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Evaluation of the Effect of Caffeine Consumption on Cognitive Functions by Electroencephalography

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Abstract

Objective: To evaluate cognitive performance and neurophysiological changes after caffeine consumption in individuals who regularly consume different amounts of caffeine.

Materials and Methods: Thirty-seven people aged 18-25 were divided into two groups according to their caffeine consumption as less than 3 mg/kg/day (low consumption group; LC) and more than 3 mg/kg/day (high-consumption group; HC). Electroencephalography (EEG) and Montreal Cognitive Assessment test (MoCA) were performed in both groups (baseline records). One week later, caffeinated (Caff) and decaffeinated (Decaff) coffee were randomly given to LC and HC. Thus, four groups were formed (LC+Caff, LC+Decaff, HC+Caff and HC+Decaff). EEG was repeated one hour after coffee, and MoCA was performed within 20 minutes.

Results: In baseline eyes-opened EEG, the power of beta2 and delta were higher in the LC group than in the HC. After coffee consumption in the eyes-opened EEG, alpha1 and alpha2 power were significantly reduced in the HC+Caff. Following coffee consumption, the MoCA score increased significantly in the HC+Decaff and LC+Caff groups compared with the baseline.

Conclusion: Our study showed that low caffeine consumers had increased brain activity and cognitive performance post consumption high amounts of caffeine. However, this effect was less in high caffeine consumers. Therefore, caffeine habituation may alter the brain's response to caffeine.

Keywords Caffeine · Cognition · Electroencephalography

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INTRODUCTION

Caffeine, a psychoactive substance found in coffee, is consumed daily by 80% of the global population, making it a significant focus in cognitive and physiological research (1, 2). The European Food Safety Authority reported that a person can consume more than 200 mg or 3 mg/kg of caffeine at a time and a maximum of 400 mg of caffeine can be consumed in 24 hours (3). Caffeine's cognitive-enhancing effects are largely mediated through its antagonistic action on adenosine A1 and A2A receptors in the brain, which increases neurotransmitter release and improves attention and alertness (4).

Cognition involves various mental processes, such as memory, executive functions, and attention, which are critical for adapting to new situations (5, 6). These cognitive processes are underpinned by specific patterns of brain activity, which can be effectively studied using electroencephalography (EEG). EEG, a noninvasive technique for measuring brain electrical activity, provides valuable information about neurophysiological changes associated with cognitive processes (7). When examining the EEG frequency bands in more detail, alpha waves from EEG frequency bands are associated with attention and working memory; beta waves are most prominent during wakefulness and the performance of mental functions; delta waves are dominant during deep sleep; and theta waves occur in certain sleep stages and during emotional stress (8). Understanding how caffeine modulates these parameters can provide deeper insights into its effects on cognition (9).

The Montreal Cognitive Assessment (MoCA) is a brief cognitive assessment tool designed to identify mild cognitive impairment and it evaluates various cognitive domains, such as attention, executive function, memory, language, visuospatial ability, numerical skills, and orientation (10).

Some studies have demonstrated significant improvements in attention and memory with moderate caffeine intake, while certain studies report insignificant effects, especially among regular coffee consumers (9, 11). Additionally, EEG studies evaluating the effects of caffeine on brain activity yielded conflicting results, making it difficult to understand caffeine-induced neurophysiological changes (12).

The study hypothesis is that people with habitual high caffeine consumption exhibit higher cognitive performance after consuming caffeinated coffee, whereas this effect is less pronounced in those with habitual low caffeine consumption. The aim of this study was to examine the neurophysiological effects of caffeinated or decaffeinated coffee consumption using EEG and its effects on cognitive functions with the MoCA test in individuals with habitual consumption of low or high caffeine.

MATERIALS AND METHODS

Study Design and Participants

This was a single blended randomized controlled study. Approval was received from the Non-Interventional Clinical Research Ethics Committee of Dokuz Eylul University, with approval number 2020/28-26.

