Jurnal Gizi Indonesia
(The Indonesian Journal of Nutrition)

Jurnal Gizi Indonesia (The Indonesian Journal of Nutrition)
Vol. 14, No. 1, December 2025 (59-67)
Received: 24 January 2025, Revised: 21 August 2025,
Accepted: 21 October 2025, Published:30 December 2025
https://ejournal.undip.ac.id/index.php/jgi, DOI:10.14710/jgi.14.1.59-67

The impact of drinking brewed coffee on VO_2max and blood lactate levels in sedentary males with physical activity treatment

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ABSTRACT

Background: Coffee is a popular ergogenic beverage that improves stamina and reduces fatigue. Coffee's caffeine content is thought to be the main ingredient in ergogenic supplements, which increase endurance during physical activity.
Objective: This study investigates the acute effect of brewed coffee on muscle fatigue markers, namely blood lactate and volume of oxygen maximum (VO_2max), in sedentary males after a standardized physical activity protocol.
Methods: This research was quasi-experimental (posttest-only with a control group design). The subjects were men aged 18-22 years, with 16 people. They were divided into a control group (n=9) and a intervention group (n=7). The intervention was heavy physical activity (1600-meter run) and Gayo Arabica coffee. Data analysis used an independent sample t-test ($p < 0.05$).
Results: Blood lactate levels were significantly higher in the intervention group than in the control group (15.04 ± 2.38 vs. 10.60 ± 4.10; $p = 0.018$), even though VO_2max values were higher but not statistically significant (39.57 ± 2.37 vs. 37.62 ± 4.17; $p = 0.297$).
Conclusions: Coffee consumption considerably raises blood lactate levels, but it has no discernible effect on VO_2max values in sedentary men who exercise vigorously. Therefore, more research is necessary to fully understand how coffee affects ergogenic and fatigue.

Keywords: Coffee; VO_2max; blood lactate; sedentary; heavy physical activity

BACKGROUND

Sedentary lifestyle refers to any waking behavior characterized by an energy expenditure of ≤1.5 metabolic equivalents (METs) while sitting, reclining, or lying, as defined by the World Health Organization (WHO).[1,2] In contrast, physical activity encompasses any bodily movement produced by skeletal muscles that requires energy expenditure.[3] Globally, physical inactivity is a significant public health problem, with the prevalence reaching 31.3% in 2022, a rise from 23.4% in 2000 and 26.4% in 2010. This upward trend has been observed in many countries and across most regions of the world, with consistently higher prevalence among women (33.8%) than men (28.7%).[4] In Indonesia, the 2018 Basic Health Research (Riskesdas) recorded that 35% of the population was physically inactive,[5] whereas data from the 2023 Indonesian Health Survey (SKI) show markedly higher proportions among younger age groups, with 58.0% of children and adolescents aged 10–14 years and 50.4% of those aged 15–19 years classified as physically inactive.[6]

Physically inactive individuals have a 20–30% higher risk of mortality compared to those who maintain adequate physical activity levels.[5] Furthermore, the WHO reports that at least 2 million deaths each year are attributable to physical inactivity or a sedentary lifestyle.[7] Despite these risks, many individuals remain inactive, often due to low health awareness and apprehension about experiencing muscle soreness during or after exercise.[8] Muscle soreness after exercising is usually linked to a build-up of lactate, which happens during hard or intense activity.[9] The excess lactate leads to metabolic acidosis, which subsequently reduces: the release and reuptake of Ca^{2+} within the sarcoplasmic reticulum, Ca^{2+} sensitivity, and the activity of adenosine triphosphatase (ATPase) along with key enzymes involved in glycolysis. These physiological effects of metabolic acidosis substantially contribute to the development of muscle fatigue.[10]

Muscle fatigue is defined as a reversible decline in the capacity of skeletal muscle to generate force or power as a consequence of contractile activity, resulting in a measurable reduction in performance relative to the expected level for a given task.[10] This phenomenon may occur independently of maximal oxygen uptake (VO_2max), which primarily reflects aerobic capacity rather than the onset of neuromuscular fatigue. Muscle fatigue arises from central mechanisms in the brain and spinal cord, or peripheral mechanisms involving the motor nerve, neuromuscular junction, or muscle fibers. It may present as acute fatigue, which develops rapidly during or shortly after exercise and is typically transient. It can also manifest as chronic fatigue, which persists

