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Attentional network deficits in patients with migraine: behavioral and electrophysiological evidence

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Abstract

Background Patients with migraine often experience not only headache pain but also cognitive dysfunction, particularly in attention, which is frequently overlooked in both diagnosis and treatment. The influence of these attentional deficits on the pain-related clinical characteristics of migraine remains poorly understood, and clarifying this relationship could improve care strategies.

Methods This study included 52 patients with migraine and 34 healthy controls. We employed the Attentional Network Test for Interactions and Vigilance–Executive and Arousal Components paradigm, combined with electroencephalography, to assess attentional deficits in patients with migraine, with an emphasis on phasic alerting, orienting, executive control, executive vigilance, and arousal vigilance. An extreme gradient boosting binary classifier was trained on features showing group differences to distinguish patients with migraine from healthy controls. Moreover, an extreme gradient boosting regression model was developed to predict clinical characteristics of patients with migraine using their attentional deficit features.

Results For general performance, patients with migraine presented a larger inverse efficiency score, a higher prestimulus beta-band power spectral density and a lower gamma-band event-related synchronization at Cz electrode, and stronger high alpha-band activity at the primary visual cortex, compared to healthy controls. Although no behavior differences in three basic attentional networks were found, patients showed magnified N1 amplitude and prolonged latency of P2 for phasic alerting-trials as well as an increased orienting evoked-P1 amplitude. For vigilance function, improvements in the hit rate of executive vigilance-trials were exhibited in controls but not in patients. Besides, patients with migraine exhibited longer reaction time as well as larger variability in arousal vigilance-trials than controls. The binary classifier developed by such attentional deficit features achieved an F1 score of 0.762 and an accuracy of 0.779 in distinguishing patients with migraine from healthy controls. Crucially, the predicted value

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available from the regression model involving attentional deficit features significantly correlated with the real value for the frequency of headache.

Conclusions Patients with migraine demonstrated significant attentional deficits, which can be used to differentiate migraine patients from healthy populations and to predict clinical characteristics. These findings highlight the need to address cognitive dysfunction, particularly attentional deficits, in the clinical management of migraine.

Keywords Migraine, Attention, Electroencephalography (EEG), Vigilance, Hypersensitivity, Machine learning

Introduction

Migraine, a prevalent neurological disorder, affects approximately 14% of the global population [1]. Characterized by episodes of severe headaches often accompanied by nausea, migraines impose significant personal burdens, impacting not only the individual's quality of life but also contributing to a higher disability adjusted life year [2]. Emerging evidence suggests that patients with migraine exhibit cognitive impairments, particularly in attention, both during the acute migraine attack and in the interictal phase [3, 4]. Despite their significance, these cognitive deficits remained underrecognized and inadequately addressed.

The Attention Network theory [5, 6] proposed that attention consists of three independent but interacting attentional networks: alerting, orienting, and executive control. These networks are crucial for detecting, selecting, and organizing internal and external information, thereby enabling adaptive behavior. Among these, dysfunctions in the executive control are the most consistently reported in patients with migraine, as evidenced by impaired performance on tasks such as the Stroop Task [7], Stop Signal Task [8], Frontal Assessment Battery Test [9], and Wisconsin Card Sorting Test [10]. Nonetheless, these studies primarily focused on executive control, neglecting the other attentional functions and integrated nature of attentional networks. To address this issue, the Attentional Network Test (ANT [11]) has been employed to assess the efficiency of each attentional network in patients with migraine [12–14]. For instance, Bonsu et al. [14] found that the reaction time (RT) to targets of patients with migraine was more influenced by whether a preceding warning predicts the target onset when compared to healthy controls. Since phasic change in alertness could be produced by setting a warning signal that involves preparation for detecting and responding to the following expected-target event [5], this finding indicated impairments in the alerting network among patients with migraine.

However, comprehensive investigations that combine clinical assessments with detailed electroencephalography (EEG) analyses are scarce. An in-depth investigation of these networks in patients with migraine could shed light on the disorder's pathogenesis and inform the development of targeted interventions. The present study aimed to (1) systematically investigate the attentional dysfunctions in patients with migraine using both behavioral and EEG measures, and to (2) emphasize the significances of these dysfunctions in migraine diagnosis and prediction. To this end, the most advanced and detailed version of the ANT (i.e., the Attentional Networks Test for Interactions and Vigilance–Executive and Arousal Components, ANTI-Vea [15]) was employed in conjunction with EEG recording. This approach allows us to assess each attentional function in patients with migraine, specifically: (1) phasic alerting, induced by warning signal alerting individual to the following expected target; (2) orienting, induced by location cue facilitating target detection or causing reorientation; (3) executive control, resolving the conflicts between the targets and the distractors; (4) executive vigilance, maintaining the ability to detect critical signals that occur rarely by executing a specific response; and (5) arousal vigilance, maintaining a fast and stable reaction to stimuli occurring rarely without specific response [5, 15, 16]. Furthermore, the contributions of the possible deficits in these networks to the clinical characteristics of migraine were evaluated by developing a classification model to distinguish patients from healthy controls, as well as a regression model to predict clinical characteristics. This study seeks to provide a better understanding of the attentional dysfunctions in migraine. Such insights could pave the way for novel diagnostic and therapeutic approaches, thereby improving the quality of life for patients suffering from migraine.

Methods

Participants

Patients with migraine were recruited through the Neurology Outpatient Clinic of the Chinese People's Liberation Army (PLA) General Hospital. Two neurologists specializing in headache disorders managed the entire enrollment process. All patients with migraine met the following inclusion criteria: (1) a confirmed diagnosis of migraine without aura, as specified by the International Classification of Headache Disorders (the 3rd Edition); (2) being in the interictal phase, defined as the period between migraine attacks (>48 h after a migraine episode and >48 h before the next episode) [17]; and (3) a migraine history of at least one year. Exclusion criteria

included those who had taken preventive migraine medication within the three months prior to the study and individuals diagnosed with other types of headaches. Importantly, participants were not restricted to newly diagnosed or entirely medication-naïve individuals. Healthy participants, without chronic pain or a family history of migraines, were also recruited through advertisements posted on the hospital bulletin board. All participants enrolled in the study were native Chinese speakers and were required to be between 18 and 65 years old, right-handed, and have normal or corrected-to-normal vision and hearing. Participants with a diagnosis of brain injuries, psychiatric or neurodegenerative disease, or any chronic conditions requiring daily medication were excluded. Written informed consent was obtained from all participants prior to the experimental procedures. The study was approved by the Ethics Committee of the Chinese PLA General Hospital in accordance with the ethical principles of the Declaration of Helsinki (2023−460).