The sample size was calculated using GPower 3.1 software with a power of 90% and an α value of 0.05. The minimum required sample size was estimated to be 17 participants per group, totaling 34 participants. For possible errors, the sample size was increased by 10%, and 37 volunteers were included in the study. Thirty-seven university students (22 females, 15 males) aged 18-25 years participated in the study. Initially, participants were enlisted from a database containing their reported caffeine consumption habits. The participants' demographic characteristics, including age and gender, were documented. We were unable to exclude participants based on their smoking and alcohol consumption. However, to minimize potential confounding effects, participants were instructed to refrain from smoking for at least 12 hours and from alcohol consumption for at least 24 hours before the study.

The participants were then requested to fill out a survey specifically prepared by us to assess their average daily caffeine intake from tea, coffee, cocoa, and caffeinated soft drinks. According to the survey, the participants were categorized into two groups. The low consumption group (LC) included individuals who consumed less than 3 mg/kg/day of caffeine. The high consumption group (HC) included those who consumed more than 3 mg/kg/day caffeine (3). On the first day, EEG recordings were conducted for 5 min with participants' eyes-opened and closed, followed by the MoCA test. The data obtained on the first day were considered as baseline measurements.

Seven days later, each main group was randomly and blindly divided into two subgroups. One subgroup received caffeinated coffee containing 200 mg caffeine (Caff), while the other received decaffeinated coffee (Decaff). In this case, the study was consisted of four groups: LC+Caff, LC+Decaff, HC+Caff, and HC+Decaff. One hour after coffee consumption, EEG recordings were taken with eyes-opened and closed for 5 minutes each. After quickly removing the EEG cap, the MoCA test was performed within 20 min, and the study was terminated.

EEG recording was performed using an electrocap with 19 silver chloride electrodes (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4 T4, T5, P3, Pz, P4, T6, O1 and O2). The electrodes were placed on the cap in accordance with the International 10-20 system and gel was used to increase the conductivity. EEG data were recorded in the range of 0-30 Hz, recorded using a Mitsar-EEG-201 system (Mitsar Co. Ltd. Saint Petersburg, Russia). WinEEG program (Version 2.130.101 Mitsar Co. Ltd. St. Petersburg, Russia) compatible with the system was used as the recording program. EEG activity was recorded in the resting and sitting position.

The MoCA test, which is scored out of 30 points, considers a score of 21 or above to be normal (13). In the MoCA test repeated 1 week later, modifications were made to certain sections to minimize recall effects. Due to the complex structure of the MoCA, which includes words, pictures, and sequences, these adjustments were designed to reduce recall bias while preserving the reliability of the test. For instance, in the word recall task, words starting with different letters were used, and in the number-letter matching task, the positions of the numbers and letters were altered.

Statistical Analyses

Statistical analysis was conducted using the IBM SPSS 20.0 software package (IBM Corp., Armonk, NY, USA).

The MoCA test scores were compared using the paired samples t-test. The WinEEG program was used for the analysis of EEG data. EEG epochs with artifacts were determined in two stages, automatic and manual, and they were excluded from the evaluation. In the first step, epochs with amplitudes exceeding 100 µV were marked and automatically excluded. In the second stage, the remaining artifact epochs were reviewed in more detail and manually excluded from the evaluation. The spectral power was calculated using the fast fourier transform (FFT) method. After calculating all 19 electrodes' average spectrum power, the frequency was separated into delta (0.5-3.9 Hz), theta (4.0-7.4 Hz), alpha1 (7.5-9.9 Hz), alpha2 (10.0-13.9 Hz), beta1 (14.0-19.9 Hz), and beta2 (20.0-30.0 Hz). In the statistical evaluation, the power and frequency changes between the two measurements of the waves obtained from the EEG records of the four groups were compared using repeated measures ANOVA (RM-ANOVA). Post hoc analyses were conducted using the Bonferroni method. The baseline EEG recordings of the LC and HC groups were compared using multivariate ANOVA (MANOVA). Data obtained from EEG recordings of caffeinated and decaffeinated coffee consumers were analyzed using MANOVA. EEG results recorded with eyes open and closed were evaluated separately. The findings were assessed with a confidence level of 95%, and values for $p<0.05$ were considered statistically significant.