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for longer periods and is often associated with underlying pathological conditions. This study focuses on acute exercise-induced muscle fatigue. Its manifestation is influenced by exercise intensity, duration, and associated changes in muscle activation, vascular function, bioenergetics, intracellular signaling, and molecular mechanics.[10] Acute muscle fatigue arises primarily from the accumulation of lactic acid and depletion of glycogen stores within the muscle fibers. In contrast, chronic muscle fatigue is associated with muscle atrophy and neurogenic impairments, which impair the muscle's ability to contract effectively.[11] This study focuses on acute exercise-induced muscle fatigue, the expression of which is influenced by exercise intensity, duration, and concurrent alterations in muscle activation, vascular function, bioenergetics, intracellular signaling, and molecular mechanics.

Maximal oxygen uptake (VO_2max) is a well-established indicator of aerobic capacity, reflecting the efficiency of the cardiovascular and respiratory systems in sustaining high-intensity exercise.[12,13] A decline in VO_2max is closely associated with the onset of muscle fatigue, making it a relevant measure for evaluating endurance performance.[14] Blood lactate concentration is a practical biomarker of acid–base balance and metabolic stress, with elevated levels indicating hydrogen ion accumulation and redox disturbances that impair muscle contraction.[10,15] Both VO_2max and blood lactate are therefore widely recognized as physiological markers of muscle fatigue.[16–18] Their combined use provides a comprehensive assessment of aerobic performance and metabolic fatigue, offering timely and relevant insights into how brewed coffee may modulate performance and fatigue resistance. This is particularly urgent in addressing the current research gap on the physiological effects of brewed coffee in sedentary males performing heavy physical activity.

Due to its well-documented capacity to reduce fatigue and enhance physical performance, coffee is widely utilized in the athletic domain. The primary active compound in coffee, caffeine, is a xanthine alkaloid known for its psychoactive stimulant properties.[19,20] Faturochman et al. (2020) demonstrated that electrolyte-carbohydrate beverages can facilitate the clearance of lactic acid, while supplementation with vitamins B1, B6, and B12 enhances carbohydrate and protein metabolism and increases oxygen delivery to muscle tissues.[21] These interventions collectively contribute to the mitigation of muscle fatigue during exercise. Furthermore, caffeine acts as a potent ergogenic aid through multiple mechanisms, including antagonism of adenosine receptors, mobilization of intracellular calcium stores, and inhibition of phosphodiesterase activity.[22] The chemical composition of coffee is complex, comprising carbohydrates, sucrose, pectin, starch, pentosan, hemicellulose, cellulose, lignin, lipids, proteins (N x 6.25), tannic acid, N-methylnicotinate, caffeine, phenolic acids such as chlorogenic acid, among others.[23] Gayo Arabica coffee, which contains caffeine concentrations ranging from 0.8% to 1.4%, is selected for this study due to its relatively low acidity, rendering it more suitable and safer for consumption.[24]

Considering the widespread consumption of coffee and the high levels of sedentary behavior observed in certain populations, it is pertinent to study the potential effects of brewed coffee on fatigue-related physiological responses. Lasmana et al. (2022) reported that medical students exhibit significantly higher sedentary behavior compared to students from other faculties, spending an average of 385.6 minutes per day engaged in activities such as sitting or lying down.[25] Prolonged sedentary behavior is well-documented to contribute to increased physical fatigue and impaired exercise performance. Additionally, epidemiological observations within the local academic environment reveal a high prevalence of brewed coffee consumption among male medical students. In a prior study conducted among 297 university students, 52.5% of respondents exhibited a high frequency of coffee consumption ($\geq$ 3–4 times per week).[26]

The concurrence of elevated sedentary time and frequent coffee intake in this population makes male medical students an appropriate group for investigating the effects of brewed coffee on fatigue-related physiological parameters following physical activity. This study aims to investigate the acute effects of brewed coffee consumption on exercise-induced fatigue markers, specifically blood lactate levels and maximal oxygen uptake (VO_2 max), in sedentary male medical students following a standardized exercise protocol. By assessing these parameters, the study seeks to explore the potential of brewed coffee as a functional beverage to reduce exercise-induced fatigue. The findings are expected to provide preliminary evidence to guide future research involving athletic populations and contribute to the development of nutritional strategies aimed at enhancing stamina and preventing fatigue.

