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The Effect of Caffeine Consumption on Post-Sprint Blood Lactate Levels

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Abstract

Caffeine is a widely used ergogenic aid known to enhance both physical and cognitive performance. Its potential to influence lactate accumulation during high-intensity exercise, such as sprinting, remains an area of interest.This study aimed to investigate the effect of 100% Arabica caffeine consumption on blood lactate levels following sprint activity.Using a pretest-posttest control group design, 32 sprint athletes from KONI Madiun (divided into treatment and control groups) participated in a 30-meter sprint test repeated three times. The treatment group consumed 3–6 mg/kg body weight of caffeine in the form of an Americano one hour prior to testing. Blood lactate levels were measured post-sprint using a lactate analyzer. Independent t-tests were conducted with a significance level of $p < 0.05$. The treatment group consistently exhibited lower lactate levels compared to the control group across all sprint stages. Statistically significant differences were observed in Sprint 1, Sprint 2, and the total lactate accumulation while Sprint 3 showed no significant difference ($p = 0.072$). The findings suggest that caffeine consumption before sprinting may reduce lactate accumulation and improve metabolic efficiency. Caffeine intake in the form of 100% Arabica Americano has a significant effect in lowering blood lactate levels during repeated high-intensity sprint efforts. This supports its potential use as an ergogenic aid in anaerobic sports. However, variability in individual response highlights the need for tailored supplementation strategies and further investigation into long-term physiological adaptations.

Keywords: Caffeine; coffee; ergogenic;lactate; sports; sprint

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Introduction

Caffeine is one of the most widely used ergogenic substances to enhance physical and cognitive performance. As a stimulant compound that acts on the central nervous system, caffeine can increase alertness, reduce fatigue, and improve endurance during physical activity (Shabir et al., 2022). One common source of caffeine is arabica coffee, which contains a lower caffeine concentration than robusta, yet still provides significant stimulant effects (Matsumura et al., 2023). In sports, particularly in high-intensity activities such as a 100-meter sprint, the body relies on anaerobic energy systems that produce lactic acid as a byproduct of metabolism (Truong et al., 2025).

The accumulation of lactic acid is often associated with muscle fatigue, which can hinder athletic performance. Therefore, various strategies have been developed to reduce lactic acid buildup and accelerate muscle recovery after physical activity (Sumedha et al., 2024). One such strategy worth investigating is the consumption of 100% Arabica caffeine before training or competition. Several studies have shown that caffeine can enhance energy metabolism by stimulating the release of catecholamines, which promote glycogen breakdown and the use of fat as an energy source (Truong et al., 2025). Additionally, caffeine is known to affect the central nervous system by reducing perceived exertion and improving muscle work capacity.

However, the specific effects of 100% Arabica caffeine on post-100-meter sprint lactate accumulation have not been extensively studied (Mor et al., 2024). Caffeine is an alkaloid compound commonly found in coffee, tea, and several energy drinks. As a central nervous system stimulant, caffeine has various effects on physical and mental performance, including its impact on energy metabolism and lactic acid accumulation (Ramesh, 2024). Physiologically, caffeine works by inhibiting adenosine, a neurotransmitter responsible for inducing fatigue and inhibiting the release of excitatory neurotransmitters such as dopamine and norepinephrine (Naoi et al., 2025).

By blocking adenosine, caffeine enhances alertness, reduces fatigue, and increases central nervous system activity (Taghizadeh et al., 2024). In the context of sports, particularly high-intensity efforts like the 100-meter sprint, caffeine boosts catecholamine release (adrenaline and noradrenaline), which helps mobilize energy substrates such as glucose and free fatty acids (Spriet et al., 2020). One of the main mechanisms associated with lactic acid production and accumulation is the increase in anaerobic metabolism during high-intensity activity (Muhammed et al., 2021). Caffeine can promote fat utilization as an energy source through lipolysis stimulation, potentially reducing muscle glycogen dependence (Schaik et al., 2021).