A total of 70 patients with migraine and 40 healthy participants were initially recruited. A larger sample size for patients due to the high dropout rate based on previous research [18, 19]. Follow-up assessments were conducted after the experiment to retrospectively identify participants in the interictal phase. Specifically, two patients and two healthy controls did not complete all tasks and withdrew from the experiment, and one patient was retrospectively identified as preictal and excluded from the analysis. Nine patients and two healthy participants were excluded due to poor task comprehension, while six patients and two healthy participants were excluded due to a poor signal-to-noise ratio of their EEG data. Consequently, 52 patients with migraine and 34 healthy participants were included in the final analyses. Patients (11 males) and healthy participants (11 males; $\chi^2_{(1)}=1.354$, $p=0.245$) were matched in terms of sex ratio.

Clinical questionnaires

The clinical characteristics of patients with migraine were documented (see Table 1). Patients with migraine completed the Allodynia Symptom Checklist (ASC), Headache Impact Test-6 (HIT-6), and Migraine Disability Assessment (MIDAS) to evaluate the severity of their headaches and the impact on daily life. Given that mood states can influence attention, all participants were required to complete the Patient Health Questionnaire-9 (PHQ-9), Generalized Anxiety Disorder-7 (GAD-7), and Perceived Stress Scale-14 (PSS-14) to assess levels of depression, anxiety, and stress. The Pittsburgh Sleep Quality Index (PSQI) was administered to compare sleep quality between patients with migraine and healthy controls.

ANTI-Vea task

A modified ANTI-Vea task [16] was employed in this study. The task consisted of two trial types: Type-1 trials, which assessed phasic alerting, orienting, executive control, and executive vigilance networks, and Type-2 trials, which assessed arousal vigilance network (see Fig. 1). In total, this task included 384 trials, divided into two blocks. All trials were presented in a pseudo-randomized order across participants.

Type-1 trials

Type-1 trials consisted of 320 trials and began with a white fixation cross on a black screen for 400–1600 ms. Subsequently, a 50 ms warning tone (838 Hz) was

Table 1 Demographics and clinical characteristics

	Patients		Healthy controls		*Z*	*p*
	Mean	*SD*	*Mean*	*SD*		
Age	29.88	7.96	29.62	6.34	0.115	0.908
Height (m)	1.67	0.07	1.68	0.09	1.033	0.301
Weight (kg)	61.14	10.50	62.71	9.09	0.894	0.372
Educational level	4.04	0.63	4.24	0.50	1.548	0.122
PSQI	6.62	4.07	7.09	3.70	0.950	0.342
GAD-7	5.69	5.05	4.50	2.98	-0.568	0.570
PHQ-9	6.90	5.46	5.00	2.93	-1.136	0.256
PSS-14	39.37	9.84	34.74	9.22	-1.866	0.062
Duration of migraine per month in the last 3 months (h)	22.38	20.24	-	-	-	-
History of migraine (year)	9.37	5.04	-	-	-	-
Frequency per month in the last 3 months	4.3	3.67	-	-	-	-
VAS for pain intensity	7	1.66	-	-	-	-
ASC	2.71	3.01	-	-	-	-
HIT-6	66.06	6.31	-	-	-	-
MIDAS	23.56	21.43	-	-	-	-

Notes SD, Standard Deviation; PSQI, Pittsburgh Sleep Quality Index; GAD-7, Generalized Anxiety Disorder-7; PHQ-9, Patient Health Questionnaire-9; PSS-14, Perceived Stress Scale-14; VAS, Visual Analogue Scale; ASC, Allodynia Symptom Checklist; HIT-6, Headache Impact Test-6; MIDAS, Migraine Disability Assessment

