JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

https://doi.org/10.61919/yykg7j65

Original Article

Effect of Oral Caffeine on Eyelid Muscle Activity and Myokymia in Healthy Adults

Ayesha Ikhlaq[1], Khuram Nasir[2], Muhammad Kashif[1], Shahzad Shabbir[1], Habiba Ishaq[1]

[1] Department of Optometry and Vision Sciences, Superior University, Lahore, Pakistan
[2] MS Rehabilitation in Clinical Optometry & Vision Sciences, PhD Scholar, Senior Lecturer, Department of Optometry and Vision Sciences, Superior University, Lahore, Pakistan

***Corresponding author: Khuram Nasir, khuram.nasir@superior.edu.pk**

"Cite this Article" Received: 27 February 2026; **Accepted**: 19 April 2026; **Published**: 16 May 2026

Author Contributions: Concept: AI and KN; Design: KN and MK; Data Collection: AI, SS and HI; Analysis: MK and KN; Drafting: AI, KN, MK, SS and HI. **Ethical Approval**: Superior University, Lahore, Pakistan. **Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest. **Funding**: No external funding; **Data Availability**: Available from the corresponding author on reasonable request; **Acknowledgments**: N/A.

ABSTRACT

Background: Caffeine is a widely consumed methylxanthine stimulant that can influence central nervous system activity, neuromuscular excitability, and motor responsiveness. Eyelid myokymia is usually benign but may cause discomfort, anxiety, and reduced concentration, particularly in healthy adults exposed to stimulants, fatigue, stress, or prolonged screen use. **Objective:** To assess the acute effect of oral caffeine on eyelid muscle activity and eyelid myokymia frequency and severity in healthy adults. **Methods:** This quasi-experimental pre-test/post-test study included 50 healthy adults aged 18–40 years. Baseline eyelid muscle activity was measured using surface electromyography, and myokymia frequency and severity were recorded before caffeine intake. Participants then received a standardized 200 mg oral caffeine dose, and post-caffeine measurements were obtained after 30–60 minutes. Pre- and post-caffeine outcomes were compared using paired sample t-tests, and the relationship between eyelid muscle activity and myokymia frequency was assessed using Pearson correlation analysis. **Results:** Mean eyelid muscle activity increased from 2.5 μV before caffeine to 3.1 μV after caffeine, with a mean difference of 0.6 μV ($p = 0.002$). Mean myokymia frequency increased from 1.2 to 1.7 episodes/min, with a mean difference of 0.5 episodes/min ($p = 0.0001$). Myokymia severity remained predominantly mild, and eyelid muscle activity showed a moderate positive correlation with myokymia frequency ($r = 0.45$, $p < 0.05$). **Conclusion:** A single 200 mg oral caffeine dose was associated with significantly increased eyelid muscle activity and myokymia frequency in healthy adults, while severity remained mild. **Keywords:** Caffeine; Eyelid Muscle Activity; Myokymia; Surface Electromyography; Neuromuscular Activity; Healthy Adults.

INTRODUCTION

The eyelids play an essential role in maintaining ocular surface integrity, visual comfort, and protection of the anterior segment through coordinated blinking and periocular muscle activity. Blinking distributes the tear film, clears debris, limits ocular surface exposure, and supports stable visual function during routine activities such as reading, screen use, and conversation.

The orbicularis oculi muscle is central to this process because it mediates voluntary, reflexive, and spontaneous eyelid closure. Even minor disturbances in its neuromuscular rhythm may manifest as fine eyelid twitching, fluttering, or quivering, clinically recognized as eyelid myokymia. Although eyelid myokymia is usually benign and self-limiting, recurrent episodes may cause discomfort, anxiety, reduced concentration, and functional disturbance, particularly among young adults exposed to academic stress, prolonged digital device use, sleep disruption, and stimulant consumption (1,2).

Eyelid myokymia is typically described as a repetitive, involuntary, localized contraction of the eyelid muscles, most commonly involving the orbicularis oculi. It is generally not associated with serious

neurological disease, but its frequent occurrence in otherwise healthy individuals makes it clinically relevant, especially when symptoms are recurrent or perceived as troublesome (3). Commonly reported triggers include fatigue, psychological stress, inadequate sleep, ocular strain, prolonged screen exposure, and caffeine intake.

Among these, caffeine is particularly important because it is widely consumed in the form of tea, coffee, soft drinks, and energy beverages by students and working adults, the same population in which eyelid twitching is commonly reported. Lifestyle-based studies have suggested that caffeine intake may be associated with eyelid myokymia, especially when combined with poor sleep quality, stress, and visual fatigue (4,5).