MATERIAL AND METHODS

Study Design and Subject

This study employed a quasi-experimental design with a post-test only control group. The research was conducted in November 2024 at the Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Aceh, Indonesia. The study population consisted of male individuals aged 18–22 years who voluntarily agreed to participate, met the sedentary criteria according to the Sedentary Behaviour Questionnaire (SBQ), and were confirmed to be physically and mentally healthy based on anamnesis and clinical examination performed by a physician. Male participants were selected because physical fitness is influenced by sex, and including both male and female participants could introduce variability that may confound the assessment of acute physiological responses.[27] Restricting the sample to males ensured a more homogeneous group, allowing clearer interpretation of the effects of brewed coffee on VO_2max and blood lactate as markers of fatigue. The exclusion criteria were as follows: individuals who were intolerant to coffee, had chronic diseases (e.g., cardiovascular disease, hypertension), had musculoskeletal injuries or a history of such injuries, regularly consumed coffee (>2 cups/day) or other sources of caffeine, including tea, energy drinks, caffeinated soft drinks, chocolate, or caffeine-containing medications/supplements, or were smokers. Regular caffeine consumption was excluded to minimize potential confounding effects on the study outcomes.

The total population consisted of 298 male students from the Faculty of Medicine. Of this total population, 22 subjects met the inclusion criteria. After providing informed consent (one day before the study), the subjects were divided into two groups. Eleven subjects were assigned to the intervention group, and eleven others were assigned to the control group. On the day of the study, four subjects from the intervention group did not attend, and two subjects from the control group could not continue the study due to physical and mental health issues. Therefore, the final total number of subjects was 16, with 9 in the control group and 7 in the intervention group. The subject recruitment procedure is illustrated in Figure 1.