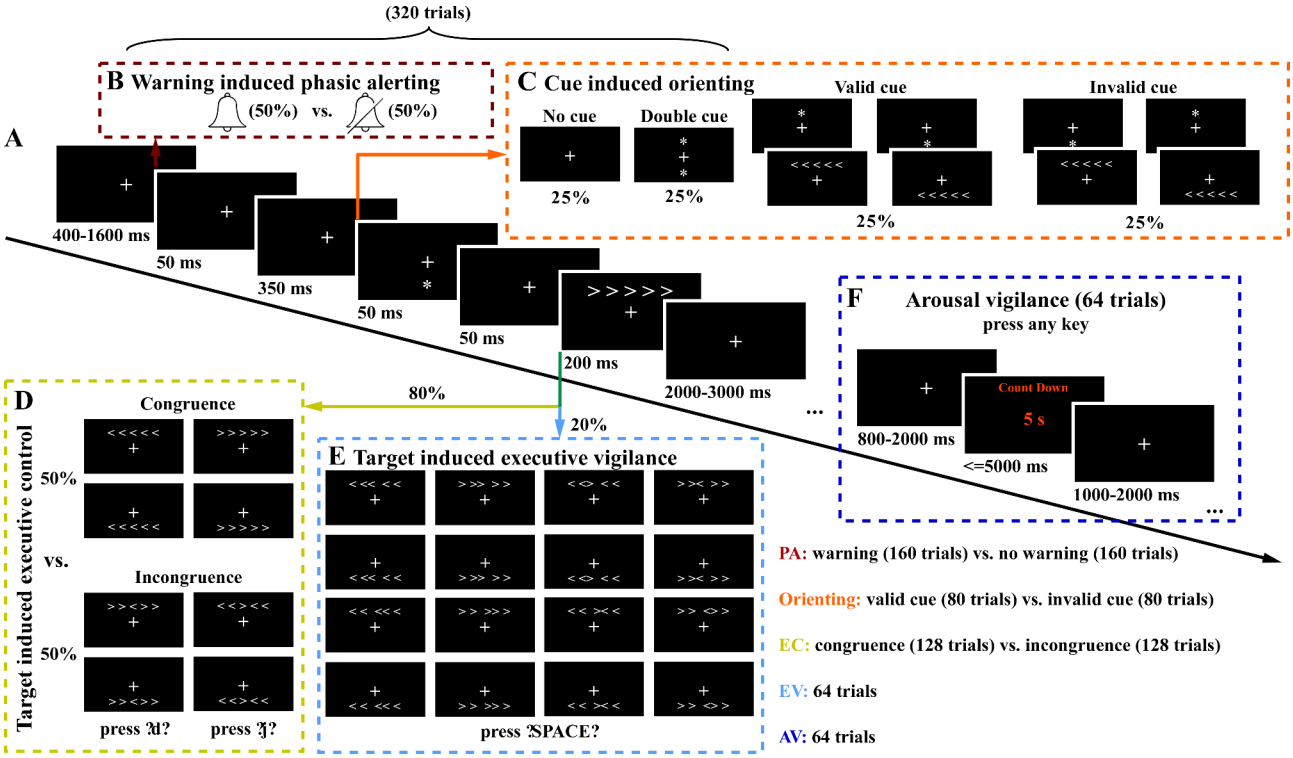

Fig. 1 The ANTI-Vea task. The task is comprised of two trial types, 320 trials for Type-1 and 64 trials for Type-2. (**A**) Type-1 trials, measuring phasic alerting, orienting, executive control, and executive vigilance networks by contrasting different combinations of warning stimuli, cue stimuli, and targets; (**B**) Warning stimulus condition, evaluating phasic alerting (trials with warning stimulus vs. without warning stimulus); (**C**) Cue stimulus condition, evaluating orienting (trials with valid cue vs. invalid cue); (**D**) Target stimulus condition account for 80%, respectively pressing 'd' or 'j' button for the left or right middle arrow, evaluating executive control (trials with congruent vs. incongruent target); (**E**) Target stimulus condition account for 20%, ignoring the arrow orientation and pressing 'SPACE' button, evaluating executive vigilance; (**F**) Type-2 trials, pressing any key as quickly as possible, measuring arousal vigilance. PA, phasic alerting; EC, executive control; EV, executive vigilance; AV, arousal vigilance

presented in half of the trials to induce phasic alerting, while the fixation cross remained visible. After 350 ms, a visual cue (no cue, double cue, upper cue, or lower cue) appeared for 50 ms, followed by a 50 ms fixation cross. The target stimulus, a row of five arrows, then appeared either above or below the fixation cross (50% each) for 200 ms. In 80% of the Type-1 trials, participants were instructed to determine the orientation of the middle arrow as quickly and accurately as possible by pressing either the 'd' key (left) or the 'j' key (right) with their index fingers while ignoring the flanking arrows. In the remaining 20% of the Type-1 trials, the middle arrow was presented off-center, and participants were asked to disregard its orientation but to promptly press the space bar. The inter-trial interval was 2000–3000 ms. All combinations of warning stimuli, cue stimuli, and target stimuli were presented in Fig. S1. Participants' RT and accuracy (ACC) were recorded.

Type-2 trials

Type-2 trials consisted of 64 trials and started with a white fixation cross in the center of a black screen, lasting for 800–2000 ms, followed by a 5-s countdown task. Participants were required to press any button as quickly as possible to stop the countdown, and the RT for each trial was recorded. The inter-trial interval was 1000–2000 ms.

Behavioral variables

General performance was evaluated for Type-1 trials using the inverse efficiency score (IES), calculated as RT/ACC, which measures overall energy consumption during the task [20]. Phasic alerting was assessed by comparing trials with and without warning tones, as well as by contrasting double-cue trials with no-cue trials. The orienting function was evaluated by comparing trials with valid cues, which correctly indicated the upcoming target location, to those with invalid cues. Executive control was estimated by contrasting trials where the middle arrow pointed in the opposite direction (i.e., incongruent) vs. the same direction (i.e., congruent) as the flanking arrows. RT and ACC were calculated to estimate the effectiveness of each attentional function (see Table 2).

Executive vigilance decrement was assessed by analyzing the hit rate (executive vigilance-hit) and correct rejection rate (executive vigilance-CR) over time: the first 15

Table 2 Features and main results for each attentional system

System	Behavior		EEG				
	Features	Main results	Components	Window size	Electrodes	Features	Main results
General [1] Averaged all Type-1 trials [2] Averaged all trials	**IES[1]**	**Patients > HCs***	**Prestimulus spectrum[2]**	**-1000 to 0 ms+**	Fz: δ & θ; **Cz**: α, **β** & γ; Pz: α, β & γ; Oz: α & γ	**PSD**	**Patients > HCs* in β-band at Cz**
			ERP[1]	**0 to 800 ms#**	**Cz**	**Averaged amp**	**Patients > HCs***
			γ-ERS[1]	**0 to 300 ms#**	**Cz**	**PSD**	**Patients < HCs***
			γ-ERD[1]	350 to 650 ms#	Cz	PSD	/
			α-band activity[1]	**0 to 2000 ms+**	**primary visual area**	**PSD**	**Patients < HCs***
Phasic alerting Trials with warning stimuli – trials without warning stimuli	RT	/	**N1**	**50 to 200 ms+**	**Cz**	**(1) Peak amp;** (2) Lat	**(1) Peak amp: Patients > HCs***; (2) Lat: /
	ACC	/	**P2**	**150 to 300 ms+**	**Cz**	(1) Peak amp; **(2) Lat**	(1) Peak amp: /; **(2) Lat**: **Patients > HCs****
			CNV	300 to 700 ms+	FCz	Averaged amp	/
Trials with double cue – trials without cue	RT	/	P1	50 to 100 ms#	primary visual area	(1) Peak amp; (2) Lat	(1) Peak amp: /; (2) Lat: /
	ACC	/	N1	100 to 150 ms#	primary visual area	(1) Peak amp; (2) Lat	(1) Peak amp: /; (2) Lat: /
Orienting Trials with valid cue – trials with invalid cue	RT	/	**P1**	**50 to 100 ms#**	**primary visual area**	**(1) Peak amp**; (2) Lat	**(1) Peak amp: Patients > HCs***; (2) Lat: /
	ACC	/	N1	100 to 150 ms#	primary visual area	(1) Peak amp; (2) Lat	(1) Peak amp: /; (2) Lat: /
			P3	350 to 650 ms#	Pz	Averaged amp	/
Executive control Trials with congruent target – trials with incongruent target	RT	/	**P1**	**50 to 100 ms#**	**primary visual area**	**(1) Peak amp;** (2) Lat	(1) Peak amp: Patients > HCs'; (2) Lat: /
	ACC	/	**N1**	**100 to 150 ms#**	**primary visual area**	(1) Peak amp; (2) Lat	(1) Peak amp: Patients > HCs'; (2) Lat: /
			N2	200 to 350 ms#	Fz	(1) Peak amp; (2) Lat	(1) Peak amp: /; (2) Lat: /
			SP	500 to 800 ms#	Pz	Averaged amp	/
Executive vigilance First 15 executive vigilance-trials (time 1) vs. last 15 executive vigilance-trials (time 2)	**Hit**	**Group × time***: **time 2 > time 1 in controls***'**; patients < controls'	**P1**	**50 to 100 ms#**	**primary visual area**	**(1) Peak amp;** (2) Lat	(1) Peak amp: Group×time'; (2) Lat: /
	CR	/	N1	100 to 150 ms#	primary visual area	(1) Peak amp; (2) Lat	/
			P3	350 to 650 ms#	Pz	Averaged amp	/
Arousal vigilance (1) First 15 arousal vigilance-trials (time 1) vs. last 15 arousal vigilance-trials (time 2); (2) Fastest 15 arousal vigilance-trials (high state) vs. lowest 15 arousal vigilance-trials (low state)	**RT**	**(1) Group**: **Patients > HCs***; **(2) Group**: **Patients > HCs***	P1	50 to 100 ms#	primary visual area	(1) Peak amp; (2) Lat	/
	IIRTV	**(1) Group**: **patients > HCs****; (2) Group: /	N1	100 to 150 ms#	primary visual area	(1) Peak amp; (2) Lat	/
			P3	**350 to 650 ms#**	**Pz**	**Averaged amp**	(1) Group×time: /; **(2) Group×states****: **high state > low state in patients****; patients > HCs under high state'