Caffeine is a methylxanthine stimulant with well-established effects on the central nervous system and neuromuscular excitability. After oral ingestion, caffeine is rapidly absorbed, and its physiological effects commonly appear within 30 to 60 minutes (6). Its primary mechanism involves antagonism of adenosine receptors, thereby reducing inhibitory neuromodulation and increasing neuronal excitability, alertness, and motor responsiveness.

While these effects may improve wakefulness and performance, they may also contribute to restlessness, tremor, increased motor unit firing, and involuntary muscle activity in susceptible individuals (7). Experimental and neurophysiological studies have shown that caffeine can influence motor cortex excitability, neuromuscular transmission, motor unit recruitment, and blink reflex activity, supporting a plausible biological pathway through which caffeine may affect periocular muscle behavior (8).

Previous ocular and periocular research further support a possible relationship between caffeine intake and eyelid motor activity. Studies evaluating blink behavior have suggested that caffeine may alter spontaneous blink rate and ocular surface stability, while investigations involving stimulant exposure and electromyographic assessment indicate that eyelid muscle activity may be responsive to stimulant-related changes in neuromuscular excitability (9).

Cross-sectional studies among university students and young adults have reported higher frequencies of eyelid myokymia among individuals with greater caffeine consumption, particularly when caffeine exposure coexists with sleep disturbance, stress, or prolonged screen time. However, much of the existing literature is observational, questionnaire-based, or focused on general neuromuscular outcomes rather than direct acute measurement of eyelid muscle activity after a standardized caffeine dose (10,11).

This gap is important because clinical advice to reduce caffeine intake in patients with eyelid twitching is common, yet objective evidence directly measuring acute caffeine-related changes in eyelid muscle activity and myokymia frequency remains limited. From a PICO perspective, the relevant population is healthy adults with normal ocular and neurological function; the intervention or exposure is a standardized oral caffeine dose; the comparator is the same participants' pre-caffeine baseline state; and the outcomes are eyelid muscle activity measured by surface electromyography, myokymia frequency, and myokymia severity.

A pre-test/post-test design therefore provides a practical method to examine short-term physiological changes after caffeine exposure while controlling for between-participant variability. Accordingly, this study aimed to determine whether a single 200 mg oral dose of caffeine acutely increases eyelid muscle activity and eyelid myokymia frequency in healthy adults, and whether any increase in frequency is accompanied by clinically meaningful worsening of myokymia severity.

MATERIALS AND METHODS

The study was conducted as a quasi-experimental pre-test/post-test study to evaluate the acute effect of a single standardized oral caffeine dose on eyelid muscle activity and eyelid myokymia in healthy adults. This design was selected because each participant served as their own control, allowing direct

comparison of eyelid muscle activity, myokymia frequency, and myokymia severity before and after caffeine intake while reducing the influence of between-participant variability. The study was carried out at Sardar Eye Trust Hospital, Lahore, over a six-month period in a controlled clinical setting suitable for ocular and periocular neuromuscular assessment.

A total of 50 healthy adult participants were recruited using a non-probability convenience sampling technique. Eligible participants were adults aged 18 to 40 years of either sex who had normal ocular and neurological function, normal baseline eyelid function, and were willing to participate after receiving information about the study procedures. Participants were excluded if they had a history of facial nerve palsy, epilepsy, chronic eyelid myokymia, blepharospasm, recent ocular infection, eyelid inflammation, ocular disease affecting eyelid movement, or any neurological disorder that could independently influence periocular muscle activity.

Participants using medications known to affect neuromuscular function, including muscle relaxants or antiepileptic drugs, were also excluded (12). Additional exclusion criteria included known caffeine sensitivity, habitual caffeine intake greater than 400 mg per day, pregnancy, lactation, and sleep duration of less than five hours before testing, as these factors could influence eyelid twitching, neuromuscular excitability, or response to caffeine.

The sample size was calculated using the formula for a single population proportion at a 95% confidence level, with a Z value of 1.96, an estimated population proportion of 0.069, and a margin of error of 0.07, resulting in a final sample of 50 participants (3). Participants were screened according to the eligibility criteria before enrollment.

The purpose of the study, nature of the procedure, caffeine intervention, measurement process, possible transient symptoms, and right to withdraw were explained to all eligible participants. Written informed consent was obtained before data collection. Each participant completed a structured proforma documenting demographic characteristic, age, sex, caffeine consumption history, medical history, ocular history, neurological history, sleep duration before testing, and adherence to pre-test caffeine abstinence.

