE: 67953622.7.0000.5546) under opinion number 6,523,247, dated November 22, 2023.

DECLARATION OF FREE AND INFORMED CONSENT

Informed consent was obtained from all subjects involved in the study.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

6. ESTUDO 3: OS EFEITOS DA SUPLEMENTAÇÃO AGUDA DE CAFEÍNA NOS PARÂMETROS DE DANOS MUSCULARES, TEMPERATURA CORPORAL E POTÊNCIA NO TREINAMENTO DE LEVANTAMENTO DE PESO PARALÍMPICO.

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The Effects of Acute Caffeine Supplementation on Muscle Damage Parameters, Body Temperature, and Power in Paralympic Powerlifting Training.

Abstract

Paralympic powerlifting is an intense modality that generates significant stress, leading to exercise-induced muscle damage (EIMD), temporary strength loss, and an increased risk of injury. To mitigate these effects and accelerate recovery, athletes often seek post-training strategies, such as the use of nutritional supplements. Caffeine is a widely used ergogenic aid. Its primary mechanism of action involves the competitive blockade of adenosine receptors, which stimulates the nervous system. This action enhances the release of catecholamines and increases neuromuscular excitability, thereby improving physical performance and recovery. Furthermore, caffeine has been shown to reduce both the perception of pain and the extent of exercise-induced muscle damage (EIMD). This study investigated the acute effects of CA supplementation (9 mg/kg) on static strength indicators and muscle damage markers in fourteen male Paralympic powerlifting athletes. The experimental protocol involved two conditions (CA and PLA), with athletes performing 5 sets of 5 repetitions (5x5) at 80% of 1-Repetition Maximum (1RM) in the adapted bench press. Variables related to strength (Rate of Force Development (RFD), Impulse, Peak Torque) and muscle damage markers (thermography, Creatine Kinase (CK), Lactate Dehydrogenase (LDH), and creatinine) were analyzed. Supplementation with CA significantly increased the Rate of Force Development (RFD), Impulse, and Peak Torque. Thermography revealed an increase in temperature with CA in all evaluated muscles when compared to the PLA condition. However, there were no significant differences observed in the markers for muscle damage, namely Creatine Kinase (CK), Lactate Dehydrogenase (LDH), and creatinine. In conclusion, CA contributed to an increase in muscular strength and did not result in a concomitant increase in muscle damage. This suggests that the use of CA at the administered dose is safe and ergogenic for Paralympic powerlifting athletes.

Keywords: Caffeine, Muscle damage, Strength training, Paralympic weightlifting

INTRODUCTION

Paralympic powerlifting is an adapted strength sport modality that demands maximum intensity, involving high loads and high levels of physiological stress on the athletes(1–3). This type of training typically results in exercise-induced muscle damage (EIMD), increased body temperature, a temporary loss in force production capacity, and a reduction in range of motion (4,5). The control of these factors is crucial, as they can elevate the risk of injury(1,4). In this context, post-training strategies are necessary to accelerate recovery, aiming to maintain the focus on the planned training schedule. Therefore, athletes commonly employ various strategies, including the use of supplements that assist in physical recovery and sport performance (1,2,4,5).

Among the nutritional strategies, we highlight the use of caffeine (CA), which is considered one of the most widely investigated ergogenic aids in the sport environment(6,7). Its primary mechanism of action is the competitive inhibition of adenosine receptors, which results in the stimulation of the sympathetic nervous system (6–8). This activation leads to an increase in catecholamine release, greater neuromuscular excitability, and the amplification of calcium release within skeletal muscle. These factors can positively influence both physical performance and post-exercise recovery processes(9,10) Furthermore, CA has also been associated with a reduction in the perception of pain and muscle discomfort, and it may even act to attenuate EIMD (11,12).

Nevertheless, results in the existing literature remain inconclusive, particularly regarding CA's efficacy in reducing muscle damage markers and enhancing functional recovery. On the one hand, studies have observed the preservation of power without a corresponding increase in muscle damage(9,13,14); on the other hand, benefits in muscle recovery have been reported (15). Furthermore, some authors reported no effect on blood markers of muscle damage following exercise (16–19). Compounding this complexity, there are reports of improved performance concomitant with an increase in muscle damage following CA use (20,21), and even the suggestion of an increased risk of injury in athletes (8).

In this context, the primary objective of this study was to analyze the acute effect of CA supplementation on post-training recovery in Paralympic powerlifting athletes, by investigating its potential influence on physiological parameters. Therefore, we hypothesized that caffeine supplementation would improve physical recovery, reduce muscle damage and body temperature, and preserve muscle power in athletes of this sport modality.

STUDY DESIGN

The experimental design of this study, illustrated in Table 1, was conducted over three consecutive weeks. In the first week, participants underwent a familiarization phase with the adapted bench press exercise, followed by the assessment of the maximum load using the one-repetition maximum (1-RM) test. The study employed a randomized crossover design. During the second and third weeks, the volunteers received an acute supplementation of either anhydrous caffeine (CA, 9 mg/kg) or placebo (PLA), administered 60 minutes prior to the start of the training session. The order of the conditions (CA or PLA) was randomly determined by drawing lots, ensuring a balanced distribution (50% for each condition). In the third week, the intervention conditions were crossed over: participants who had previously ingested CA received PLA, and vice versa. The exercise sessions consisted of five sets of five repetitions (5x5) at 80% of the load determined during the 1-RM test, with recovery intervals between sets ranging from three to five minutes (1,5,22).