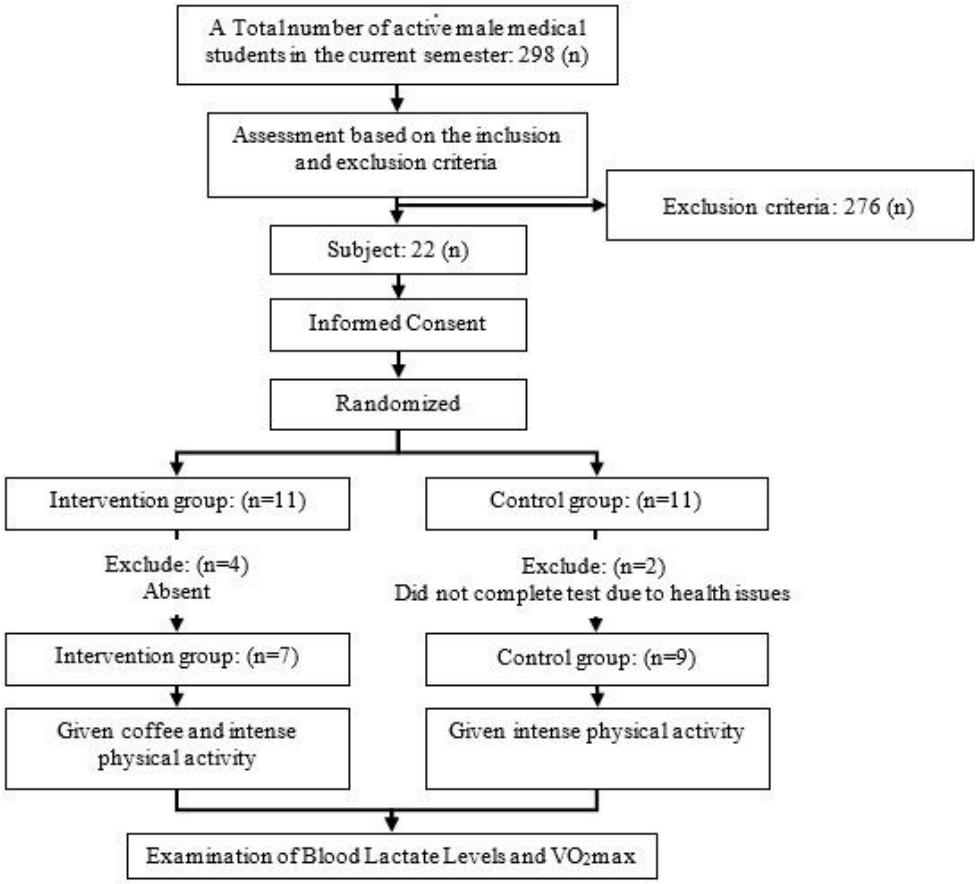

Figure 1. Subject recruitment procedure

Intervention and Examination

The treatment for the intervention group consisted of administering a single serving brewed coffee and engaging in standardized physical activity. Participants received 10 g of Arabica Gayo coffee, brewed with 150 mL of boiling water, yielding an estimated caffeine content of approximately 281 mg per serving, based on the caffeine concentration reported for Arabica coffee by Olechno et al. (2021).[28] Coffee was administered 1–2 hours before the physical activity, after which participants performed standardized physical activity, operationalized as the Rockport test. The control group performed the same standardized physical activity without coffee administration. The intervention was administered only once.

The coffee provided was brewed coffee (local term: *tubruk*), prepared without added sugar or other ingredients. Brewed coffee was chosen for preparation because it is a widely consumed beverage globally, including in Indonesia, and is popular for its caffeine content and stimulant properties.[28] Additionally, the *tubruk* method is easy to prepare and does not require special equipment. The coffee used was Arabica Gayo coffee, purchased directly from a producer in Gayo (Bener Meriah Village, Central Aceh District, Aceh, Indonesia).

The measurements of blood lactate concentration and VO_2max were also conducted only once, immediately after the exercise protocol. This single-exposure design was chosen to specifically investigate the acute physiological effects of brewed coffee on muscle fatigue. The study focused on two established fatigue markers: blood lactate and VO_2max. A repeated or long-term intervention was not used. The primary aim was to assess immediate metabolic and aerobic responses following coffee ingestion in sedentary males performing heavy physical activity, not to evaluate chronic effect or adaptations.

VO2max was estimated based on the time taken to complete a 1-mile (1,609 m) run, following the Ministry of Health guideline (2017). VO_2max was estimated using the Rockport 1-mile (1,609 m) walk test, following the protocols described by Kumar and Rowland.[29,30] Blood lactate levels were measured immediately after the Rockport 1-mile test using the Edge Blood Lactate Meter. For the measurement, a test strip was inserted into the meter, and the participant's hand and fingers were gently massaged toward the puncture site to form a blood drop. The finger was then pricked with a lancet, and the blood was applied to the sensor. Results were displayed within approximately 45 seconds, after which the test strip was removed and disposed of as infectious waste. Measurements were automatically saved by the device.

All participants were instructed to obtain adequate sleep (6–8 hours within 24 hours) before the study and to refrain from consuming coffee or other caffeinated beverages, such as tea, soft drinks, and energy drinks, for at least 24 hours before the examination. Before the intervention, anthropometric measurements, including weight, height, and Body Mass Index (BMI), as well as systolic and diastolic blood pressure and heart rate, were recorded. BMI was calculated using the formula: body weight (kg) divided by the square of height (m^2).[31]

Statistical Analysis

Data analysis was conducted using SPSS software. Normality was assessed with the Shapiro–Wilk test ($p > 0.05$), and homogeneity with Levene's test ($p > 0.05$). VO_2max data were both normally distributed and homogeneous, while blood lactate data were normal but not homogeneous. An independent samples t-test ($p < 0.05$) was then used to compare post-intervention blood lactate levels and VO_2max between the intervention and control groups to assess the effect of coffee consumption.