RESULTS

A total of 37 participants, including 22 females and 15 males, were enrolled in the study, with a mean age of 22.24 ± 0.33 years. According to the survey, 17 participants (11 females) in the LC group had a mean age of 21.88 ± 1.99, whereas 20 participants (11 females) were in the HC group with a mean age of 22.5 ± 2.06.

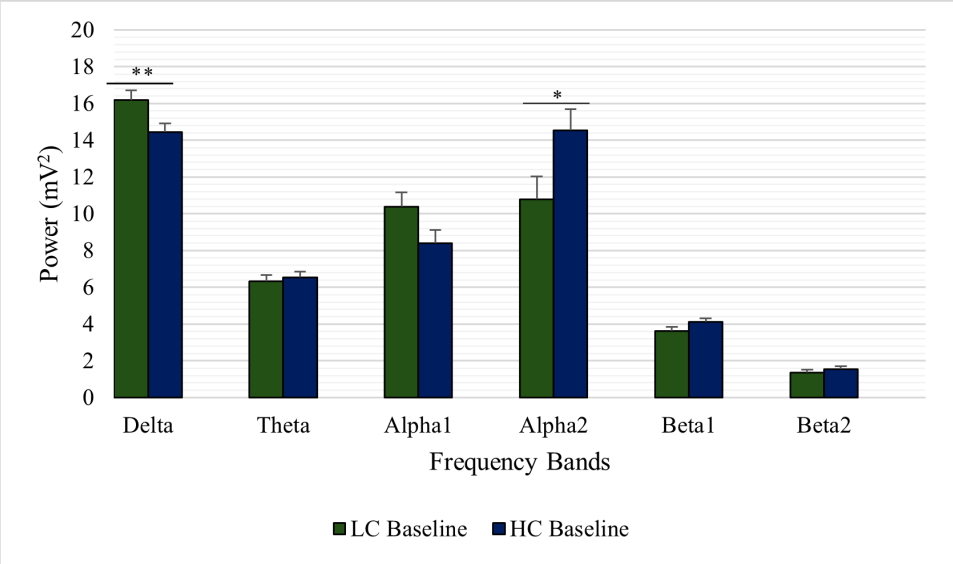

Figure 1. Baseline eyes-closed EEG findings for the LC and HC groups, MANOVA.
*the power of alpha2 was significantly higher (p=0.026),
**the power of delta was significantly lower (p=0.014) in the HC group when compared with LC group.

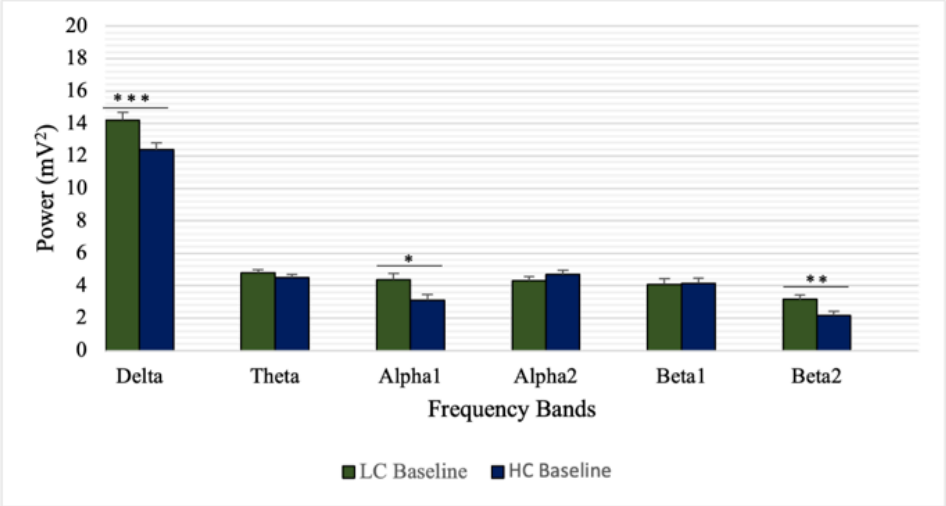

Figure 2. Baseline eyes-opened EEG findings for the LC and HC groups, MANOVA. *the power of alpha1 was significantly higher (p=0.016), **the power of beta2 was significantly higher (p=0.013), *** the power of delta was significantly higher (p=0.005) in the LC group when compared with HC group.