Throughout the experimental protocol, variables related to strength and muscle damage were evaluated. The strength indicators included the Rate of Force Development (RFD), Impulse, and Peak Torque. Muscle damage indicators comprised Skin Temperature (assessed via thermography), Creatine Kinase (CK), Lactate Dehydrogenase (LDH), and creatinine (CRE). Blood samples and physiological measurements were consistently collected at four specific time points: 60 minutes before exercise, immediately post-exercise, and 24 and 48 hours after each session.

Prior to initiating the training protocol, participants performed a general warm-up focused on the upper limbs, consisting of three exercises: 1- cable triceps push-down (or elbow extension on the pulley), 2- shoulder rotations with dumbbells, and 3- shoulder abduction with dumbbells. Three sets of each general exercise were executed, using loads corresponding to 10–20% of the 1-RM, totaling approximately 10 minutes in duration. Subsequently, a specific warm-up was performed for the adapted bench press exercise, utilizing a load equivalent to 30% of the 1-RM.(23). This specific warm-up consisted of 10 repetitions with a slow execution (3-second eccentric phase and 1-second concentric phase) followed by 10 repetitions with a fast execution (1 second per phase). Throughout the entire protocol, participants were verbally encouraged to exert maximal effort (23).

Figure 1 Experimental design of the study

Week 1 (Familiarization and tests)		Warm- up	Test 1 RM 		rest
Week 2 and 3 50% Placebo or 50% caffeine supplementation	Before training: variables of strength and muscle damage	Warm- up	Training 80% 1RM  5x5	Immediately after training, 24h and 48h: variables of strength and muscle damage	rest

SAMPLE

The sample was composed of fourteen male Paralympic Powerlifting (PP) athletes. All participants were ranked among the top 10 nationally in their respective body weight categories. The participants presented with distinct physical impairments, detailed as follows: two athletes with left leg atrophy; three with left lower limb amputation; two with right lower limb amputation; one with an injury to the right lower limb, resulting in functional loss and muscle hypotonia; and one athlete with a spinal cord injury resulting from schistosomiasis. Furthermore, three participants exhibited sequelae of poliomyelitis in the lower limbs, and two were diagnosed with arthrogryposis.

The inclusion criteria were defined as follows: being clinically stable, possessing a minimum of 18 months of systematized experience in the sport, engaging in regular competitive activity, and being eligible according to the established criteria for participation in Paralympic sport modalities.

Exclusion criteria included the presence of cardiorespiratory diseases, pre-existing cardiac conditions, the use of any type of substance with potential ergogenic effects, and the participation of female athletes. The exclusion of female participants was aimed at avoiding potential interferences resulting from natural or induced hormonal fluctuations (e.g., hormonal supplementation), which could compromise the validity of the effects attributed to the intervention (24).

All volunteers participated in the study with informed consent. The research was approved by the Ethics Committee on Research of the Federal University of Sergipe (UFS), under CAAE No. 67953622.7.0000.5546, Opinion No. 6.523.247, issued on November 22, 2023. The study was conducted in accordance with the ethical principles established in the Declaration of Helsinki (1964, revised in 2013) by the World Medical Association, and adhered to the guidelines of Resolution No. 466/2012 of the National Health Council (CNS), through the National Research Ethics Committee (CONEP).

A detailed characterization of the sample is presented in Table 2.

Table 1: Characteristics of the study participants (n = 14)

Features	(Mean $\pm$ SD)
Age (years)	32,4 $\pm$ 8,5
Body mass (kg)	81,7 $\pm$ 21,2
Experience (years)	3,1 $\pm$ 1,0
Systolic blood pressure (mmHg)	126,4 $\pm$ 15,2
Diastolic blood pressure (mmHg)	75,5 $\pm$ 11,9
1RM test (bench press) (kg)	126,9 $\pm$ 41,2
1RM test/body mass (kg)	1,6 $\pm$ 0,4

1RM: one repetition maximum.

INSTRUMENTS

The measurement of participants' body mass was performed using a digital electronic scale specifically designed for wheelchair users (MicWelchair, Michetti®, São Paulo, SP, Brazil), ensuring precision and accessibility during the weighing process.

The exercise execution was conducted on an official competition bench (2.10 m in length; Eleiko®, Halmstad, Sweden) and with a standardized Olympic barbell (220 cm in length, 20 kg mass). Both the bench and the barbell were certified by the International Paralympic Committee (25), ensuring conformity with the technical parameters required for the practice of Paralympic Powerlifting.

CAFFEINE ANHYDRO (CA) AND PLACEBO (PLA)

The CA and PLA capsules were prepared by a specialized compounding pharmacy, ensuring standardization in terms of color, size, and volume to maintain the double-blind nature of the study. The CA dose was individually adjusted to 9.0 mg/kg of body weight, as per recommendations based on the scientific literature. The PLA capsules were filled with inert

pharmaceutical talc, in an equivalent amount to that used in the caffeine formulation, ensuring the indistinguishability between the experimental conditions.