All participants were instructed to abstain from caffeine for 12 to 24 hours before testing to minimize the effect of recent caffeine exposure on baseline measurements. Data collection was performed in a quiet clinical room with stable lighting and comfortable room temperature to reduce external triggers of eyelid twitching. Participants were seated comfortably and asked to remain relaxed before baseline recording. Surface electromyography was used to record eyelid muscle activity from the periocular region corresponding to the orbicularis oculi muscle.

Baseline eyelid muscle activity was recorded before caffeine intake and expressed in microvolts. Baseline myokymia frequency was recorded as the number of visible or detected eyelid twitching episodes per minute. Myokymia severity was graded using an ordinal severity scale in which lower scores represented absent or minimal twitching and higher scores represented increasing clinical intensity of twitching.

After baseline assessment, each participant received a standardized oral caffeine dose of 200 mg. The same caffeine dose was administered to all participants to maintain uniform exposure. Participants remained under observation in a relaxed seated position for 30 to 60 minutes after caffeine intake, corresponding to the expected period of acute physiological response.

Post-caffeine measurements were then obtained using the same procedure as baseline assessment. Eyelid muscle activity was again measured by surface electromyography, myokymia frequency was recorded as episodes per minute, and myokymia severity was graded using the same ordinal scale. The use of identical pre- and post-caffeine measurement procedures was intended to maintain consistency across repeated observations.

The primary outcome variable was change in eyelid muscle activity measured by surface electromyography before and after caffeine intake. Secondary outcome variables included change in myokymia frequency, change in myokymia severity, and the relationship between eyelid muscle activity and myokymia frequency.

Eyelid muscle activity was operationally defined as the recorded surface electromyographic activity of the orbicularis oculi region in microvolts. Myokymia frequency was operationally defined as the number of eyelid twitching episodes per minute during the observation period. Myokymia severity was operationally defined as the graded clinical intensity of eyelid twitching based on the predefined ordinal severity scale. Adverse effects following caffeine intake were recorded using a checklist that included headache, restlessness, palpitations, gastric discomfort, and other symptoms reported by participants.

Potential sources of bias and confounding were addressed through standardized eligibility criteria, caffeine abstinence before testing, uniform caffeine dose, consistent timing of post-caffeine assessment, and repeated measurements within the same participants.

Participants with heavy caffeine intake, known caffeine sensitivity, inadequate sleep, neurological disorders, ocular conditions, chronic eyelid twitching, or medication use affecting neuromuscular activity were excluded to reduce confounding. Environmental conditions during testing were kept consistent, and the same outcome definitions were applied before and after caffeine intake. Data were recorded on structured forms to reduce variation in documentation and to support consistency across participants.

Data were entered and analyzed using SPSS version 26. Descriptive statistics were used to summarize demographic characteristics, caffeine consumption history, ocular and medical history, baseline eyelid muscle activity, post-caffeine eyelid muscle activity, myokymia frequency, myokymia severity, and adverse effects.

Frequencies and percentages were calculated for categorical variables, while means, medians, modes, standard deviations, and variances were calculated for continuous or ordinal study variables as appropriate. Because the same participants were measured before and after caffeine intake, paired sample t-tests were used to compare pre-caffeine and post-caffeine eyelid muscle activity and myokymia frequency. Pearson correlation coefficient was used to assess the relationship between eyelid muscle activity and myokymia frequency. Statistical significance was set at $p < 0.05$.

Data integrity was maintained through standardized data collection procedures, uniform caffeine administration, consistent timing of measurements, and structured recording of all study variables. Completed proformas were reviewed for completeness before data entry.

Missing or incomplete observations were handled by excluding the affected variable from the relevant analysis while retaining available complete data for other analyses. Ethical conduct was maintained by obtaining informed consent from all participants, protecting participant confidentiality, allowing voluntary withdrawal at any stage, and monitoring participants for adverse effects after caffeine intake.