When divided into four groups, 9 participants (6 females) were in the LC+Caff group with a mean age of 21.77 ± 1.92, 8 participants (5 females) in the LC+Decaff group with a mean age of 22.00 ± 2.20, 10 participants (6 females) in the HC+Caff group with a mean age of 22.50 ± 2.27 and 10 participants (5 females) in the HC+Decaff group with a mean age of 22.60 ± 1.95.

EEG Results

In the baseline eyes closed EEG condition, the power of alpha2 was significantly higher in the HC group compared with the LC group ($F_{1,7}$=5.003; p=0.026), and the power of delta was significantly lower ($F_{1,7}$=6.31; p=0.014) (Figure 1). In the baseline eyes-opened condition, the power of alpha1 ($F_{1,7}$=5.885; p=0.016), beta2 ($F_{1,7}$=6.248; p=0.013), and delta ($F_{1,7}$=8.014; p=0.005) was significantly higher in the LC group than in the HC group (Figure 2).

In the eyes-closed EEG condition, the power of delta ($F_{1,3}$=19.504; p<0.0001) and theta ($F_{1,3}$=6.976; p=0.009) was significantly lower, while the power of beta2 ($F_{1,3}$=10.459; p=0.001) was significantly higher in the LC+Caff group after consuming coffee compared with the LC+Decaff group (Figure 3). In the eyes-closed condition of the HC+Decaff group, it was found that the power of alpha2 was significantly decreased compared with the HC+Decaff group's own baseline ($F_{1,9}$=9.166; p=0.014) (Figure 4). In the eyes-opened condition of the HC+Caff group, the power of alpha1 was significantly decreased compared with baseline ($F_{1,9}$=9.630; p=0.013).

In the eyes-opened condition of the LC+Caff group, the power of beta2 significantly increased compared with the LC+Caff group's own baseline ($F_{1,8}$=5.687; p=0.044). In the eyes-opened condition of the LC+Decaff group, it was found that the power of beta2 significantly decreased compared with the LC+Decaff group's own baseline ($F_{1,7}$=12.557; p=0.009) (Figure 5). In the eyes-opened after coffee intake, the power of alpha1 ($F_{1,3}$=11.310; p=0.001) and alpha2 ($F_{1,3}$=17.835; p<0.0001) were significantly lower, and the power of delta ($F_{1,3}$=9.985; p=0.002) was higher in the HC+Caff group compared with the HC+Decaff group (Figure 6). In the eyes closed condition after coffee intake, the power of alpha1 ($F_{1,3}$=6.443; p=0.012) and alpha2 ($F_{1,3}$=4.653; p=0.032) were significantly lower in the HC+Caff group compared with the HC+Decaff group.

Table 1. Difference in MoCA scores between baseline and after coffee

Groups	n	Baseline Mean ± SE	After coffee Mean ± SE
LC+Caff	9	23.5 ± 1.43	26.1 ± 0.93*
LC+Decaff	8	22.5 ± 1.25	24.9 ± 1.11
HC+Caff	10	24.9 ± 1.23	25.5 ± 0.93
HC+Decaff	10	24.5 ± 0.83	26.5 ± 1.02**

*p=0.038, **p=0.002 compared with the baseline, paired samples t-test. MoCA: Montreal Cognitive Assessment test; LC: Low caffeine consumption group; HC: High caffeine consumption group; Caff: Caffeinated coffee; Decaff: Decaffeinated coffee; SE: Standart error.

MoCA Test Results

A significant increase was observed in MOCA test scores after caffeine intake in LC+Caff (p=0.038) and HC+Decaff (p=0.002) groups compared with the baseline (Table 1).