COLLECTION OF UPPER LIMB MUSCLE STRENGTH INDICATORS

The measurement of the Rate of Force Development (RFD), Impulse, and Peak Torque was performed using a Chronojump load cell (Chronojump®, BoscoSystem, Madrid, Spain), featuring a 500 kg capacity, an output impedance of $A\ 350 \pm 3\ \text{ohm}$, insulation resistance of $>2000\ \text{cc}$, input impedance of $365 \pm 5\ \text{ohm}$, and a 24-bit, 80 Hz Analog-to-Digital converter. The load cell was coupled to the bench press by means of Spider HMS Simond carabiners (Chamonix, France), which have a breaking load of 21 kN and are certified for climbing by the Union Internationale des Associations d'Alpinisme (UIAA). A steel chain with a breaking load of 2300 kg was employed to secure the load cell to the bench. The RFD was determined using the force-time ratio until maximum force was achieved ($\text{RFD} = \Delta\text{force}/\Delta\text{time}$) at the 300 ms time point. During the assessment, volunteers were instructed to perform a single elbow extension movement with maximal force and speed, followed by relaxation.

THERMOGRAPHIC DATA COLLECTION

The evaluation of body temperature was performed using an infrared thermographic camera (Seek Thermal Compact Pro®, Moscow, Russia), with a resolution of 320×240 pixels and a thermal detection range of $-4\ ^\circ\text{C}$ a $330\ ^\circ\text{C}$. Image acquisition occurred at a distance ranging between 0.91 m and 5.48 m from the participants, in accordance with the equipment's technical specifications. The body areas evaluated included the Pectoralis Major muscle (both the sternal and clavicular heads), the anterior deltoid, and the long head of the Triceps Brachii (Fig. 2).

The images were recorded in an environment with artificial lighting, controlled ambient temperature (approximately $24\ ^\circ\text{C}$), and relative humidity maintained at 50%. These conditions were monitored using air conditioning and a digital hygrometer (Model HM-01; HIGHMED®, USA) (26). During image acquisition, participants remained in a seated position, avoiding sudden movements to ensure thermal stability and image quality. As part of the methodological control, all participants were previously instructed to abstain from intense physical activity for 24 hours preceding the examination, and to refrain from applying lotions, creams, or any topical substances to the skin in the 6 hours prior to the assessment, in order to ensure standardized conditions and the reliability of the obtained data.

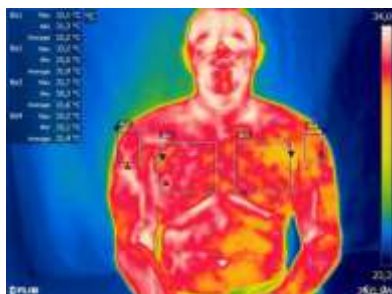


Figure 2. Photographic model of infrared thermography.

COLLECTION OF BLOOD SAMPLES

Blood collection was performed by a duly qualified nursing professional via venipuncture of the participants' antecubital vein, obtaining 10 mL of blood per individual. Immediately following collection, the samples were transferred to tubes containing the anticoagulant ethylenediaminetetraacetic acid (EDTA) to preserve the blood components for analysis. The samples were initially subjected to centrifugation at $3000\times g$ for 10 minutes to separate the plasma and serum. The separated components were aliquoted and stored under refrigeration at 4°C for short-term biochemical analyses. Additionally, a fraction of the collected blood was processed by centrifugation at $800\times g$ for 15 minutes at 4°C to obtain serum with greater stability. This fraction was then stored in an ultra-low temperature freezer at -80°C , ensuring its integrity for subsequent analyses of sensitive biochemical markers.

MUSCULAR ANALYSIS OF MUSCLE DAMAGE MARKERS

The extent of muscle damage was determined by analyzing enzymatic markers of tissue injury. Serum levels of creatine kinase (CK), lactate dehydrogenase (LDH), and creatinine (CRE) were quantified. For the measurement, $20\ \mu\text{L}$ of plasma from each athlete was used, which was processed using standardized commercial kits (Labtest®, Lagoa Santa, Minas Gerais, Brazil). The enzymatic reactions occurred under a controlled temperature of $37 \pm 0.2^{\circ}\text{C}$. Subsequently, the absorbances were read on a spectrophotometer (Biospectro Model SP-22 UV/Visible, Curitiba, Brazil) at a wavelength of 340 nm.

STATISTICAL ANALYSIS

Measures of central tendency were applied, expressed as mean ($\bar{X}$) $\pm$ standard deviation (SD), 95% confidence interval (95% CI). The normality of the data was verified using the Shapiro-Wilk test, taking into account the sample size. For performance comparisons between

different time points (Before $\times$ After $\times$ After 24h $\times$ After 48h), the necessary assumptions for applying a two-way ANOVA were met. Specific differences between time points were analyzed using the Bonferroni post-hoc test. The effect size in the ANOVA was evaluated using the partial eta-squared (η^2_p), considering the following parameters: small effect (≤ 0.05), medium (0.05 to 0.25), large (0.25 to 0.50) and very large (> 0.50) (27). All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS, version 22.0) software, adopting a significance level of $p < 0.05$.

RESULTS

Table 2 presents the results for the static force indicators, and Table 3 shows the results for skin temperature.