Ethical Clearance

This study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Syiah Kuala, Banda Aceh, Indonesia (Approval No. 153/EA/FK/2024).

RESULT

Analysis of the physical characteristics of the research subjects, as presented in Table 1, included demographic and baseline variables such as age, height, weight, body mass index (BMI), systolic blood pressure, diastolic blood pressure, and initial heart rate. The results indicated no statistically significant differences between the intervention and control groups in age ($p = 0.352$), height ($p = 0.280$), weight ($p = 0.765$), BMI ($p = 0.854$), systolic blood pressure ($p = 0.527$), and diastolic blood pressure ($p = 0.344$). However, a statistically significant difference was observed in the initial heart rate ($p = 0.050$).

Table 2 presents the results of the analysis examining the effect of coffee consumption combined with intense physical activity on the outcome variables. The findings revealed a significant effect on blood lactate levels ($p = 0.018$), with the intervention group demonstrating a mean increase of 15.04 mmol/L compared to

10.60 mmol/L in the control group. In contrast, no significant effect was found on VO_2max (p = 0.297), with mean values of 39.57 mL/kg/min in the intervention group and 37.62 mL/kg/min in the control group.

Table 1. Subject Demographic in Intervention and Control Group

Characteristic	Group	N	Mean ± SD	p-value
Age (years)	Intervention	7	20.28 ± 1.60	0.352
	Control	9	19.55 ± 1.42	
Weight (kg)	Intervention	7	170.85 ± 4.48	0.280
	Control	9	167.66 ± 6.36	
Height (cm)	Intervention	7	59.14 ± 10.66	0.765
	Control	9	57.22 ± 13.68	
BMI (kg/m^2)	Intervention	7	20.27 ± 3.64	0.854
	Control	9	19.93 ± 3.51	
Systolic blood pressure (mmHg)	Intervention	7	114 ± 9.34	0.527
	Control	9	117.44 ± 11.33	
Diastolic blood pressure (mmHg)	Intervention	7	73.42 ± 4.64	0.344
	Control	9	75.55 ± 4.03	
Heart Rate	Intervention	7	78.14 ± 5.33	0.050*
	Control	9	88.22 ± 11.46	

Note: * = Significant difference; BMI = Body Mass Index

Table 2. Differences in Blood Lactate Levels and VO_2max Between the Control and Intervention Groups

Variabel	Group	N	X ± SD	F	p-value
Lactate	Intervention	7	15.04 ± 2.38	7.585	0.018*
	Control	9	10.60 ± 4.10		
VO_2max	Intervention	7	39.57 ± 2.37	4.199	0.297
	Control	8	37.62 ± 4.17		

Note: * = Significant difference

DISCUSSION

Coffee is a complex beverage containing a wide range of chemical constituents, including carbohydrates, sucrose, pectin, starch, pentane, hemicellulose, cellulose, lignin, lipids, protein N x 6.25, tannic acid, N-methyl nicotinic acid, caffeine, phenolic acids (e.g., chlorogenic acid), among others.[23] Caffeine, one of the most widely consumed stimulants globally, is recognized for enhancing endurance and reducing perceived fatigue. Caffeine increases plasma adrenaline levels and promotes the mobilization of free fatty acids (FFAs), thereby providing a readily available energy source during exercise.[32] Pharmacokinetically, caffeine is rapidly absorbed via the gastrointestinal tract and readily crosses the blood–brain barrier due to its lipophilic properties.[18] According to Antonio et al. (2024), caffeine begins to manifest physiological effects upon reaching peak plasma concentrations, typically within 15–120 minutes following oral intake.[33] Peak absorption occurs within approximately 45 minutes, although interindividual variations, such as gastric emptying rates, may alter this timing. In healthy adults, caffeine exhibits a plasma half-life of approximately 5 hours and is eliminated within 1.5–9.5 hours.[33] In this study, vigorous physical activity was initiated 15 minutes post-coffee ingestion, considering the short duration of the intervention.