DISCUSSION

Many studies investigating the effect of caffeine on alpha waves have indicated that caffeine reduces alpha power in humans and rats (14, 15). In our study, alpha1 and alpha2

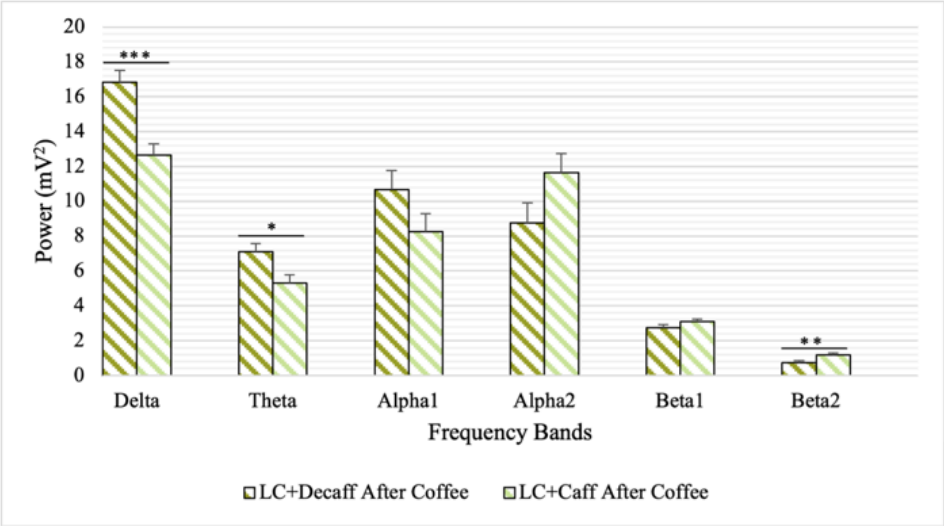

Figure 3. Eyes closed EEG after coffee consumption findings for the LC+Caff and LC+Decaff groups, MANOVA. *the power of theta was significantly lower (p=0.009), **the power of beta2 was significantly higher (p=0.001), ***the power of delta was significantly lower (p=0.000) in the LC+Caff group when compared with LC+Decaff group.

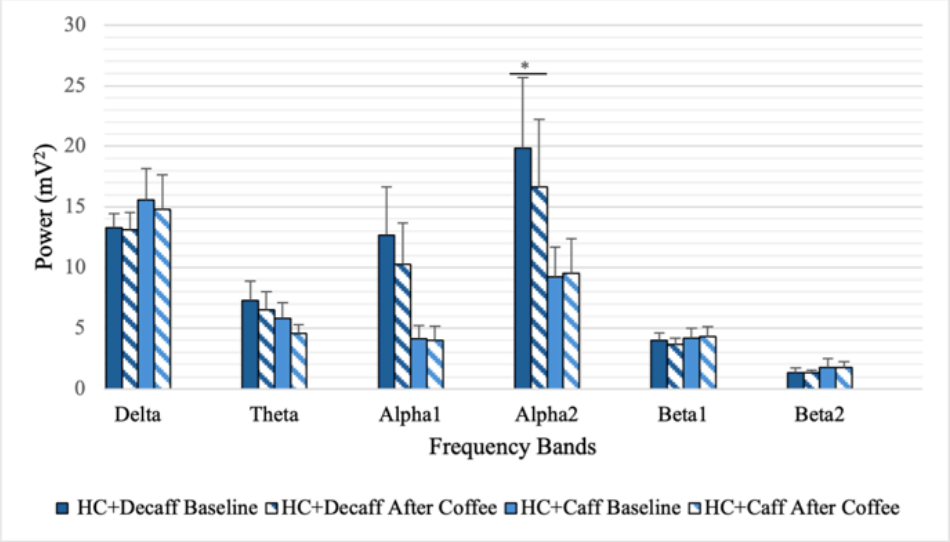

Figure 4. The comparison between the baseline and after coffee consumption in eyes-closed EEG findings for the HC+Caff and HC+Decaff groups (RM-ANOVA). *the power of alpha2 was significantly lower (p=0.014) in the HC+Decaff group when compared with baseline.