Table 2. Static strength indicators (RFD, Impulse, Peak torque) (Mean $\pm$ SD; 95% CI) at various times with Placebo and Caffeine supplementation in Paralympic Powerlifting

	RFD (N/s) X $\pm$ DP (IC 95%)	Impulse (N*s) X $\pm$ DP (IC 95%)	Torque peak (Nm) X $\pm$ DP (IC 95%)
Placebo Before	2266,61 $\pm$ 1525,11 (1386,04-3147,18)	3621,09 $\pm$ 1159,83a,b (2951,43-4290,76)	405,04 $\pm$ 123,71 a (333,61-476,47)
Caffeine Before	1978,36 $\pm$ 1180,83c,d (1296,57-2660,15)	3741,84 $\pm$ 1245,43 e (3022,75-4460,92)	407,09 $\pm$ 131,57 (331,12-483,05)
Placebo After	2021,75 $\pm$ 2043,45a,b (841,90-3201,60)	2883,96 $\pm$ 1196,70a,c,f (2193,01-3574,92)	324,08 $\pm$ 129,00 a,b,d (249,60 -398,56)
Caffeine After	1426,41 $\pm$ 840,90e,f (940,88-1911,93)	3330,45 $\pm$ 1049,97 f (2724,22-3936,68)	371,71 $\pm$ 111,50 d (307,33 -436,09)
Placebo 24 hours	4231,24 $\pm$ 3508,75 a (2205,35-6257,13)	2749,36 $\pm$ 1264,54b,d (2019,23-3479,48)	339,99 $\pm$ 121,24 c (269,99 -409,990)
Caffeine 24 hours	4342,24 $\pm$ 2346,98c,e (2987,1-5697,35)	3147,20 $\pm$ 841,29 e (2661,45-3632,95)	367,47 $\pm$ 99,37 (310,10 -424,850)
Placebo 48 hours	5708,11 $\pm$ 4534,83 b (3089,78-8326,45)	3498,53 $\pm$ 1000,08c,d (2921,10-4075,96)	393,12 $\pm$ 114,96 b,c (326,74-459,490)
Caffeine 48 hours	5415,94 $\pm$ 3261,54d,f (3532,79-7299,10)	3401,26 $\pm$ 1020,25 (2812,18-3990,33)	374,04 $\pm$ 130,22 (298,86 -449,230)
P	“a” p=0.017* “b” p=0.003* “c” p=0.001* “d” p=0.002* “e” p=0.001* “f” p=0.004*	“a” p=0.016* “b” p=0.027* “c” p=0.017* “d” p=0.020* “e” p=0.043* “f” p=0.012#	“a” p=0.015* “b” p=0.008* “c” p=0.002* “d” p=0.005#

η^2p

0.692

0.446*

0.424*

0.177#

0.212#

$p < 0.05$ (two-way ANOVA, and Bonferroni post hoc, ES η^2p). In the statistical analysis, the letters “a” to “f” indicate which conditions and at which times the differences were observed. * Intraclass, # Interclass. A vs B; A vs C and B vs C (t-test and Cohen's d). η^2p = partial eta square (small effect ≤ 0.05 , medium effect 0.05 to 0.25, high effect 0.25 to 0.50, and very high effect (>0.50)). The results in bold in the RFD, impulse, and peak torque columns, and the letters after the mean and standard deviation values, refer to the time and corresponding methods of the first column.

For RFD, differences were observed between the time points, PLA after and 24 hours ($p=0.017$), PLA after and 48 hours ($p=0.003$), CA before and 24 hours ($p=0.001$), CA before and 48 hours ($p=0.002$), CA after and 24 hours ($p=0.001$), CA after and 48 hours ($p=0.004$).

For impulse, differences were observed between the time points, PLA before and after ($p=0.016$), PLA before and 24 hours ($p=0.027$), PLA after and 48 hours ($p=0.017$), PLA 24 hours and 48 hours ($p=0.020$), CA before and 24 hours ($p=0.043$), and between the PLA after and CA after groups ($p=0.012$).

For peak torque, a difference was observed between the moments, PLA before and after ($p=0.015$), PLA after and 48 hours ($p=0.008$), PLA 24 hours and 48 hours ($p=0.002$) and between the PLA after and CA after groups ($p=0.005$).

Table 3: Skin temperature (Mean $\pm$ SD; 95% CI) at various times with caffeine and placebo use in Paralympic Powerlifting.