Consequently, the conversion of glycogen to pyruvate and then to lactic acid under anaerobic conditions may decrease, limiting post-sprint lactate elevation (Matsumura et al., 2023). Caffeine is also linked to enhanced buffering capacity in the body, aiding in neutralizing hydrogen ions (H^+) produced during lactic acid production, thereby reducing muscle fatigue effects (Muhammed et al., 2021). Psychologically, caffeine also plays a crucial role in the perception of fatigue. Several studies indicate that caffeine intake can reduce the Rate of Perceived Exertion (RPE), allowing individuals to feel stronger and less fatigued even when blood lactate levels increase (Ferretti & di Prampero, 2026).

This effect enables athletes to maintain training intensity longer before experiencing significant fatigue. While caffeine can reduce lactic acid accumulation and improve muscle endurance, these effects may vary based on dosage, individual tolerance, and genetic factors (Antonio et al., 2024). Some studies have also reported that high caffeine doses may increase lactic acid production due to greater glycogenolysis stimulation (Schaik et al., 2021). Therefore, it is essential to determine the optimal dose that provides benefits without excessively increasing lactate levels. Overall, caffeine can influence lactate levels through physiological mechanisms such as fat mobilization, glycogen breakdown suppression, and enhanced buffering capacity.

Psychologically, it helps reduce fatigue perception, allowing athletes to perform at peak levels despite increased lactate production. A deeper understanding of caffeine's effects on energy metabolism can aid athletes and coaches in designing optimal nutritional strategies to improve sports performance. The research question of this study is does consuming 100% arabica caffeine on repeated sprint activity decreased blood lactate levels? H_o consumption of 100% arabica caffeine does not decrease affect post-sprint blood lactate levels. H_a consumption of 100% arabica caffeine decrease affects post-sprint blood lactate levels.

Methods

This research used an experimental method with a posttest only with control group design. It aimed to determine the effect of caffeine consumption on lactate levels after sprinting. The study applied purposive sampling with the following criteria Age 19-20 years, Healthy and free of metabolic diseases (verified with a statement letter), No caffeine sensitivity (also verified with a statement letter). The study population consisted of 36 sprint athletes from KONI in Madiun City and Regency. Participants meeting the inclusion criteria were selected and divided into two groups the experimental group and the control group. The experimental group consumed 100% Arabica caffeine from the Hayati brand, prepared as an Americano and consumed at a dosage of 3mg/kg body weight (Guest et al., 2021).

The control group received no caffeine. Data were analyzed using an independent t-test with a significance level of $p < 0.05$. The caffeine dosage protocol was also standardized based on body weight. Participants in the experimental group received 100% Arabica caffeine at a fixed dose of 3 mg/kg body weight approximately 60 minutes before the sprint test. This dosage was selected based on recommendations from the International society of sports nutrition and previous studies reporting that moderate caffeine intake effectively improves anaerobic exercise performance while minimizing adverse side effects. Standardizing the dose based on body weight ensured that each participant received a proportional amount of caffeine relative to their physiological characteristics.

In addition, participants completed a 24-hour caffeine intake questionnaire prior to testing. This questionnaire was used to monitor habitual caffeine consumption from coffee, tea, energy drinks, chocolate, or supplements that could potentially affect baseline physiological responses. Participants were instructed to avoid caffeine-containing products for at least 24 hours before the experiment to reduce confounding effects related to prior caffeine intake. The warm-up procedure was also standardized to ensure consistency across participants. Before

performing the sprint test, all participants completed a structured warm-up consisting of 10 minutes of light jogging followed by 5 minutes of dynamic stretching exercises. This standardized warm-up protocol was designed to prepare the neuromuscular system, reduce injury risk, and minimize variability in physiological readiness before sprint activity.

Result

The table presents the performance measurement data for two groups experimental and control across three sprint sessions and their total accumulation. In Sprint 1, the control group recorded higher lactate levels (3.22 ± 0.29) compared to the experimental group (2.56 ± 0.34). This trend continued in Sprint 2 and sprint 3, where the control group consistently displayed higher values. The experimental group showed a gradual increase in lactate levels across the sprints (from 2.56 to 2.69), as did the control group. In terms of total accumulation, the control group recorded a significantly higher total of 9.89 ± 0.37 compared to the experimental group's 7.84 ± 0.59. This consistent difference at each stage of the sprint suggests that the intervention in the experimental group was effective. Additionally, the relatively low standard deviation values indicate homogeneity within the groups. The stability of the trend in the experimental group indicates more sustained performance.