Notes *$p<0.05$; **$p<0.01$; ***$p<0.001$; '$p<0.1$. + time locked to warning signal onset; # time locked to target onset; /, no significant group difference. Features that showed significant group differences are highlighted in bold. Primary visual area refers to PO5, PO7, O1, PO6, PO8 and O2 electrodes. Abbreviations: GLM, generalized linear model; IES, inverse efficiency score; HCs, healthy controls; PSD, power spectral density; ERP, event-related potential; amp, amplitude; ERS, event-related synchronization; ERD, event-related desynchronization; RT, reaction time; Lat, latency; ACC, accuracy; CNV, contingent negative variation; SP, slow positivity; GEE, generalized estimate equation; CR, correct rejection rate; IIRTV, intra-individual reaction time variability

executive vigilance-trials (time 1) vs. the last 15 executive vigilance-trials (time 2) [16]. By contrast, arousal vigilance decrement was measured using the mean RT (arousal vigilance-RT) and intra-individual RT variability (arousal vigilance-IIRTV; calculated as standard deviation of RT divided by the mean RT [21]) across relevant trials over time: the first 15 arousal vigilance-trials vs. the last 15 arousal vigilance-trials, as well as under two arousal vigilance states: the fastest 15 arousal vigilance-trials (high arousal vigilance state) vs. the slowest 15 arousal vigilance-trials (low arousal vigilance state) [16, 22].

Data acquisition and analyses

After completing the clinical questionnaires, participants underwent EEG preparation and then completed the ANTI-Vea task with continuous EEG recording. Both the migraine and control groups were examined by the same examiner in the same room, with fully standardized instructions and procedures to ensure consistency in data collection. To minimize bias, group labels were blinded during data preprocessing and feature extraction, before conducting group analyses.

EEG recording and preprocessing

EEG data were recorded using 64 Ag-AgCl scalp electrodes placed according to the International 10–20 System (Compumedics Neuroscan; sampling rate: 1000 Hz, online reference: average). All electrode impedances were kept below 10kΩ.

EEG signals were preprocessed using the open-source toolbox EEGLAB [23], running in the MATLAB environment (MathWorks, USA). Continuous EEG data were filtered with a 0.1–60 Hz band-pass filter, and a 49–51 Hz notch filter was applied to remove 50 Hz powerline interference. EEG epochs were extracted using a time window of 3000 ms (1000 ms before and 2000 ms after the onset of the events of interest) and baseline corrected using the prestimulus interval. Specifically, the events of interest were the warning tones for Type-1 trials and the countdown challenges for Type-2 trials. All epochs were detrended to remove polynomial trends, and those contaminated with eyeblinks, movements, or other artifacts were corrected using an independent component analysis algorithm [23].

Frequency domain analyses

Prestimulus data (-1000 to 0 ms) were extracted and transformed to the frequency domain using the periodogram estimate at the single-trial level to obtain the power spectral density (PSD) for each frequency point within 1–60 Hz. Spectrograms at each electrode were averaged across all trials at the single-subject level and then averaged across subjects within each group. The spectrum was divided into the following bands: δ (1–3 Hz), θ (4–7 Hz), α (8–13 Hz), β (14–30 Hz), and γ (31–60 Hz) [24]. To investigate the prestimulus attentional processes and movement preparation [25–28], the PSD for each frequency band was calculated as the average within respective band and compared between the two groups in regions of interest, that is, δ- and θ-bands at the frontal cortex (Fz electrode); α-, β- and γ-bands at the sensorimotor cortex (Cz electrode) and parietal cortex (Pz electrode), and α- and γ-bands at the occipital cortex (Oz electrode).

Time domain analyses

Preprocessed EEG signals were further filtered with a 30 Hz low-pass filter and averaged across trials in the time domain, yielding event-related potential (ERP) waveform for each condition. In line with behavioral measurements, general ERP waveforms were obtained by averaging all Type-1 trials, and the 0-800 ms ERP was extracted from Cz where the significant inter-group differences were most pronounced. ERP waveforms associated with phasic alerting, orienting, and executive vigilance were generated by averaging Type-1 trials with and without warning tones, with valid and invalid cues, and with congruent and incongruent targets, respectively. In addition, ERP waveforms associated with executive vigilance were obtained by averaging the first and the last 15 executive vigilance-trials. ERP waveforms associated with arousal vigilance were obtained by averaging the first and the last 15 arousal vigilance-trials as well as the fastest and the slowest 15 arousal vigilance-trials. Referring to the previous study and the representative areas for specific components [16, 29–31], features extracted for each attentional system were presented in Table 2.