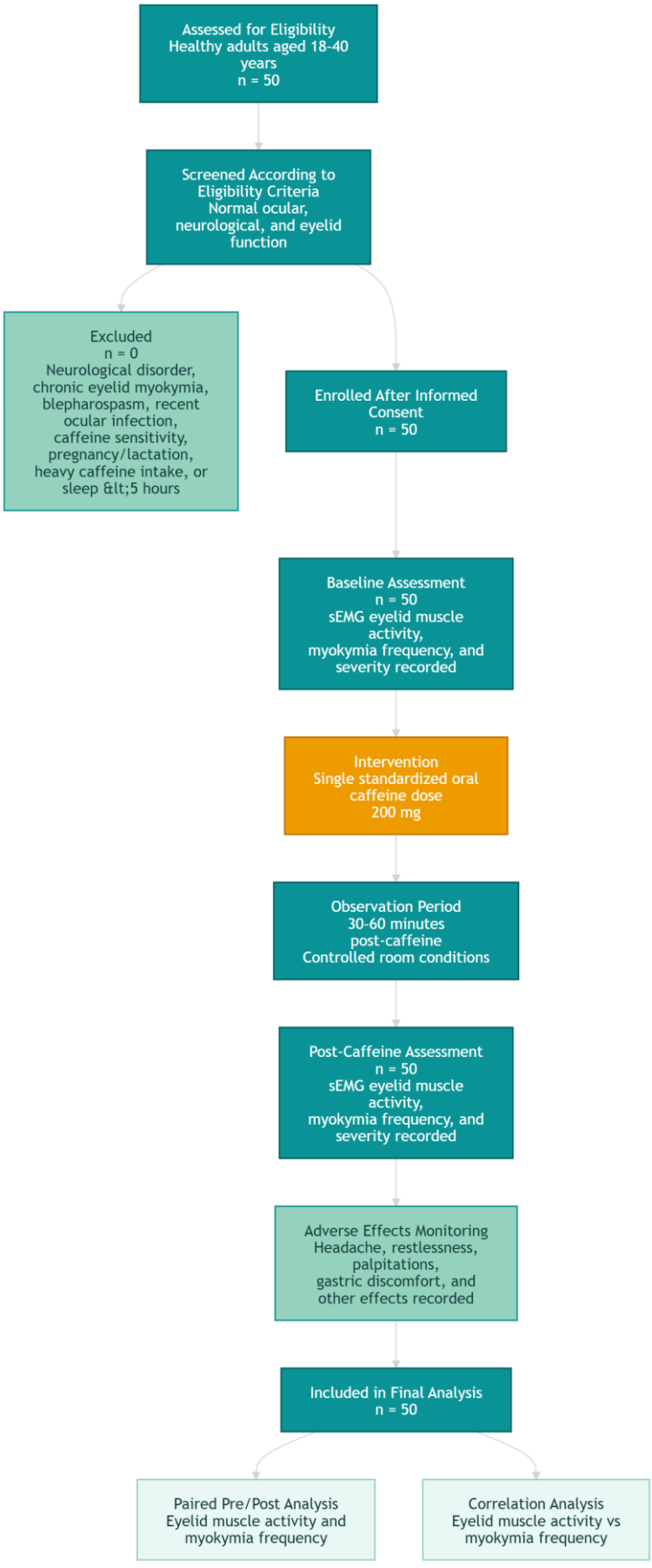

Figure 1. CONSORT Flow Diagram of Participant Enrollment, Caffeine Intervention, Follow-Up, and Final Analysis

RESULTS

A total of 50 healthy adults were included in the analysis. The study population was almost equally distributed by sex, with 26 females (52.0%) and 24 males (48.0%). The largest age subgroup was 21–25 years, comprising 18 participants (36.0%), followed by 26–30 years with 14 participants (28.0%). Most participants reported regular caffeine intake, with 20 participants (40.0%) consuming 2–3 cups/day and 16 participants (32.0%) consuming 1 cup/day. Only 4 participants (8.0%) reported no habitual caffeine consumption. Most participants had no relevant medical or ocular history (47/50; 94.0%). After caffeine intake, adverse effects were uncommon; headache was the most frequent symptom, reported by 5 participants (10.0%), followed by restlessness in 3 participants (6.0%) and palpitations in 2 participants (4.0%).

Table 1. Participant Characteristics and Post-Caffeine Adverse Effects

Variable	Category	Frequency (n)	Percentage (%)
Age group	18–20 years	8	16.0
	21–25 years	18	36.0
	26–30 years	14	28.0
	31–35 years	6	12.0
	36–40 years	4	8.0
Sex	Male	24	48.0
	Female	26	52.0
	Other	0	0.0
Medical/ocular history	History of eyelid myokymia	2	4.0
	Ocular disease/eyelid disorder	1	2.0
	Neurological disorder	0	0.0
	No medical/ocular history	47	94.0
Habitual caffeine intake	None	4	8.0
	1 cup/day	16	32.0
	2–3 cups/day	20	40.0
	>3 cups/day	10	20.0
Post-caffeine adverse effects	Headache	5	10.0
	Restlessness	3	6.0
	Palpitations	2	4.0
	Gastric discomfort	1	2.0
	Other effects	1	2.0