	Pectoralis Sternal (°C)	Pectoralis Clavicular (°C)	Deltoid (°C)	Triceps (°C)
Before Placebo	33,67 $\pm$ 2,35 (32,37-34,97)	33,53 $\pm$ 2,17 (32,33-34,73)	33,73 $\pm$ 1,71 (32,79-34,68)	32,73 $\pm$ 0,70 (32,34-33,12)
Before Caffeine	34,07 $\pm$ 0,70 (33,68-34,46)	34,20 $\pm$ 1,37 (33,44-34,96)	34,73 $\pm$ 1,10 (34,12-35,34)	33,33 $\pm$ 1,45 (32,53-34,13)
After placebo	33,00 $\pm$ 2,56 a (31,58-34,42)	33,80 $\pm$ 2,18 (32,59-35,01)	33,87 $\pm$ 2,20 (32,65-35,08)	32,80 $\pm$ 1,70 a (31,86-33,74)
After Caffeine	34,80 $\pm$ 2,48 a (33,42-36,18)	35,00 $\pm$ 2,39 (33,68-36,32)	35,33 $\pm$ 1,99 (34,23-36,43)	34,47 $\pm$ 1,81 a (33,47-35,47)
24 hours placebo	33,67 $\pm$ 2,38 b (32,35-34,98)	33,47 $\pm$ 2,10 a (32,30-34,63)	33,87 $\pm$ 1,85 a (32,84-34,89)	32,47 $\pm$ 1,73 b (31,51-33,42)
24 hours Caffeine	35,20 $\pm$ 2,04 b (34,07-36,33)	35,20 $\pm$ 1,90 a (34,15-36,25)	35,47 $\pm$ 1,68 a (34,53-36,40)	34,13 $\pm$ 1,51 b (33,30-34,97)
48 hours placebo	33,13 $\pm$ 2,36 c (31,83-34,44)	33,67 $\pm$ 2,02 b (32,55-34,79)	33,93 $\pm$ 1,83 b (32,92-34,95)	32,87 $\pm$ 1,19 c (32,21-33,52)
48 hours Caffeine	34,93 $\pm$ 1,28 c (34,22-35,64)	35,20 $\pm$ 1,32 b (34,47-35,93)	35,47 $\pm$ 0,99 b (34,92-36,02)	33,87 $\pm$ 1,13 c (33,24-34,49)

P	“a” $p=0.017\#$ “b” $p=0.029\#$ “c” $p=0.003\#$	“a” $p=0.006\#$ “b” $p=0.005\#$	“a” $p=0.008\#$ “b” $p=0.001\#$	“a” $p=0.020\#$ “b” $p=0.004\#$ “c” $p=0.004\#$
F	14.361	12.877	16.817	23.133
η^2p	0.506	0.479	0.546	0.623

$p \leq 0.05$ (two-way ANOVA and Bonferroni post-hoc test). η^2p = partial eta squared (small effect ≤ 0.05 , medium effect 0.05 to 0.25, high effect 0.25 to 0.50, and very high effect (>0.50)). In the statistical analysis, the letters “a” to “c” indicate which conditions and at which times the differences were observed. * Intraclass, # Interclass. The results in bold in the columns Pectoralis Sternal, Pectoralis Clavicular, Deltoid, Triceps, and the letters after the mean and standard deviation values, refer to the time point and the corresponding methods of the first column.

In the sternal pectoral muscle, there was a difference in the following time points: PLA after and CA after ($p=0.017$); PLA 24 hours and CA 24 hours ($p=0.029$); PLA 48 hours and CA 48 hours ($p=0.003$).

In the clavicular pectoral muscle, there was a difference in the following time points: PLA 24 hours and CA 24 hours ($p=0.006$); PLA 48 hours and CA 48 hours ($p=0.005$).

In the deltoid muscle, there was a difference in the following time points: PLA 24 hours and CA 24 hours ($p=0.008$); PLA 48 hours and CA 48 hours ($p=0.001$).

In the triceps muscle, there was a difference in the following time points: PLA after and CA after ($p=0.020$); PLA 24 hours and CA 24 hours ($p=0.004$); PLA 48 hours and CA ($p=0.004$).

Figure 3 shows the results for muscle damage, creatine kinase (CK), lactate dehydrogenase (LDH), and creatinine (CRE) at the times before, after, 24 hours, and 48 hours.

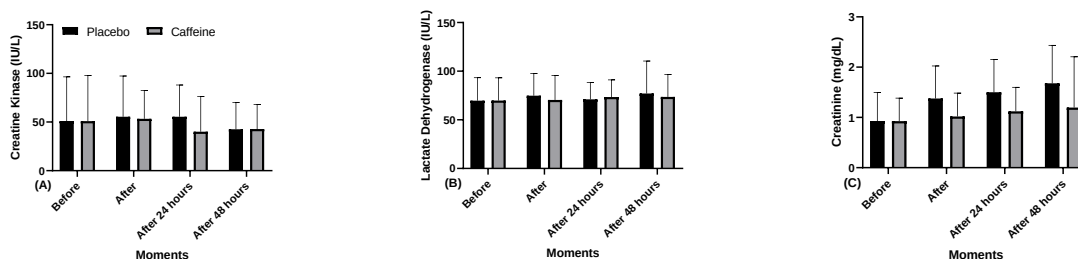


Figure 3: Muscle damage at various times with placebo and caffeine supplementation in Paralympic powerlifting.

No differences were observed in blood indicators of muscle damage.

DISCUSSION

This study aimed to analyze the effect of caffeine (CA) on post-training recovery in Paralympic powerlifting athletes. When evaluating static strength using the Rate of Force Development (RFD) indicator, our findings demonstrated a significant increase for both the Placebo (PLA) and CA conditions at more than one time point post-training. The RFD is widely used to assess neuromuscular performance, allowing researchers to monitor training adaptations, identify fatigue, and differentiate levels of athletic performance(4,28–30). This refers to the ratio between the change in force and time, with maximum values generally observed between 100 and 300 ms. (4). Because it reflects the ability to generate force rapidly, RFD is considered an essential indicator in modalities that require explosive muscle contractions (4,28). Therefore, our results corroborate the efficacy of CA in preserving muscle power.