Table 1. Two-group statistic

Group	Sprint 1	Sprint 2	Sprint 3	Acumulation
Eksperimen	2.56 ± 0.34	2.59 ± 0.38	2.69 ± 0.32	7.84 ± 0.59
Control	3.22 ± 0.29	3.34 ± 0.26	3.31 ± 0.23	9.89 ± 0.37

The table above displays statistical test results on blood lactate levels (in mmol/L) between the treatment and control groups across the three sprint sessions, using independent t-tests. In the first measurement, the treatment group had a lower mean (2.56 mmol/L) compared to the control group (3.22 mmol/L), with a highly significant p-value (0.00000061). Similar patterns were seen in the second sprint. However, in the third sprint, the difference was not statistically significant (p = 0.07218), although the treatment group still showed a lower average. The total lactate accumulation from sprint 1 to sprint 3 was significantly lower in the treatment group (p = 0.00000017), indicating better lactate management under workload stress.

Table 2. Normality test shapiro-wilk

Vaiable	Sample	Statistic-Shapiro-Wilk	Sig (p)	
Sprint 1	Caffein	0.974	0.904	Normal
	Control	0.960	0.665	Normal
Sprint 2	Caffein	0.930	0.243	Normal
	Control	0.869	0.246	Normal
Sprint 3	Caffein	0.969	0.830	Normal
	Control	0.967	0.792	Normal
Acumulation	Caffein	0.988	0.997	Normal
	Control	0.958	0.627	Normal

The Shapiro–Wilk normality test showed that all variables in both the caffeine and control groups were normally distributed. This is indicated by the significance values ($p > 0.05$) across sprint 1, 2, 3, and accumulation measurements. The caffeine group demonstrated p-values ranging from 0.243 to 0.997, while the control group showed p-values ranging from 0.246 to 0.792. Therefore, the data met the assumption of normality, indicating that parametric statistical analyses could be appropriately applied in the subsequent tests.

Table 3. Homogenity test lavene's

Vaiable	Statistic lavene's	Sig. (p)	
Sprint 1	0.162	0.690	Homogen
Sprint 2	0.002	0.963	Homogen
Sprint 3	4.019	0.104	Homogen
Acumulation	4.201	0.253	Homogen

The levene's test results indicated that all variables had homogeneous variances between the caffeine and control groups. This is demonstrated by the significance values ($p > 0.05$) for sprint 1, 2 3, and accumulation measurements. The obtained p-values ranged from 0.104 to 0.963, confirming that the assumption of homogeneity was fulfilled. Therefore, the data were considered suitable for further parametric statistical analysis.

Table 4. Analysis statistic

Variable	Group	N	Mean (mmol/L)	SD	t-Value	t-Table (α = 0,05; df = 30	p-value
Sprint 1	Treatment	16	2.56	0.34	-6.30	2.042	Sig (p = .00000061)
	Control	16	3.22	0.29			
Sprint 2	Treatment	16	2.59	0.38	-5.67	2.042	Sig (p = .00000355)
	Control	16	3.34	0.26			
Sprint 3	Treatment	16	2.69	0.32	-1.86	2.042	No Sig (p = .07218)
	Control	16	3.31	0.23			
Acumulation	Treatment	16	7.84	0.59	-6.75	2.042	Sig (p = .00000017)
	Control	16	9.89	0.37			

The table presents the statistical test results for lactate levels (in mmol/L) between the treatment and control groups during sprint 1, analyzed using an independent t-test. In the first measurement, the treatment group had an average lactate level of 2.56 mmol/L with a standard deviation of 0.34, whereas the control group showed a higher mean of 3.22 mmol/L with a standard deviation of 0.29. The calculated t-value of -6.30 was smaller than the critical t-value (2.042), and the p-value was extremely low (0.00000061), indicating a statistically significant difference between the groups. This suggests that the training or intervention applied to the treatment group effectively reduced lactate levels compared to the control group. This difference can be interpreted as a positive effect of the treatment on metabolic efficiency during sprinting.