At baseline, the mean eyelid muscle activity was 2.5 μV, with a median of 2.4 μV and standard deviation of 0.8 μV, indicating relatively stable pre-caffeine orbicularis oculi activity. After caffeine intake, mean eyelid muscle activity increased to 3.1 μV, with a median of 3.0 μV and standard deviation of 1.0 μV. Myokymia frequency also increased after caffeine exposure, rising from a baseline mean of 1.2 episodes/min to 1.7 episodes/min post-caffeine. Myokymia severity remained low overall, increasing only slightly from a mean score of 0.15 before caffeine to 0.20 after caffeine.

Table 2. Descriptive Statistics of Study Outcomes Before and After Caffeine Intake

Outcome	Time Point	Mean	Median	Mode	Standard Deviation	Variance
Eyelid muscle activity, sEMG (μV)	Pre-caffeine	2.5	2.4	2.0	0.8	0.64
Eyelid muscle activity, sEMG (μV)	Post-caffeine	3.1	3.0	3.2	1.0	1.00
Myokymia frequency (episodes/min)	Pre-caffeine	1.2	1.0	0	0.5	0.25
Myokymia frequency (episodes/min)	Post-caffeine	1.7	2.0	1	0.6	0.36
Myokymia severity score	Pre-caffeine	0.15	1.0	1	0.2	0.05
Myokymia severity score	Post-caffeine	0.20	3.0	2	0.2	5.00

The paired comparison demonstrated a statistically significant increase in eyelid muscle activity after caffeine intake. Mean sEMG activity increased by 0.6 μV, from 2.5 μV pre-caffeine to 3.1 μV post-caffeine.

The paired t-test showed this change was statistically significant (t = 3.33, df = 49, p = 0.002), with a moderate standardized paired effect size (Cohen's dz = 0.47). Myokymia frequency also increased significantly, with a mean rise of 0.5 episodes/min, from 1.2 episodes/min to 1.7 episodes/min. This increase was statistically significant (t = 4.50, df = 49, p = 0.0001) and showed a moderate paired effect size (Cohen's dz = 0.64).

Table 3. Paired Comparison of Pre-Caffeine and Post-Caffeine Outcomes

Outcome	Pre-Caffeine Mean	Post-Caffeine Mean	Mean Difference	SD of Difference	SE of Difference	95% CI for Mean Difference	t-value	df	p-value	Cohen's dz
Eyelid muscle activity, sEMG (μV)	2.5	3.1	+0.6	0.8	0.18	0.24 to 0.96	3.33	49	0.002	0.47
Myokymia frequency (episodes/min)	1.2	1.7	+0.5	0.6	0.10	0.30 to 0.70	4.50	49	0.0001	0.64
Myokymia severity score	0.15	0.20	+0.05							

Baseline myokymia severity was minimal in most participants. Before caffeine intake, 47 participants (94.0%) had no myokymia, 2 participants (4.0%) had mild myokymia, and 1 participant (2.0%) had moderate myokymia. After caffeine intake, 35 participants (70.0%) had no myokymia, while 10 participants (20.0%) had mild symptoms, 4 participants (8.0%) had moderate symptoms, and 1 participant (2.0%) had severe symptoms. Although the frequency of twitching increased after caffeine exposure, the overall severity profile remained predominantly absent-to-mild.

Table 4. Distribution of Myokymia Severity Before and After Caffeine Intake

Myokymia Severity	Pre-Caffeine n (%)	Post-Caffeine n (%)
None	47 (94.0)	35 (70.0)
Mild	2 (4.0)	10 (20.0)
Moderate	1 (2.0)	4 (8.0)
Severe	0 (0.0)	1 (2.0)
Total	50 (100.0)	50 (100.0)

Correlation analysis showed a positive relationship between eyelid muscle activity and myokymia frequency. The overall reported correlation between eyelid muscle activity and myokymia frequency was r = 0.45, indicating a moderate positive association. This suggests that participants with higher eyelid muscle activity tended to show greater myokymia frequency. The relationship was statistically significant at $p < 0.05$. A stronger post-caffeine association was also reported, with the correlation increasing to r = 0.56, indicating that the relationship between neuromuscular activity and twitching frequency became more pronounced after caffeine exposure.