Caffeine can promote the recovery of muscle function after injuries by reducing impairment of excitation-contraction coupling. Its direct action on skeletal muscle increases calcium sensitivity and stimulates the sodium-potassium ATPase pump, minimizing potassium accumulation in the intercellular space (9,11,31). Potassium accumulation in the T-tubules, which causes fatigue by impairing the propagation of the electrical signal, is reversed by caffeine. The substance facilitates the action potential and optimizes calcium release in the sarcoplasmic reticulum, improving contraction and strength(31). Additionally, the attenuation of fatigue may be linked to better oxygen availability for muscle tissue(32). Or, it may act directly on the central nervous system (33).

Our results corroborate the study by Hurley et al., (9) which found that resistance exercises for the upper body showed improved performance with the use of CA. And also with Enes et al. (34) observed an increase in neuromuscular performance in powerlifting athletes during training with 80 and 90% of 1RM, in addition to a reduction in the subjective perception of effort. Furthermore, a meta-analysis also showed an increase in RFD with the use of CA (28).

Supplementation with CA demonstrated an increase (average 446.49) in the static impulse strength indicator in the post-training period compared to placebo. In weightlifters, doses of 9 mg/kg of CA maintained speed and power compared to the placebo group(22). In the study by Pallarés et al. (35), only high doses of CA resulted in increased bar speed and power during high-intensity exercises (90% of 1RM), although with side effects. However, it has already been shown that high doses of CA are safe for Paralympic weightlifters(24). Similarly, supplementation of 9 mg/kg CA in professional athletes had a significant effect on the mechanical activity of skeletal muscles, significantly improving contraction time, due to changes in the central nervous system and also reduced calcium pump activity (36).

Regarding the static force indicator, peak torque, our study also showed an increase (average of 47.63) in the CA condition after training when compared to the PLA condition after training. Thus, there was less loss of force with the use of CA. Fatigue results in a reduction in the ability to generate force and compromises the motor control of movements. Its causes can be central or peripheral, and both can generate disorganization in the execution of the movement, hindering recovery (37).

Corroborating our results, an investigation with physically active individuals reported an ergogenic effect on peak isokinetic torque with the use of CA, both in previously injured skeletal muscles and in intact muscle tissues (38). CA improved upper body muscle strength and endurance in moderate and high intensity muscle endurance tests (39,40). Duncan et al., (41) also observed improved performance in endurance exercises to muscle failure with the use of CA, in addition to a reduction in perceived exertion and muscle soreness.

The ergogenic effect of CA on endurance exercise performance can be explained by its action on multiple systems, including skeletal muscle and the central nervous system (38,39). Evidence indicates that CA can act directly on the sarcoplasmic reticulum, increasing calcium release and, dose-dependently, thus favoring muscle contractile capacity (42). In addition, its potential to stimulate the central nervous system may result in greater recruitment of motor units and increased neuromuscular activation, contributing to improved physical performance (32,38,39).

High-intensity exercise tends to cause a transient reduction in body temperature, followed by a subsequent increase, reflecting the activation of inflammatory and metabolic processes resulting from physical exertion (26). Our study used infrared thermography to investigate potential changes in upper body temperature resulting from CA administration. There was an increase with CA in all muscles evaluated when compared to the PLA condition, at the following times: post, 24h and 48h, which indicates changes in blood flow in the evaluated areas, which may indicate the activation of a systemic inflammatory process.

Caffeine is a CNS stimulant that leads to the release of catecholamines, thus increasing metabolism and generating heat. In addition, the increased metabolic demand and adrenaline release can lead to increased blood flow to the active muscle (peripheral vasodilation) (32). Increased blood flow carries more heat to the skin surface, which is captured by thermography.

The study by Ely et al (43) supplementing 9mg/kg of CA in continuous moderate-intensity exercise in a hot environment observed an elevation in basal body temperature, increased oxygen consumption, and increased heat production when compared to the placebo condition. Fraga et al. (4) supplementing ibuprofen in Paralympic weightlifting athletes also found an increase in skin

temperature with the use of the anti-inflammatory drug. Measuring basal temperature in the upper body in people who use wheelchairs has methodological limitations, as the recurrent movements required for small locomotion during the experimental protocol make it difficult to standardize a stable resting condition (4).

Muscle injury can be assessed by detecting intracellular proteins and enzymes in the circulation, which function as sensitive biomarkers of tissue impairment (11,44). Damage to the extracellular matrix and sarcolemma allows these molecules to leak into the extracellular space and blood. Among the main indicators are CK, LDH, and creatinine, which reflect both the extent of the damage and the physiological response to mechanical or metabolic stress (11,44). They are particularly sensitive after resistance exercises or predominantly eccentric contractions (11,44). However, our results did not indicate significant differences in the biochemical markers of muscle damage evaluated.

High-intensity exercise would result in damage (45). Thus, leading to CK extravasation, however, the interpretation of serum CK levels should be done with caution, since variables such as hydration status can significantly influence its plasma concentration (9). In general, peak CK activity in the blood, as well as the maximum intensity of delayed onset muscle soreness (DOMS), occur between 24 and 48 hours after exercise (9). In the present study, CK levels did not show significant differences between conditions, even after this time interval, however, in absolute values there was a reduction when compared to pre-exercise. Even though these are not statistically significant differences in absolute terms, this can represent an important gain in terms of competitions. Any small difference would represent better performance.