In the second measurement (sprint 2), the treatment group recorded an average lactate level of 2.59 mmol/L (SD = 0.38), while the control group remained higher at 3.34 mmol/L (SD = 0.26). The t-value of -5.67 and p-value of 0.00000355 also indicate a significant result, as the p-value is far below 0.05. This confirms that in the follow-up measurement, the treatment

continued to have a positive effect on reducing lactate levels. This implies that the treatment may have enhanced the body's tolerance to lactic acid accumulation. This effect is particularly relevant in sports contexts, as high lactate levels are often associated with quicker onset of muscle fatigue. These results reinforce previous findings and demonstrate consistent benefits of the treatment.

However, in the third measurement (sprint 3), although the lactate level in the treatment group (2.69 mmol/L) was still lower than in the control group (3.31 mmol/L), the statistical test yielded a t-value of -1.86, which did not exceed the critical threshold (2.042). The p-value of 0.07218 (> 0.05) indicates that the difference was not statistically significant. This suggests that although there was a mean difference, data variation and sample size were not sufficient to rule out the possibility of random chance. It is possible that the effect of the treatment began to diminish, or that other factors such as fatigue, physiological adaptation, or technical measurement issues influenced the outcome. Further investigation is needed to understand the body's response dynamics during this third measurement.

In terms of total lactate accumulation from sprint 1 to sprint 3 (Accumulation), there was once again a significant difference, with the treatment group showing 7.84 mmol/L (SD = 0.59) compared to 9.89 mmol/L (SD = 0.37) in the control group. The t-value of -6.75 indicates a strong difference, supported by an extremely low p-value of 0.00000017. This suggests that after a more intense workload or sprint effort, the treatment group was better able to suppress lactate accumulation compared to the control group. Practically, this shows that the treatment successfully enhanced the body's buffering capacity or anaerobic metabolic efficiency. These results are highly significant in athletic training, as the ability to withstand lactate accumulation is closely linked to sustained performance in high-intensity activities.

Discussion

Based on the analysis, the treatment group consistently showed lower lactate levels than the control group in most sprint measurements. This statistically significant difference in three out of four tests suggests that the caffeine intervention successfully reduced lactate production or enhanced its clearance. These findings align who stated that high-intensity interval training combined with caffeine intake can improve aerobic and anaerobic capacity and accelerate lactate metabolism. Such adaptations may result from physiological changes like increased oxidative enzymes, higher mitochondrial density, and enhanced muscle blood flow, supporting better metabolic efficiency during intense activity (Junior et al., 2021).

Interestingly, in sprint 3, the difference between the groups was not statistically significant, despite lower average levels in the treatment group. This may reflect a nonlinear physiological response to exercise load, possibly influenced by fatigue, recovery time, or measurement timing. Studies noted that lactate metabolism adaptations might plateau before improving again with long-term training (Brooks et al., 2022). The result highlights the importance of considering individual variation in training responses key in sports science and suggests that interpretation should not rely solely on statistical significance but also physiological dynamics.

This finding may be explained through the *lactate shuttle theory*, which proposes that lactate is not merely a metabolic waste product but also an important energy substrate that can be transported and utilized by other tissues such as oxidative muscle fibers, the heart, liver, and brain. During repeated sprint activity, lactate produced by highly active glycolytic muscle fibers can be shuttled to neighboring oxidative fibers and other organs for oxidation and energy production (Brooks, 2023). Therefore, by the third sprint, part of the accumulated lactate may have already been reutilized as an energy source, reducing the observable difference in blood lactate concentration between groups.

The absence of statistical significance in sprint 3 may also indicate that repeated sprint exercise induced a progressive metabolic adaptation response. Lactate turnover increases substantially during high-intensity exercise, and a large proportion of lactate disposal occurs through oxidation rather than simple accumulation in the bloodstream (Maqdasy et al., 2022) . Consequently, after repeated sprint bouts, the body may become more efficient at redistributing and oxidizing lactate, thereby attenuating differences in measured blood lactate levels despite continued physiological stress. This mechanism may explain why caffeine supplementation significantly affected lactate responses during sprint 1 and sprint 2, yet the effect diminished during sprint 3.