Table 5. Correlation Between Eyelid Muscle Activity and Myokymia Frequency

Analysis Condition	Variables Compared	Correlation Coefficient (r)	Direction/Strength	p-value
Overall analysis	Eyelid muscle activity and myokymia frequency	0.45	Moderate positive correlation	<0.05
Post-caffeine condition	Eyelid muscle activity and myokymia frequency	0.56	Moderate positive correlation	<0.05

Overall, caffeine intake was followed by measurable increases in both primary physiological outcomes. Eyelid muscle activity increased by 24.0%, from 2.5 μV to 3.1 μV, while myokymia frequency increased by 41.7%, from 1.2 to 1.7 episodes/min. The statistically significant paired differences indicate that the acute post-caffeine period was associated with greater eyelid muscle activation and more frequent eyelid twitching episodes, while severity remained largely mild and adverse effects were infrequent.

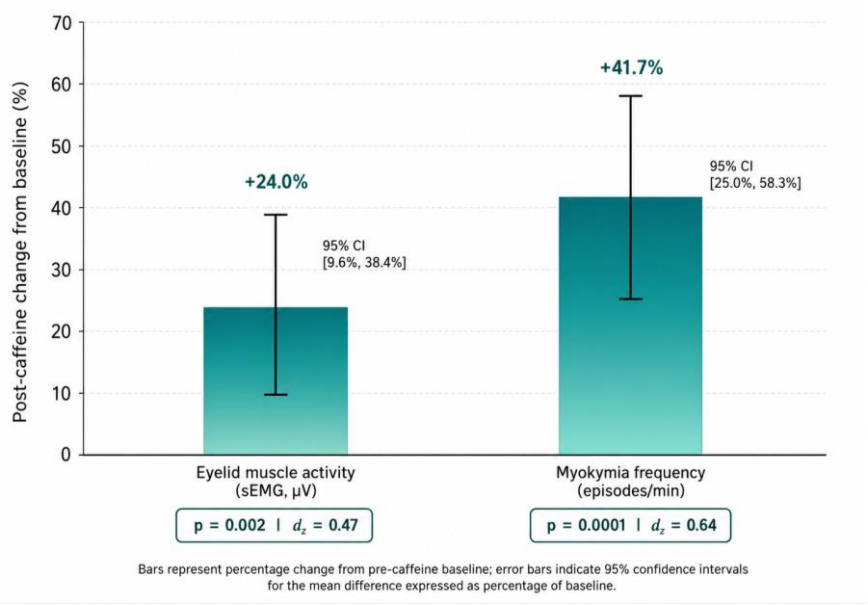

Figure 2. Acute Post-Caffeine Percentage Change in Eyelid Muscle Activity and Myokymia Frequency

The acute post-caffeine response showed a larger proportional rise in myokymia frequency than in eyelid muscle activity. Eyelid muscle activity increased by 24.0% from baseline, with a moderate paired effect size (dz = 0.47; p = 0.002), while myokymia frequency increased by 41.7%, with a stronger paired effect size (dz = 0.64; p = 0.0001). The confidence intervals indicate that both outcomes shifted upward after caffeine intake, with the frequency response showing the greater relative clinical gradient.

DISCUSSION

The present study demonstrated that a single standardized oral caffeine dose was followed by measurable acute increases in eyelid muscle activity and eyelid myokymia frequency among healthy adults. Mean eyelid muscle activity increased from 2.5 μV before caffeine to 3.1 μV after caffeine, representing a mean rise of 0.6 μV, while myokymia frequency increased from 1.2 to 1.7 episodes/min, with a mean difference of 0.5 episodes/min. Both changes were statistically significant, indicating that caffeine exposure was associated with increased periocular neuromuscular activity in the acute post-ingestion period. The magnitude of change was modest but consistent with the expected stimulant profile of caffeine, particularly its ability to increase neuronal excitability and motor responsiveness through adenosine receptor antagonism and downstream effects on central and peripheral neuromuscular pathways (13,14).

The increase in eyelid muscle activity after caffeine intake supports the biological plausibility that the orbicularis oculi muscle is responsive to stimulant-related changes in neuromuscular excitability. Caffeine has been reported to influence motor unit recruitment, motor cortex excitability, and reflexive motor responses, which may explain the observed elevation in surface electromyographic activity after ingestion (15). In this study, the post-caffeine rise in sEMG activity suggests that caffeine may lower the threshold for periocular muscle activation or increase spontaneous low-grade motor activity in the eyelid region. Although the absolute increase of 0.6 μV was not large, its statistical significance indicates a consistent within-participant direction of change, supporting the concept that even a commonly consumed caffeine dose may produce detectable physiological effects in small periocular muscles (16).