Time-frequency domain analyses

Time-frequency domain analyses were conducted on all Type-1 trials. Each Type-1 trial was transformed to the time-frequency domain using a short-term Fourier transform with a 400 ms window size to calculate the PSD for each time-frequency point at each electrode. The PSD for each time-frequency point was then baseline-corrected by subtracting the averaged PSD across baseline time window (-800 to -200 ms) at the corresponding frequency point. Event-related synchronization/desynchronization (ERS/ERD) related to motor preparation and execution (i.e., γ-ERS [500 to 800 ms, according to the first 300 ms after target appearance] and γ-ERD [850 to 1150 ms, according to 350ms-650 ms after target appearance] at Cz electrode [32]) and visual processing (i.e., α-band activity [0 to 2000 ms] at the primary visual area [33]) were compared between patients and controls.

Statistical analysis

Basic analysis

Considering the unequal sample size between the two groups and non-normal distribution of the data, all dependent variables were analyzed using non-parametric methods. Group differences in participant demographics and migraine characteristics were compared using Mann-Whitney *U* test (e.g., age) or Chi-squared (χ^2) test (e.g., sex ratio). Age and educational level were considered as covariates in the following analyses for behavioral and EEG data. A generalized linear model (GLM) was performed to estimate group differences, and a generalized estimate equation (GEE) was conducted to assess the main effects and interaction effect of two independent variables (e.g., group × time for executive vigilance). Pairwise comparisons were performed when there was a significant interaction. Coefficient (*B*) was calculated to reflect the effect size. $p<0.05$ were considered statistically significant. Bonferroni correction was applied for multiple comparisons when necessary.

Spearman rank correlations were estimated between features assessing corresponding attentional function, separately. Additionally, classification and regression models were established to test the potential diagnostic and monitoring ability of the dysfunctions in the attentional system revealed by the present study.

Classification modeling to distinguish patients from healthy controls

For binary classification, we developed a model based on the extreme gradient boosting (XGB [34, 35]) machines which performed effectiveness in small and imbalanced datasets. Grid search with leave-one-out cross-validation (LOOCV) was utilized to obtain optimal model parameters of XGB classifier developed. Specifically, in each iteration, one participant's data was reserved as the validation set, while the remaining participants' data constituted the training set. A classification model was developed to predict the outcomes for the validation data, and this process was repeated until every participant had been used as the validation data. This study identified the model parameters that yield the highest mean F1 score and mean classification accuracy, and the corresponding precision and recall values were reported.

Regression modeling to predict clinical characteristics of patients

XGB regression was performed to predict clinical characteristics (e.g., headache duration in the past three months). The individual prediction values for each clinical characteristic were calculated using LOOCV. To assess the performance of the regression model, Spearman rank correlation was calculated between the real and predicted values for each clinical characteristic.

Results

Demographics and clinical characteristics

There was no significant group difference in terms of sex ratio, age, educational level, height, or weight (all $p>0.05$). Migraine participants reported a higher trend in stress level compared to healthy controls, although the difference was not statistically significant ($p=0.062$). Demographics and clinical characteristics of participants are summarized in Table 1.

Differences in general response between patients and controls

Patients showed a significant larger IES than controls (patients: 932.71 ± 203.50 [hereinafter *Mean* ± *SD*]; controls: 839.15 ± 97.77; $\chi^2_{(3)}=5.443$, $p=0.020$, $B=78.743$; Fig. 2A). In line with this observation, GLM revealed that patients showing higher prestimulus β-band PSD (patients: -7.19 ± 2.35 dB; controls: -8.54 ± 2.53 dB; $\chi^2_{(3)}=6.389$, $p_{corrected}=0.033$, $B=1.352$; Fig. 2B-D) and larger averaged amplitude of target-locked 0-800 ms interval (patients: 0.08 ± 1.61 μV; controls: -0.58 ± 1.23 μV; $\chi^2_{(3)}=3.840$, $p=0.050$, $B=0.638$; Fig. 3A-C) than controls. In the time-frequency domain, group differences were signified in γ-ERS ($\chi^2_{(3)}=6.197$, $p_{corrected}=0.026$, $B=-0.267$; Fig. 3D-F) and high α-band activity (11–13 Hz; $\chi^2_{(3)}=5.022$, $p=0.025$, $B=-0.723$; Fig. 3G-I). Specifically, patients (0.14 ± 0.49 dB) showed smaller γ-ERS than controls (0.41 ± 0.51 dB) around the motor cortex during motor preparation. Additionally, patients (-1.28 ± 1.09 dB) exhibited larger high α-band activity than controls (-1.98 ± 1.89 dB) around the primary visual cortex during visual information processing. No other spectral activities showed significant differences between the two groups.

Differences in phasic alerting, orienting, and executive control between patients and controls

There were no significant group differences in either phasic alerting-RT or phasic alerting-ACC. However, ERP analyses indicated that warning tones (versus no warning tones) evoked a larger N1 amplitude ($\chi^2_{(3)}=3.756$, $p=0.050$, $B=-1.203$; see Fig. 4A-C) in patients with migraine (-6.13 ± 2.68 μV) compared to that in controls (-4.89 ± 3.00 μV). Moreover, warning tones evoked a delayed P2 latency ($\chi^2_{(3)}=8.143$, $p=0.004$, $B=10.262$; see Fig. 4A-B, D) in patients with migraine (231.83 ± 14.32 ms) compared to that in controls (222.68 ± 20.77 ms). For orienting, patients exhibited a higher orienting effect, as reflected by the difference amplitude (valid vs. invalid) of target evoked visual P1 (0.23 ± 1.35 μV) than controls (-0.27 ± 1.04 μV; $\chi^2_{(3)}=4.232$, $p=0.040$, $B=0.556$; see Fig. 4E-G). Although the differences (congruency vs. incongruency) in target-evoked P1 and N1 amplitudes showed a lower trend in patients (see Fig. S2; all $p<0.1$)