Caffeine impacts multiple physiological systems, including the central nervous system, skeletal muscle, cardiovascular system, renal function, and pulmonary system.[18] Its metabolism primarily occurs in the liver via the cytochrome P450 enzyme CYP1A2, converting caffeine into theobromine, paraxanthine, and theophylline. Structurally analogous to purines, caffeine competitively binds to adenosine receptors (predominantly A2A), thereby increasing intracellular cyclic adenosine 3',5'-monophosphate (cAMP) concentrations and modulating inflammatory responses. Low caffeine doses may exacerbate inflammation, whereas higher doses can attenuate inflammatory mediators through phosphodiesterase inhibition and elevated cAMP levels. Increased cAMP promotes lipolysis by hydrolyzing triglycerides into glycerol and FFAs. By stimulating the sympathetic nervous system, caffeine enhances catecholamine release, elevates plasma glucose, and augments intracellular calcium availability.[18]

Effect of Coffee and Intense Physical Activity on Blood Lactate Levels

The present study demonstrated a significant elevation in blood lactate levels in participants consuming coffee before intense exercise. The mean lactate concentration in the intervention group was 4.44 mmol/L, surpassing that of the control group. These findings align with Abbotts et al. (2023), in which 20 adults received caffeine (6 mg/kg) 60 minutes before moderate-intensity exercise, resulting in higher post-exercise lactate levels compared to placebo.[34] Similarly, Moreno et al. (2020) reported a 14.3 ± 3.6% increase in fingertip lactate levels following caffeine ingestion before an ergometer test (25 W/min).[12] Yamada et al. (2022) further demonstrated that caffeine (6 mg/kg) consumed one hour before a cycling endurance test increased lactate and plasma epinephrine levels.[35] Caffeine activates adenosine A2 receptors, which stimulate ryanodine receptor channels (RyR), promoting Ca^{2+} release in skeletal muscle. This calcium signaling activates Ca^{2+}/calmodulin-dependent protein kinase II (CaMKII) and IV (CaMKIV) and, together with elevated cAMP, enhances glycogen utilization and mitochondrial biogenesis, inducing metabolic stress and subsequent lactate accumulation.[18,35]

A meta-analysis of 21 studies confirmed that caffeine, combined with submaximal exercise, elevates blood lactate levels. Caffeine antagonizes adenosine, whose concentration rises during exercise due to ATP hydrolysis. Adenosine modulates glucose homeostasis, lipid metabolism, central nervous system activity, cardiovascular function, and respiration, linking metabolic and psychological responses. Lactate levels during exercise represent a balance between production and clearance. The precise mechanism by which caffeine increases lactate—via impaired clearance or augmented gluconeogenesis, remains incompletely elucidated.[36]

Caffeine exhibits ergogenic effects across aerobic, anaerobic, and mixed exercise modalities.[37] Ergogenic benefits are consistently observed at doses of 3–6 mg/kg, particularly in endurance activities such as cycling, walking, cross-country skiing, and swimming.[38] Campos et al. (2022) reported increased lactate levels during anaerobic exercise (Wingate test) following caffeine ingestion.[39] Under anaerobic conditions, phosphocreatine serves as the primary energy source; its depletion elevates ADP, activates glycolysis, and increases intramuscular H^+ accumulation. Elevated H^+ competes with Ca^{2+} for myofilament binding, impairing muscle contraction and contributing to fatigue.[39]

Caffeine enhances myoplasmic Ca^{2+} availability and neuromuscular activation through adenosine receptor antagonism. This effect mitigates H^+-induced inhibition of contraction, sustaining ATP generation via glycolysis and phosphagen systems, thereby increasing lactate production. Additionally, caffeine exerts analgesic effects, reducing perceived exertion.[39] Lactate generated via glycolysis follows the reaction:

$$\text{Glucose} + 2\ \text{ADP} + 2\ \text{Pi} \rightarrow 2\ \text{Lactate} + 2\ \text{ATP} + 2\ H_2O$$