The observed rise in myokymia frequency further strengthens the interpretation that caffeine may act as an acute trigger for eyelid twitching in susceptible individuals. Myokymia frequency increased by approximately 41.7% from baseline, compared with a 24.0% increase in eyelid muscle activity, suggesting that twitch frequency may be more sensitive than average sEMG amplitude to acute stimulant exposure. This finding is consistent with previous clinical and observational reports linking caffeine intake with eyelid myokymia, particularly among young adults, students, and individuals exposed to stress, poor

sleep, and prolonged screen use (17). The moderate positive relationship between eyelid muscle activity and myokymia frequency also supports a coherent physiological pattern: participants with greater periocular muscle activation tended to show more frequent twitching episodes.

Despite the statistically significant increase in myokymia frequency, the clinical severity of twitching remained low. Most participants had no or mild myokymia after caffeine intake, and the mean severity score increased only slightly from 0.15 to 0.20. This distinction between frequency and severity is important. The results suggest that caffeine may increase the likelihood or recurrence of brief eyelid twitching episodes without necessarily producing severe, disabling, or sustained symptoms in healthy adults. Clinically, this supports the common observation that caffeine-related eyelid twitching is usually benign and self-limiting, although it may still be bothersome for individuals who are sensitive to stimulants or exposed to other aggravating factors such as fatigue, stress, and digital eye strain (18,19).

The findings also indicate that caffeine is unlikely to act as an isolated determinant of eyelid myokymia in all individuals. Although mean values increased after caffeine intake, not every participant developed clinically meaningful twitching, and a substantial proportion still reported no myokymia after exposure. This variability may reflect differences in habitual caffeine consumption, caffeine metabolism, sleep quality, stress level, baseline neuromuscular excitability, and ocular surface status. Participants who regularly consume caffeine may respond differently from those with low habitual intake because tolerance can modify the physiological response to a fixed dose. Similarly, individuals with fatigue or high visual demand may have a lower threshold for twitching after caffeine exposure. These interactions may explain why caffeine is commonly described as a contributing or aggravating factor rather than a sole cause of eyelid myokymia (20).

The safety profile observed in the study was generally favorable. Post-caffeine adverse effects were infrequent, with headache reported by 10.0%, restlessness by 6.0%, palpitations by 4.0%, and gastric discomfort by 2.0% of participants. These symptoms are compatible with the known mild stimulant effects of caffeine and did not suggest severe intolerance in the study population. The low adverse-effect frequency is relevant because the administered dose of 200 mg reflects a moderate caffeine exposure comparable to commonly consumed caffeinated beverages. However, individuals with caffeine sensitivity, high baseline anxiety, cardiovascular symptoms, or habitual high caffeine intake may experience different responses, and these groups were not the focus of the present analysis.

Several methodological considerations should be acknowledged when interpreting these findings. The pre-test/post-test design allowed each participant to serve as their own control, which reduced between-person variability and strengthened the assessment of acute within-participant change. However, the absence of a placebo or non-caffeine control condition limits the ability to separate caffeine-specific effects from time effects, expectation effects, repeated measurement effects, or environmental influences. Because participants were aware that caffeine had been administered, subjective awareness and physiological arousal may have influenced twitch frequency or reporting. In addition, although the testing environment was controlled, factors such as recent screen exposure, hydration, stress, sleep quality beyond the exclusion threshold, and individual caffeine tolerance may still have contributed to variability in response.

Another important consideration is the measurement of myokymia severity. While sEMG provides an objective measure of eyelid muscle activity, myokymia severity grading depends on the consistency and clarity of the ordinal scale used. The interpretation of severity would be strengthened by a fully standardized and validated grading system, repeated observations, and assessor masking in future work. Similarly, longer recording periods could provide more stable estimates of twitch frequency, particularly because eyelid myokymia can occur intermittently. Future studies may also benefit from comparing different caffeine doses, including placebo control, assessing habitual caffeine intake as an effect modifier, and evaluating whether sleep deprivation, digital eye strain, or anxiety amplify the caffeine-related response.

Overall, the study provides clinically useful evidence that acute caffeine intake is associated with increased eyelid muscle activity and more frequent eyelid myokymia episodes in healthy adults, while the severity of twitching remains predominantly mild. These findings support the practical clinical advice that individuals experiencing recurrent eyelid twitching may benefit from moderating caffeine intake, particularly when symptoms occur alongside fatigue, stress, or prolonged screen exposure. At the same time, the mild severity pattern suggests that caffeine-related eyelid myokymia in healthy adults is generally not severe but rather reflects a transient increase in neuromuscular excitability.