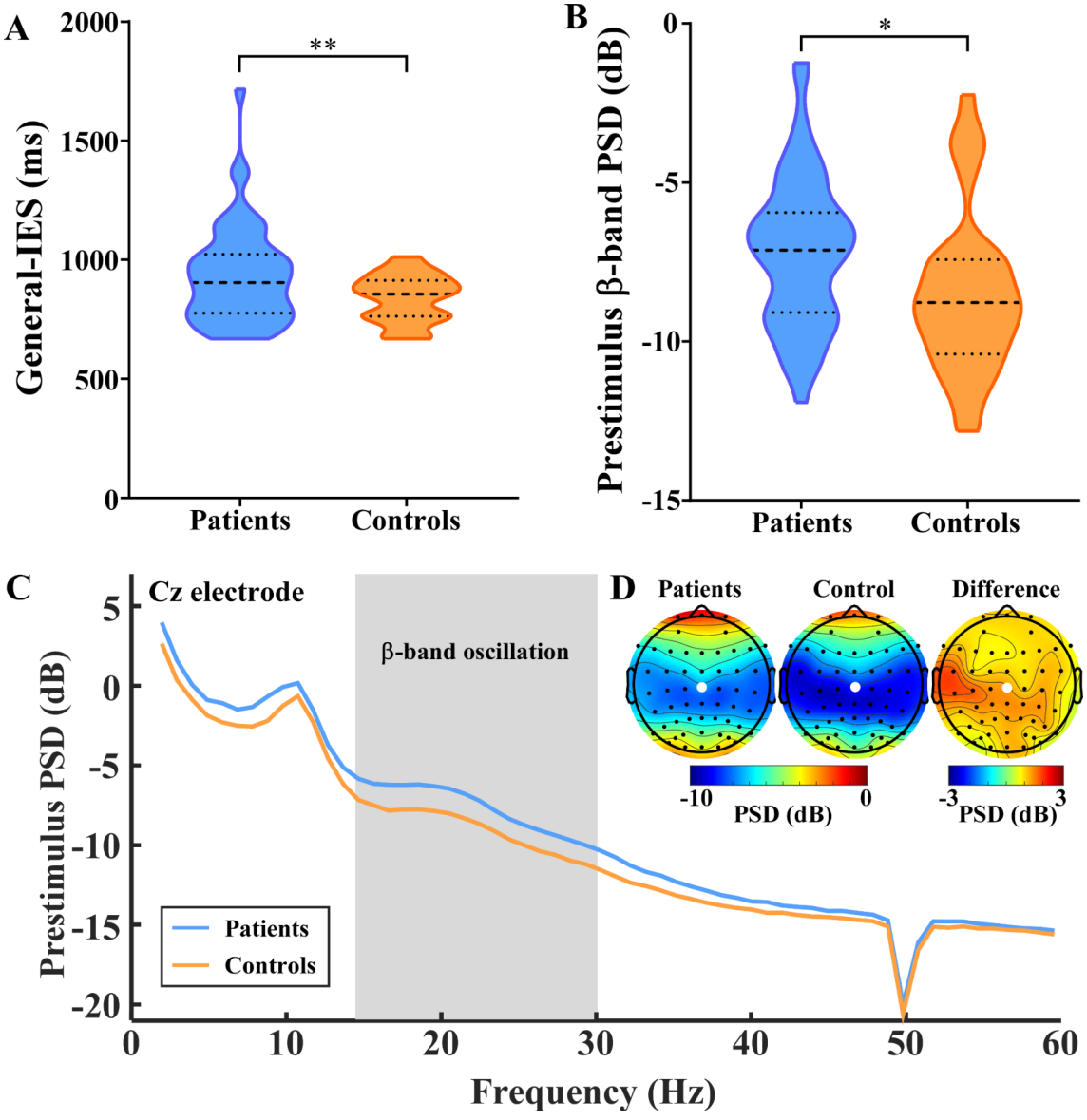

Fig. 2 Comparisons of prestimulus spectra and general behaviors between patients and healthy controls. (**A**) Comparison of general IES; (**B**) Comparison of prestimulus β-band PSD; (**C**) Baseline spectra at Cz electrode at group level, the shadow area highlighting the prestimulus β-band PSD; (**D**) Topographies of β-band PSD for patients, healthy controls, and their difference (patients − controls). $^{*}p<0.05$, $^{**}p<0.01$. IES, inverse efficiency score; PSD, power spectral density

than those in controls, no statistically significant difference was observed in executive control between the two groups. No other behavioral or EEG responses (see Table 2) for phasic alerting, orienting, and executive control systems presented significant group differences.

Differences in maintaining vigilance between patients and controls

GEE analyses demonstrated that patients with migraine were less vigilant than controls, as revealed by a significant group × time interaction of the hit rate in executive vigilance trials ($\chi^2_{(4)}=4.169$, $p=0.041$, $B=0.047$; Fig. 5A). Pairwise comparisons illustrated that the hit rate of the last 15 executive vigilance-trials was higher than that of the first 15 executive vigilance-trials in controls ($p_{corrected} < 0.001$), but not in patients ($p_{corrected} = 0.760$). Additionally, a marginally significant group × time interaction was observed in the P1 amplitude ($\chi^2_{(4)}=3.602$, $p=0.058$, $B=1.137$; Fig. 5B-C). This interaction was characterized by a smaller increase of P1 amplitude from the first 15 executive vigilance-trials (3.98 ± 2.59 μV) to the last 15 executive vigilance-trials (4.91 ± 2.58 μV) in patients ($p_{corrected} = 0.034$) compared to controls (the first 15 executive vigilance-trials: 2.79 ± 1.97 μV; the last 15 executive