power were significantly lower in the HC+Caff group than in the HC+Decaff group in both eyes closed and opened EEG. In the eyes-opened EEG findings of the HC+Caff group, alpha1 power was significantly reduced after coffee consumption compared to the HC+Caff group's own baseline. However, in the LC+Caff group with both eyes open and closed EEG recordings, no significant decrease in alpha1 and alpha2 power were observed compared with the LC+Decaff group. Unlike our study, Foxe et al. showed that caffeine consumption (50 mg) significantly reduced alpha power in individuals consuming less than 3 mg/kg caffeine per day (16). In another study, caffeine consumption (200 mg) caused a significant decrease in alpha1 and alpha2 power in both eyes-opened and closed EEG recordings in subjects consuming 300 mg or more of caffeine daily (17). Since individuals who consume less coffee have a minimal regular intake of caffeine, their responses to the acute effects of caffeine may be less pronounced. These individuals may not have developed caffeine tolerance to the same extent as those who consume more coffee and thus may exhibit a typical neurophysiological response to caffeine without significant changes in alpha power. While both our study and previous research point to caffeine's potential impact on alpha power, the varying results may reflect differences in participant characteristics,

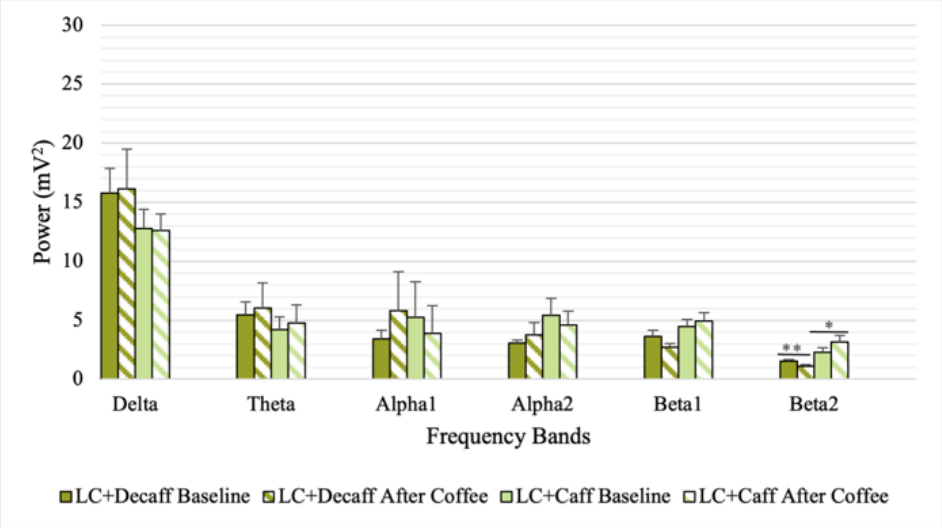

Figure 5. The comparison between the baseline and after coffee consumption in eyes-opened EEG findings for the LC+Caff and LC+Decaff groups (RM-ANOVA). *the power of beta2 was significantly higher (p=0.044) in the LC+Caff group when compared with baseline, **the power of beta2 was significantly lower (p=0.009) in the LC+Decaff group when compared with baseline.