CONCLUSION

This study concluded that a single 200 mg oral dose of caffeine was associated with a significant acute increase in eyelid muscle activity and eyelid myokymia frequency among healthy adults. Mean eyelid muscle activity increased from 2.5 μV to 3.1 μV, while myokymia frequency increased from 1.2 to 1.7 episodes/min, indicating a measurable stimulatory effect of caffeine on periocular neuromuscular activity. However, the severity of myokymia remained predominantly mild, suggesting that caffeine increased the frequency of eyelid twitching without producing clinically severe symptoms in most participants. These findings support the role of caffeine as a potential contributing trigger for benign eyelid myokymia, particularly in otherwise healthy adults, and highlight the importance of considering caffeine intake when evaluating recurrent eyelid twitching. Future controlled studies with placebo comparison, longer follow-up, and assessment of individual caffeine sensitivity may further clarify dose-response patterns and longer-term neuromuscular effects.

REFERENCES

1. Thompson RK, White P. Stress, sleep, and stimulants: The trifecta of benign eyelid fasciculation. Eye Brain. 2023;15:22-30.
2. Brown T, Smith J, Williams R. Effects of caffeine on motor control: A systematic review. Neurosci Biobehav Rev. 2022;134:1-12.
3. Lam BL, Patel BC. Eyelid myokymia: Clinical features and triggers. Ophthalmology. 2024;131(4):e45-e52.
4. Brown C, Wilson L. Historical vs modern perspectives on myokymia and lifestyle stimulants. Hist Ophthalmol. 2022;4(2):77-85.
5. Garcia M, Lopez A, Fernandez P. Caffeine intake and eyelid myokymia in young adults. Clin Ophthalmol. 2021;15:2101-2109.
6. Johnson T, Williams R, Brown L. Effects of caffeine on neuromuscular transmission: A systematic review. Neurosci Biobehav Rev. 2021;126:1-10.
7. Grgic J, Grgic I, Ekstedt M, Shala RW, Schoenfeld BJ. Effects of acute caffeine intake on muscular power during resistance exercise: A systematic review and meta-analysis. Sports Med. 2025;55(10):124-138.
8. White P, Henderson J, O'Connell K. Caffeine-induced changes in motor unit recruitment frequency: An EMG study. Muscle Nerve. 2023;67(4):289-296.
9. Lee JH, Kim SY, Park SH. Effects of caffeine on motor cortex excitability and blink reflex. J Clin Neurophysiol. 2021;38(3):180-188.
10. Anonaba CA, Anyanwu E, Isiguzo GC, Osuagwu UL. Effect of caffeine coffee on blink rates of adults during conversation. Acta Sci Ophthalmol. 2022;5(4):3-13.

11. Nguyen T, Patel S, Wilson L. Oral caffeine and its effect on ocular surface stability: A blink rate analysis. J Optom. 2024;17(1):15-22.

12. Garcia-Gomez R, Lopez-Mendoza A, Rodriguez-Soto J. Eyelid muscle activity and stimulant intake: A quantitative electromyography analysis. Clin Neurophysiol. 2024;135:88-96.

13. Lee HY, Park SY, Kim JH. Prevalence of eyelid myokymia in high-caffeine consuming university students: A cross-sectional study. Ophthalmic Epidemiol. 2022;29(5):450-458.

14. Zhao X, Wang Q, Liu F, Zhang Y. Influence of digital eye strain and caffeine on orbicularis oculi quivering. J Vis. 2025;25(3):10-18.

15. Wang Y, Liu X, Zhang H. Effects of caffeine on neuromuscular excitability and ocular motor responses. J Clin Neurophysiol. 2022;39(4):215-223.

16. Miller AM, Thompson RK, Garcia-Gomez R. The role of adenosine receptors in the regulation of spontaneous blink rate. Brain Res Bull. 2025;190:104-112.

17. Singh R, Kumar A, Sharma P. Lifestyle factors and eyelid myokymia in medical students. Indian J Ophthalmol. 2021;69(7):1850-1856.

18. Al-Mutairi S, Khan MA. Visual discomfort and eyelid quivering in computer users: The caffeine variable. Middle East Afr J Ophthalmol. 2022;29(1):12-18.

19. Kumar V, Sharma P, Singh R. Digital eye strain and caffeine-related eyelid myokymia. Indian J Ophthalmol. 2022;70(9):3105-3112.

20. Singh R, Kumar A, Sharma P. Digital eye strain, caffeine intake, and eyelid myokymia. Indian J Ophthalmol. 2023;71(8):3201-3208.