Physical training is directly related to the increased mechanical and metabolic demands placed on muscle tissue, favoring the occurrence of microlesions (37,44,45). This process can lead to the escape of intracellular enzymes, such as creatine kinase CK, into the extracellular environment (13). This is a fact that may have interfered with our CK results, as powerlifting is a short-duration sport. Another point is related to the heterogeneity of our sample, as some variables such as: individual factors including: age, body composition, muscle mass, lipid profile, ethnic origin, level of physical conditioning, as well as the use of medications or ergogenic substances can alter CK levels (46).

Like our findings, several studies also found no significant difference in markers of muscle damage. Filip et al., (16) for example, did not observe significant changes in CK and LDH levels when supplementing with CA in trained men. Similarly, Hurley et al., (9) and Ribeiro et al., (13) in studies with upper body exercises and handball athletes, respectively, also found no effects on CK

levels. However, the literature presents controversies, as increases in blood concentrations of CK and LDH have been reported in previous investigations (8).

CK, LDH, and creatinine are related to muscle damage(13). At rest, these enzymes are present in low concentrations, but after intense exercise, their release into the bloodstream occurs due to cell injury, indicating muscle damage (46). In our study, no significant increases in serum levels of CK, LDH, and creatinine were found in the athletes; however, we observed an increase in skin temperature. However, this is one of the signs of tissue repair; after exercise, an inflammatory response occurs, characterized by an increase in leukocytes and an elevation in local temperature (46). Thus, our study showed that caffeine increased static strength indicators without increasing damage indicators.

CONCLUSIONS

Strength training involves successive concentric and eccentric muscle contractions, which commonly lead to the development of muscle damage (44). This condition is identified by changes in the structure of muscle fibers and by characteristic clinical signs, such as decreased strength and range of motion, increased sensitivity and muscle edema, as well as the marked release of proteins from inside muscle cells (44).

In the present study, acute supplementation with CA, at a dose of 9 mg/kg during Paralympic powerlifting training performed at 80% of 1RM, promoted an increase in strength, as well as an increase in skin temperature measured by thermography, which may be related to the tissue repair process, a common occurrence after strength training. Furthermore, there was no difference in blood indicators of muscle damage such as CK, LDH, and creatinine.

Therefore, it can be concluded that caffeine administration (9mg/kg) contributed to increased muscle strength and did not result in increased muscle damage, suggesting that its use is safe. Thus, nutritional interventions with caffeine may help high-performance athletes cope with greater physiological overload during intense training.

This study has some limitations that should be considered when interpreting the results. Firstly, the participating athletes belong to a sport with a single classification, not stratified by type of disability, which may have influenced the observed responses. Furthermore, the sample size was relatively small, even though it included athletes of national and international competitive level, which limits the generalizability of the findings to other sporting populations.

PRACTICAL APPLICATIONS

From a practical standpoint, the results suggest that using 9 mg/kg of CA could be a strategy to increase strength in weightlifters' training without altering muscle damage compared to PLA.

AUTHORS' CONTRIBUTIONS

Conceptualization, J.L.M. and F.J.A.; methodology, F.J.A.; material preparation, data collection and analysis, J.L.M.; F.J.A.; J.U.d.O.; J.L.d.S. and A.C.M.; software, J.L.d.S.; investigation, J.L.M.; and F.J.A.; data curation, F.J.A.; writing - original draft preparation, J.L.M.; writing - review & editing, all authors; visualization, J.L.d.S.; project administration, F.J.A. All authors read and agreed to the published version of the manuscript.

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INSTITUCIONAL INSTITUTIONAL REVIEW BOARD STATEMENT

This study was carried out in accordance with Resolution 466/2012 of the National Commission for Research Ethics (CONEP) of the National Health Council and following the ethical principles of the Declaration of Helsinki (1964, revised in 2013) of the World Medical Association. This study was approved by the Research Ethics Committee of the Federal University of Sergipe (ID-CAAE: 67953622.7.0000.5546) under opinion number 6,523,247, dated November 22, 2023. **DECLARATION OF FREE AND INFORMED CONSENT** Informed consent was obtained from all subjects involved in the study. **CONFLICTS OF INTEREST**

The authors declare no conflict of interest.

7. CONCLUSÕES GERAIS

No início do estudo foram elencados problemas às quais pretendíamos dar resposta com os estudos realizados na presente investigação. Assim, com base nos estudos realizados respondemos às questões inicialmente estabelecidas.

Quais os efeitos da suplementação de cafeína durante o treinamento sobre o desempenho de força dinâmica e estática, e suas implicações sobre o estresse oxidativo e o dano muscular em atletas paralímpicos?

-A suplementação de 9mg/kg de cafeína melhorou o desempenho apenas de forma aguda (durante o treino). Além disso, ela não acelerou e nem prejudicou a recuperação muscular em relação a fadiga nas 24 ou 48 horas pós o treino.

-A cafeína aumentou a força, explosão e estabilidade no halterofilismo paralímpico, no entanto, ela não atenuou, e sim intensificou o estresse oxidativo induzido pelo exercício.