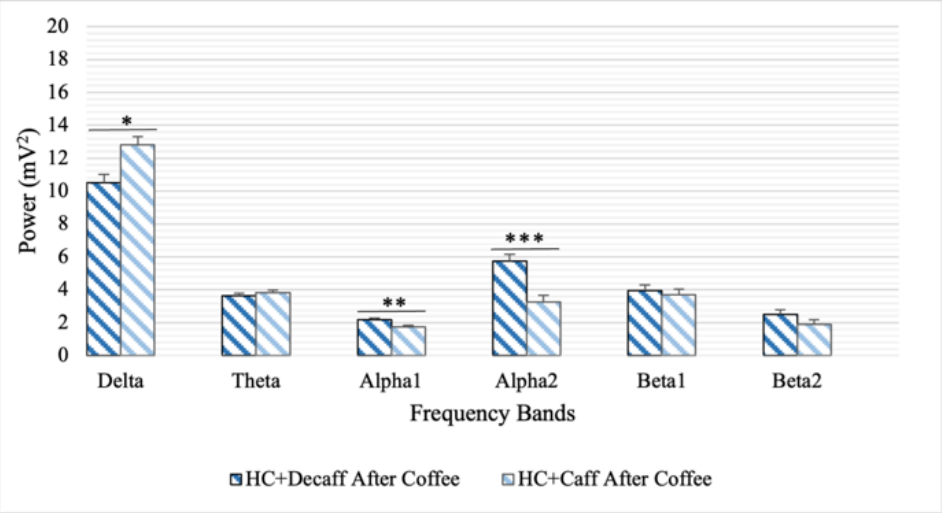

Figure 6. Eye-opened EEG after coffee consumption findings for the HC+Caff and HC+Decaff groups, MANOVA. *the power of delta was significantly higher (p=0.002), **the power of alpha1 was significantly lower (p=0.001), ***the power of alpha2 was significantly lower (p=0.000) in the HC+Caff group compared with the HC+Decaff group.

caffeine consumption levels, and the development of caffeine tolerance, all of which may modulate caffeine's effect on brain activity.

Research findings indicate that caffeine increases the power of beta, which is associated with an increase in mental activity (18). In a study conducted with women who consumed large amounts of coffee (150 mL×6.4 cups/day), EEG was recorded by giving different amounts of caffeine to the subjects. It was found that the power of the beta wave was significantly higher in groups that consumed highly caffeinated coffee (19). In our study, it was observed that in the eyes-closed EEG record of the LC+Caff group, the power of beta2 increased significantly after the intake of caffeine compared with the LC+Caff group's own baseline. In addition, in the eyes-closed EEG record, the power of beta2 was significantly higher in the LC+Caff group than in the LC+Decaff. However, no significant increase in beta power was observed in the HC+Caff group compared with the HC+Decaff group. An increase in beta power was not observed in the HC+Caff and HC+Decaff groups when compared with their own baseline. High caffeine consumption may lead to caffeine tolerance in individuals, which can attenuate the effect of caffeine on beta power.

In the findings of our study, it was observed that in the baseline recordings with both eyes-opened and closed, the

delta power was higher in the LC group than in the HC group. In the eyes-closed EEG data, the power of delta was significantly lower in the LC+Caff group than in the LC+Decaff group. Furthermore, no significant decrease in eyes-closed delta power was observed in the HC+Caff group compared with the HC+Decaff group. Siepmann et al. showed that delta power was significantly lower in those consuming 200 mg/day of caffeinated coffee (17). In another study, caffeine consumption (200-400 mg/day) significantly reduced delta wave power in EEG recordings, both with eyes open and closed (20). The observed reduction in delta power in the LC+Caff group is consistent with previous studies suggesting that caffeine has a stimulating effect, which may lead to a decrease in the power of slow-wave activity such as delta waves (21).

The lack of a significant change in delta power in the HC+Caff group indicates that high caffeine exposure may limit this effect.

Conversely, the different results of our study regarding eyes-opened EEG findings, the power of delta was significantly higher in the HC+Caff group than in the HC+Decaff group. This result is in contrast to the anticipated reduction in delta power associated with caffeine consumption. A possible explanation could be the development of tolerance in habitual high-dose caffeine consumers, leading to altered neurophysiological responses. This divergence from the expected outcomes highlights the need for further research to explore how chronic caffeine consumption modifies brainwave activity and its implications for cognitive function.

In our study, we observed a significant improvement in cognitive performance in the LC+Caff group following coffee intake, which suggests that individuals with lower habitual caffeine consumption may benefit more from its cognitive-enhancing effects. Haskell et al.'s study (22) included groups with low and high caffeine intake (less than 50 mg/day and more than 50 mg/day, respectively). Although no significant differences were observed in cognitive performance. The study highlighted the complexity of caffeine's effects, which might depend on the dosage, individual sensitivity, and baseline caffeine consumption. However, it should be noted that the low caffeine intake in Haskell's study might not have been sufficient to elicit noticeable changes in cognitive performance, which could support the differing results in our study. A separate study involving college students who consumed caffeine chronically compared cognitive performance between one half of the group, which consumed 4 mg/kg/day of caffeine, and the other half, which abstained (23). In contrast to our findings, this study did not report a notable distinction in cognitive performance between chronic caffeine consumers and non-consumers. Zhang et al. evaluated cognitive performance after administering 3, 6, or 9 mg/kg of caffeine and found that only the group consuming 3 mg/kg showed a significant improvement in cognition (24). This suggests that lower caffeine doses may be particularly effective for enhancing cognitive performance in individuals with lower baseline caffeine intake, similar to the findings of our study where significant cognitive improvement was observed in the LC+Caff group.