In the early sprint phases, caffeine may enhance neuromuscular activation and energy efficiency, resulting in lower lactate accumulation. However, during later sprint repetitions, lactate utilization through intracellular and cell-to-cell lactate shuttling may become increasingly dominant, minimizing differences between groups. Thus, the sprint 3 findings should not necessarily be interpreted as evidence that caffeine lost its physiological effect entirely, but rather that lactate metabolism during repeated sprint exercise is dynamic and influenced by both lactate production and lactate reutilization processes. The fourth measurement (cumulative data) indicates that under more prolonged or intense efforts, the treatment group showed greater lactate tolerance.

This aligns who found that athletes following polarized or HIIT training adapted more effectively to suppress lactate buildup (Bartoloni et al., 2024). These adaptations relate to cardiovascular efficiency and improved muscle capacity to use lactate as an energy source. Therefore, this study supports caffeine as an effective strategy for enhancing performance in sports that demand speed and explosive power, such as sprinting. The findings also emphasize the critical role of load management in achieving physiological adaptation (Hussain, 2022). However, not all research agrees that reduced lactate indicates improved performance. Through the "lactate shuttle" concept, explained that lactate is not merely waste but an important fuel during exercise (Brooks, 2020).

High lactate does not always signal poor performance; its role depends on context and timing. Study also showed that elite runners may exhibit high lactate levels yet maintain peak performance (Certo et al., 2022). Thus, while lower lactate in this study may be seen as beneficial, it must be interpreted alongside overall performance indicators, not as a sole metric. This study has several limitations that should be considered when interpreting the findings. First, the study did not employ a placebo-controlled design. Participants in the experimental group were aware that they consumed caffeinated arabica coffee, which may have introduced

a placebo effect or psychological expectancy that could influence sprint performance and physiological responses.

The absence of a placebo beverage makes it difficult to determine whether the observed effects were entirely caused by caffeine or partially influenced by participant expectations. Second, the sample size was relatively small (n = 32), which may limit the generalizability of the findings to broader athletic populations. A larger sample size could provide greater statistical power and improve the reliability of the observed effects of caffeine on post-sprint blood lactate levels. Third, blood caffeine concentrations were not measured during the study. As a result, individual differences in caffeine absorption, metabolism, and physiological response could not be objectively verified.

Measuring blood caffeine levels would allow researchers to confirm whether participants reached similar caffeine concentrations prior to sprint performance testing. Therefore, future studies are recommended to use a double-blind placebo-controlled design, involve larger sample sizes, and include biochemical measurements of blood caffeine concentration to provide more comprehensive evidence regarding the physiological effects of caffeine on repeated sprint performance and lactate metabolism.

Conclusion

Based on the data analysis, it can be concluded that caffeine intake influences the reduction of blood lactate levels in most sprint measurements. Caffeine consumption significantly reduces blood lactate in early repeated sprints (sprint 1 & 2), but the effect may diminish in subsequent sprints (sprint 3). This suggests that caffeine potentially enhances energy metabolism efficiency during high-intensity activities such as sprinting, likely through mechanisms involving free fatty acid mobilization and glycogen sparing, thereby reducing lactate production. However, one measurement (sprint 3) did not show a statistically significant difference, even though the treatment group's mean remained lower. This indicates that responses to caffeine may vary depending on timing, intensity, or individual physiological conditions.

These findings reinforce that the effectiveness of caffeine as an ergogenic aid is not absolute and may depend on factors such as individual tolerance and training context. Overall, this study supports previous findings suggesting that caffeine acts as an effective ergogenic agent in reducing lactate accumulation, potentially delaying fatigue and enhancing performance in high-intensity physical activity. These results are relevant in athletic training, especially in sports relying on anaerobic energy systems. Nevertheless, caffeine use during training or competition should account for dosage, timing, and individual response to optimize benefits and minimize side effects. Further research is needed to explore long-term adaptation mechanisms to caffeine and its effects on other performance indicators beyond lactate levels. Future studies should measure blood caffeine levels, include female athletes, and examine performance outcomes (e.g., sprint time) alongside lactate.

Author Statement

The authors hereby declare that this manuscript has not been previously published, nor is it under consideration for publication elsewhere

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