-A cafeína além de contribuir para aumento da força muscular, ela não resultou em aumento do dano muscular, sugerindo que sua utilização é segura nesse quesito para esta população.

Sendo assim, a suplementação aguda de cafeína 9 mg/kg em atletas powerlifting paralímpico demonstra efeitos ergogênicos relevantes, especialmente na manutenção da força dinâmica em altas intensidades e na melhora da força estática, evidenciada pelo aumento da força isométrica máxima e da estabilidade de produção de força. Em contraste, a cafeína não promove efeito protetor sobre o estresse oxidativo, podendo inclusive intensificá-lo, apesar de não aumentar os marcadores de dano muscular. Contudo, esses efeitos parecem depender da intensidade do exercício, não sendo observados em cargas moderadas. Dessa forma, conclui-se que a cafeína é um recurso eficaz para otimizar o desempenho neuromuscular em atletas paralímpicos, especialmente em condições de alta exigência, sendo considerada segura quanto ao dano muscular, porém com possíveis implicações negativas no equilíbrio redox.

Do ponto de vista prático, os resultados sugerem que o uso de 9mg/kg de CA pode ser uma estratégia ergogênica benéfica, permitindo aos atletas manter a intensidade do treinamento ao longo da sessão, mesmo ao utilizar cargas mais elevadas, além de aumentar a força no treino sem alterar o dano muscular. Portanto, se o foco do atleta for puramente performance imediata, o uso é recomendado; se o foco for a recuperação celular rápida entre sessões diárias, o estresse oxidativo gerado deve ser estrategicamente monitorado.

8. REFERÊNCIAS

8.1 REFERÊNCIAS DA INTRODUÇÃO

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APÊNDICE A – TERMO DE CONSENTIMENTO LIVRE E ESCLARECIDO

Eu _____, abaixo assinado, autorizo a Universidade Federal de Sergipe, por intermédio da aluna Jainara Lima Menezes, assistida pelo seu orientador Dr. Felipe José Aidar Martins, a desenvolver a pesquisa abaixo descrita:

1- Título do Experimento: “Análise da influência da suplementação de cafeína sobre indicadores hemodinâmicos no powerlifting paralímpico”.

2- Objetivo: Analisar os efeitos da influência da suplementação de cafeína sobre indicadores hemodinâmicos no powerlifting paralímpico.

3- Descrição de procedimentos: Os atletas serão orientados pelo pesquisador e irão assinar o termo, após esta etapa os atletas vão participar da avaliação corporal, familiarização aos treinos e assim serão incluídos na pesquisa em dois grupos durante duas semanas, e consumiram um suplemento, após isso, irão realizar o treinamento de 5x5.

4- Desconfortos e riscos esperados: Alterações na hemodinâmica e instabilidades do atleta. Fui devidamente informado dos riscos acima descritos e de qualquer risco não descrito, não previsível, porém que possa ocorrer em decorrência da pesquisa será de inteira responsabilidade do pesquisador.

5- Benefícios esperados são: o suplemento utilizado não causa risco cardiovascular.

6- Informações: Os participantes têm a garantia que receberão respostas a qualquer pergunta e esclarecimento de qualquer dúvida quanto aos assuntos relacionados à pesquisa. Também o pesquisador supracitado assume o compromisso de proporcionar informações atualizadas obtidas durante a realização do estudo.

7- Retirada do consentimento: O voluntário tem a liberdade de retirar seu consentimento a qualquer momento e deixar de participar do estudo, não acarretando nenhum dano ao voluntário.

8- Aspecto Legal: Elaborado de acordo com as diretrizes e normas regulamentadas de pesquisa envolvendo seres humanos atende à Resolução nº 466/2012 da Comissão Nacional de Ética em Pesquisa (CONEP) do Conselho Nacional de Saúde e seguindo os princípios éticos da Declaração de Helsinque (1964, revisada em 2013) da Associação Médica Mundial. Este estudo foi aprovado pelo Comitê de Ética em Pesquisa da Universidade Federal de Sergipe (ID-CAAE: 67953622.7.0000.5546) sob o parecer número 6.523.247, datado de 22 de novembro de 2023. Os voluntários terão direito à privacidade, portanto a identidade (nomes e sobrenomes) do participante não será divulgada. Porém os voluntários assinarão o termo de consentimento para que os resultados obtidos possam ser apresentados em congressos e publicações.

10- Quanto à indenização: Não há danos previsíveis decorrentes da pesquisa, mesmo assim fica prevista indenização, caso se faça necessário.

11- Os participantes receberão uma cópia deste Termo assinada por todos os envolvidos (participante e pesquisador).

12- Dados da pesquisadora responsável:

Nome: Jainara Lima Menezes

Endereço/e-mail: Av Barão do Rio Branco, centro, nº: 396/jhaynnara@hotmail.com

ATENÇÃO: A participação em qualquer tipo de pesquisa é voluntária. Em casos de dúvida quanto aos seus direitos, escreva para o Comitê de Ética em Pesquisa da Universidade Federal de Sergipe. CEP/UFS

Avenida Marechal Rondon Jardim s/n - Rosa Elze, São Cristóvão - SE, 49100-000

São Cristóvão, ____de ____de 201__.

ASSINATURA DO VOLUNTÁRIO

ASSINATURA DO PESQUISADOR RESPONSÁVEL