These contrasting results may reflect differences in study design or caffeine dosages. Notably, our results indicate that regular high caffeine consumers (HC+Caff group) might develop tolerance to caffeine's cognitive benefits, supporting the hypothesis that the effects of caffeine are more pronounced in individuals with lower baseline consumption. This tolerance mechanism could explain the absence of significant cognitive enhancement in high-consumers, as also claimed by previous literature (25). Further research is required to explore the interplay between habitual caffeine intake, tolerance development, and cognitive performance.

In our study, we observed a significant increase in cognitive performance in the HC+Decaff group, which could potentially be influenced by placebo-like effects. This finding highlights a potential interplay between expectations, caffeine consumption habits, and cognitive outcomes.

Wang et al. reported that individuals consuming 3 mg/kg of caffeine demonstrated better cognitive performance than those consuming 6 or 9 mg/kg (26). The researchers attributed this discrepancy to potential side effects associated with higher doses of caffeine, such as increased anxiety, jitteriness, or overstimulation, which may counteract its cognitive benefits. Interestingly, the placebo effect observed in our HC+Decaff group could be explained by the strong association between habitual caffeine consumption and expected cognitive benefits. In high caffeine consumers, the perception of receiving caffeine even in its absence may have triggered a performance boost through psychological or physiological mechanisms. These findings, when considered along with Zhang et al. and Wang et al.'s results, suggest that both the caffeine dose and individuals' baseline consumption levels are critical factors influencing cognitive performance (24, 26). Furthermore, the reduced effectiveness of higher caffeine doses in the aforementioned studies might reflect a ceiling effect, where excessive caffeine begins to impair rather than enhance cognitive function.

However, our findings did not support our hypothesis. Nevertheless, the findings shed light on the possibility of different mechanisms regarding how caffeine tolerance and consumption influence consumers' expected cognitive outcomes. Future research could explore how the placebo

effect interacts with different levels of habitual caffeine intake and the extent to which subjective expectation influence performance outcomes. These factors are particularly relevant in understanding the nuanced and dose-dependent effects of caffeine on cognition.

We had to perform this work during the timeline of Covid-19 pandemic. Although our sample size was sufficient, according to the power analysis, we were unable to further increase the sample size and enhance the study, as all university students were attending online classes during this period. We could not exclude participants based on smoking and alcohol use. To minimize potential confounding effects, participants were instructed to abstain from smoking for at least 12 hours and from alcohol for at least 24 hours before the study. In the future, we would like to further develop this study, further increase the number of participants (excluding smoking and alcohol use), and interpret the effects of caffeine on cognitive functions in comparison with EEG and other imaging methods.

CONCLUSION

Our findings demonstrate the differential effects of caffeine on cognitive performance and EEG activity based on habitual daily caffeine consumption. Low caffeine consumers exhibited improved cognitive performance and increased beta power, whereas high caffeine consumers showed limited changes, indicating the development of tolerance.

In our study, we found that individuals who regularly consumed low amounts of caffeine showed increased brain activity in EEG recordings and improved cognitive functions after consuming high amounts of caffeine. Individuals who regularly consumed high amounts of caffeine showed a smaller increase in brain activity and cognitive functions following caffeine administration compared with those who routinely consumed low amounts of caffeine. Therefore, cognitive performance, as assessed using the MoCA test in our study, may vary among individuals with different caffeine consumption habits, depending on their usual intake levels.

This is a pilot study investigating the effect of caffeine consumption on neurophysiological findings. Our findings will shed light on further studies with larger numbers of participants and different imaging techniques (e.g. functional magnetic resonance imaging) combined with EEG.

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Ethics Committee Approval: Permission was obtained from the Non-Interventional Clinical Research Ethics Committee of Dokuz Eylul University for the study (Approval number: 2020/28-26).

Peer Review: Externally peer-reviewed.

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