During intense exercise, lactate serves as a substrate for gluconeogenesis, being transported to the liver and kidneys and reconverted to glucose, which then contributes to ATP and GTP synthesis, enhancing metabolic rate.[40]

Theofilidis et al. (2018) demonstrated that lactate accumulation may induce metabolic acidosis, reducing Ca^{2+} release and uptake in the sarcoplasmic reticulum, decreasing Ca^{2+} sensitivity, and impairing glycolytic enzyme activity, thereby contributing to muscle fatigue.[10] However, lactate is not the sole determinant of fatigue, as ex vivo muscle studies in lactic acid solutions showed no contraction inhibition. Lactate accumulation primarily establishes the lactate threshold, correlating with fatigue and reflecting increased muscle acidity and glycogen depletion.[10] Reactive oxygen species (ROS) also contribute to fatigue by directly impairing sarcolemmal depolarization, Ca^{2+} regulation, myofilament sensitivity, and actomyosin interactions, and indirectly by disrupting bioenergetics and blood supply.[10]

Effect of Coffee and Intense Physical Activity on VO_2max

The findings showed no significant difference in VO_2max between the coffee group (39.57 mL/kg/min) and the control group (37.62 mL/kg/min). These results are consistent with those of Brietzke et al. (2017), who also found no significant improvement in VO_2max following caffeine intake (43.3 vs. 43.6 mL/kg/min in caffeine versus placebo groups).[17] The lack of effect may be attributable to the relatively low coffee dose of 2 mg/kg body weight, insufficient to elicit measurable improvements in aerobic capacity. Studies demonstrating VO_2max enhancement typically employed higher caffeine doses administered 60 minutes pre-exercise. For instance, Aji et al. (2023) reported increased VO_2max following 4 mg/kg caffeine ingestion 1–2 hours before a beep test, with faster metabolism subjects achieving peak VO_2max earlier.[41] Similarly, Stadheim et al. (2021) observed improved VO_2max and a 63% enhancement in performance after

4.5 mg/kg caffeine 45 minutes pre-exercise.[42] Usman et al. (2017) reported a mean VO_2max increase of 3.83 mL/kg/min in badminton athletes after caffeine consumption.[16] Moreno et al. (2020) found acute caffeine intake (3 mg/kg, 60 minutes pre-test) led to a 6.1 ± 8.5% higher VO_2max compared to controls.[12]

VO_2max is influenced by oxygen delivery, regulated via cardiac output (heart rate × stroke volume).[43] Caffeine modulates all four adenosine receptor isoforms, but the role of adenosine in exercise-induced heart rate increase is unclear.[44] Caffeine-induced elevations in plasma adrenaline and norepinephrine enhance heart rate, while bronchodilation via adenosine receptor antagonism improves ventilation.[45] Additionally, caffeine increases maximal oxygen deficit accumulation and lactate production, collectively supporting endurance during high-intensity aerobic activity.[42] Beyond VO_2max, maximal fat oxidation influences aerobic performance. Enhanced fat oxidation conserves glycogen stores, supporting prolonged exercise. Caffeine promotes endurance by: (1) augmenting mobilization and oxidation of FFAs through elevated epinephrine, (2) antagonizing adenosine receptors (A1, A2A, A2B) to reduce pain perception and delay fatigue, (3) enhancing motor neuron activity to improve muscle contraction and alertness, and (4) increasing muscle oxygen saturation, which optimizes fat utilization and supports VO_2max during high-intensity exercise.[14]

CONCLUSIONS

Single-dose brewed coffee intake increased blood lactate concentrations but did not significantly affect VO_2 max in sedentary males with physical activity treatment. These findings indicate that acute coffee intake may influence metabolic responses, suggesting that further longitudinal research is needed to determine its chronic effects on muscle fatigue and physical fitness.

ACKNOWLEDGMENT

We are grateful to Universitas Syiah Kuala for providing financial support for this study through the 2024 Professor Research Grant, which was issued on May 3, 2024, and had the number 167/UN11.2.1/PG.01.03/SPK/PTNBH/2024.

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