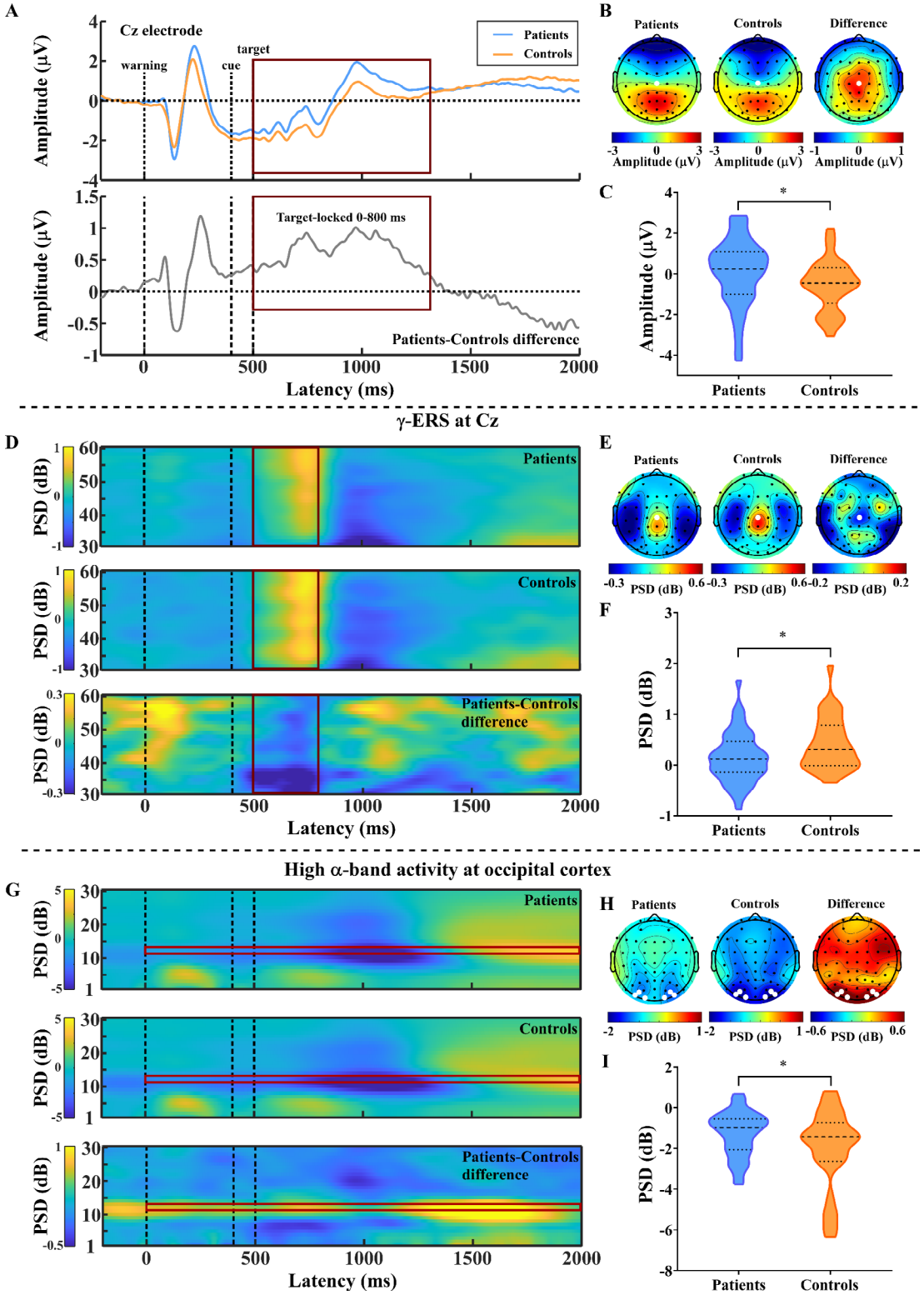

Fig. 3 Comparisons of general ERPs and time-frequency representations between patients and healthy controls. (**A**) General ERP waveforms at Cz electrode at group level and their difference; (**B**) Topographies of averaged amplitude across target-locked 0-800 ms; (**C**) Comparison of general amplitude at Cz; (**D**) γ-ERS at Cz electrode; (**E**) Topographies of γ-ERS; (**F**) Comparison of γ-ERS at Cz electrode; (**G**) High α-band activity at the primary visual cortex (i.e., PO5, PO7, O1, PO6, PO8 and O2 electrodes); (**H**) Topographies of high α-band activity; (**I**) Comparison of High α-band activity at the primary visual cortex. $^{*}p < 0.05$. PSD, power spectral density; ERS, event-related synchronization; ERPs, event-related potentials

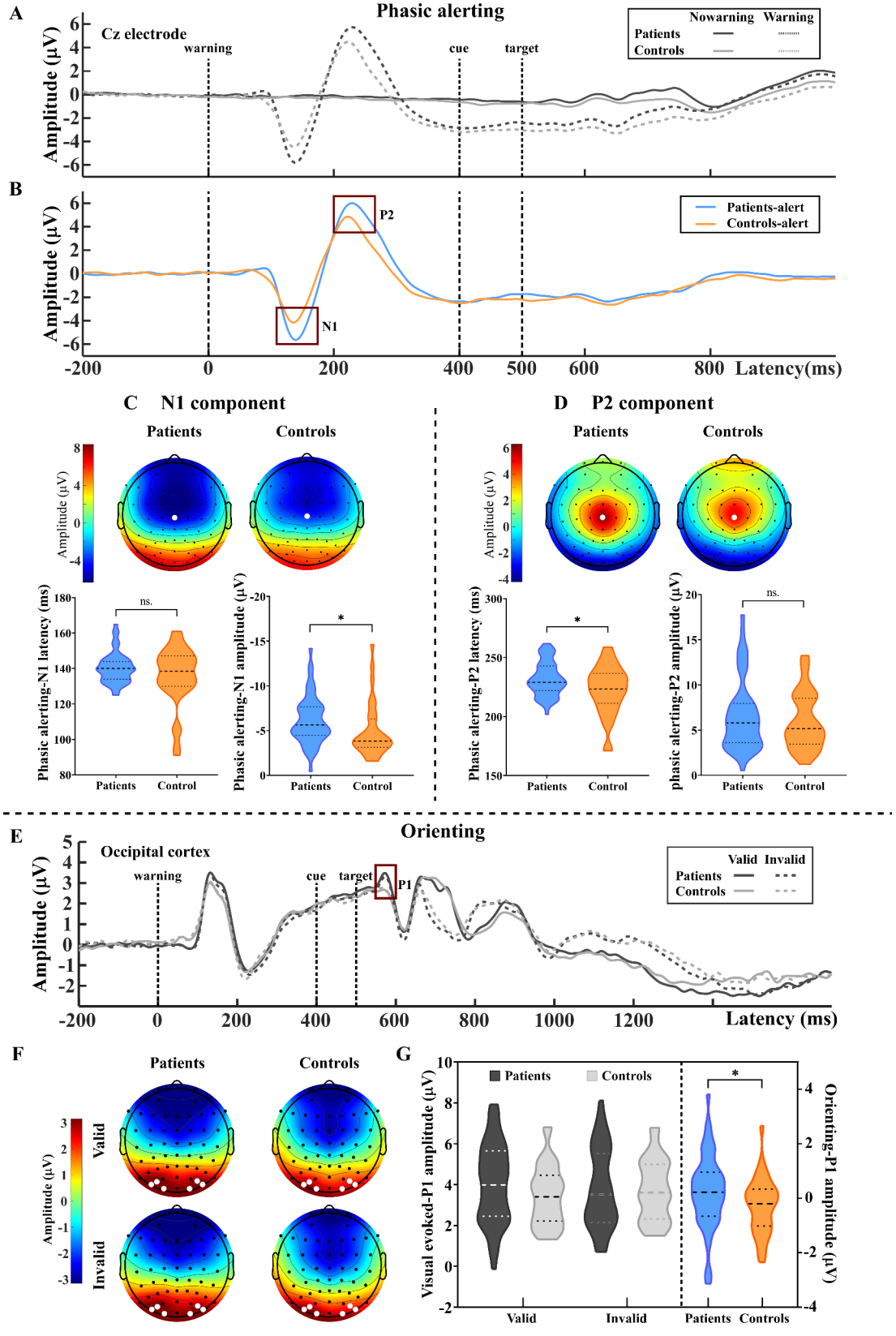

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Fig. 4 Comparisons of phasic alerting and orienting systems between patients and healthy controls. (**A**) ERP waveforms at Cz electrode for trials with and without warning stimuli; (**B**) ERP waveforms at Cz electrode presenting phasic alerting; (**C**) Topographies of peak amplitude of N1 and comparisons of latency of N1 and peak amplitude of N1 extracted from ERP waveforms presenting phasic alerting; (**D**) Topographies of peak amplitude of P2 and comparisons of latency of P2 and peak amplitude of P2 extracted from ERP waveforms presenting phasic alerting; (**E**) ERP waveforms at the primary visual cortex (i.e., PO5, PO7, O1, PO6, PO8 and O2 electrodes) for trials with valid cue and invalid cue; (**F**) ERP waveforms at the primary visual cortex for trials with valid cue and invalid cue; (**F**) Topographies of peak amplitude of P1 extracted from ERP waveforms for trials with valid cue and invalid cue; (**G**) Comparison of P1 difference amplitude (trials with valid cue – trials with invalid cue). ns., not significant; $^{*}p < 0.05$

vigilance-trials: 4.85 ± 2.25 μV; $p_{corrected} < 0.001$). Besides, the P1 amplitude in patients was higher than that in controls during the first 15 executive vigilance-trials ($p_{corrected} = 0.034$).

For arousal vigilance, patients showed slower RT ($\chi^2_{(4)}=6.095$, $p=0.014$, $B=67.891$; Fig. 5D) and a larger IIRTV ($\chi^2_{(4)}=9.061$, $p=0.003$, $B=0.090$; Fig. 5E) than controls across time, as revealed by significant main effects of group. Besides, GEE analysis confirmed longer RT in patients than in controls across arousal vigilance states ($\chi^2_{(4)}=4.513$, $p=0.034$, $B=155.945$; Fig. 5F). A group × state interaction was found on P3 amplitude ($\chi^2_{(4)}=10.718$, $p=0.001$, $B=1.722$; Fig. 5F), defined by a significant difference between attended-state (i.e., the fastest 15 arousal vigilance-trials; 5.13 ± 3.28 μV) and unattended-state (i.e., the slowest 15 arousal vigilance-trials; 4.13 ± 3.54 μV; $p_{corrected} = 0.006$) in patients, and a trend showing a larger P3 amplitude for patients (5.13 ± 3.28 μV) than for controls (4.04 ± 2.68 μV; $p_{corrected} = 0.082$) under high arousal vigilance state. No other features for vigilance illustrated significant abnormal in patients (see Table 2).

Classification model and regression model performance

To discriminate patients with migraine from healthy controls, all features exhibiting between-group differences were incorporated into the binary classifier and the regression model (see Fig. 6A). The XGB classifier (max depth: 3; learning rate: 0.1) achieved the best binary discrimination with an F1 score at 0.762, an ACC at 0.779, a precision at 0.774, and a recall at 0.756. The importance ranking for features is presented in Fig. 6B. The first 10 features, in order of their importance, are: IIRTV for the first 15 arousal vigilance-trials (10.52%), PSS-14 score (10.02%), IIRTV for the last 15 arousal vigilance-trials (7.94%), N1 difference amplitude reflecting alerting (7.19%), general γ-ERS (5.93%), IES (5.80%), RT for the last 15 arousal vigilance-trials (5.73%), prestimulus β-band PSD (5.42%) and P2 difference latency associated with alerting (4.92%).

For regression model, attention deficits found in the present study could predict the headache frequency per month in the last three months, indicated by the significant correlation between the predicted and the real values for headache frequency ($\rho=0.314$, $p_{\text{corrected}} = 0.024$; Fig. 6C). The average gain representing reduction in loss across all splits and trees for each feature in the regression model to predict headache frequency was presented in the Fig. 6D.

Discussion

Based on the ANTI-Vea task, we found that patients with migraine exhibit hypersensitivity to external stimuli, general deficits in attentional networks, and impaired vigilance system at both behavioral and electrophysiological levels. Crucially, these impairments could distinguish patients with migraine from healthy individuals and predict headache frequency in those with migraine. The specific features assessed in the present study may enhance the understanding of secondary diagnosis indicators for migraine.

Hypersensitivity to external stimuli is a frequently reported symptom in patients with migraine [36, 37]. Our study corroborated this finding, revealing sensory overload across auditory and visual modalities. Specifically, while there were no significant corresponding behavioral features, the neural hypersensitivities, particularly the N1 response to auditory stimuli, played a crucial role in differentiating patients with migraine from healthy controls (see Fig. 6B). Notably, ERP amplitudes are highly variable and not consistently increased in all migraine studies, depending on stimulus types, specific ERP components, and task conditions [19, 38]. Thus, although the hypersensitivity observed in our study showed potential as an indicator for diagnosing and predicting migraine characteristics, the specificity and generalizability of these findings need further validation before they can be applied clinically [39].

Except for extensively studied finding of hypersensitivity in migraine patients, the present study highlighted an integrate impairment in attention networks revealed by enhanced IES scores. Similar findings were reported by Bonsu et al. [14] and Chen et al. [13], who demonstrated prolonged overall RTs in patients with migraine using the ANT task. These findings provide consistent evidence that patients with migraine experience concurrent attention deficits. Supporting this, prestimulus spectral analyses showed increased PSDs across frequency bands in patients with migraine, with a significant increase in the β-band at Cz (Fig. 2B-D), which was considered to manifest the activity of the parietal attentional network [40]. In addition, movement-related γ-ERS, which was diminished in patients with migraine (Fig. 3D-F), supports the idea that patients with migraine may have a reduced

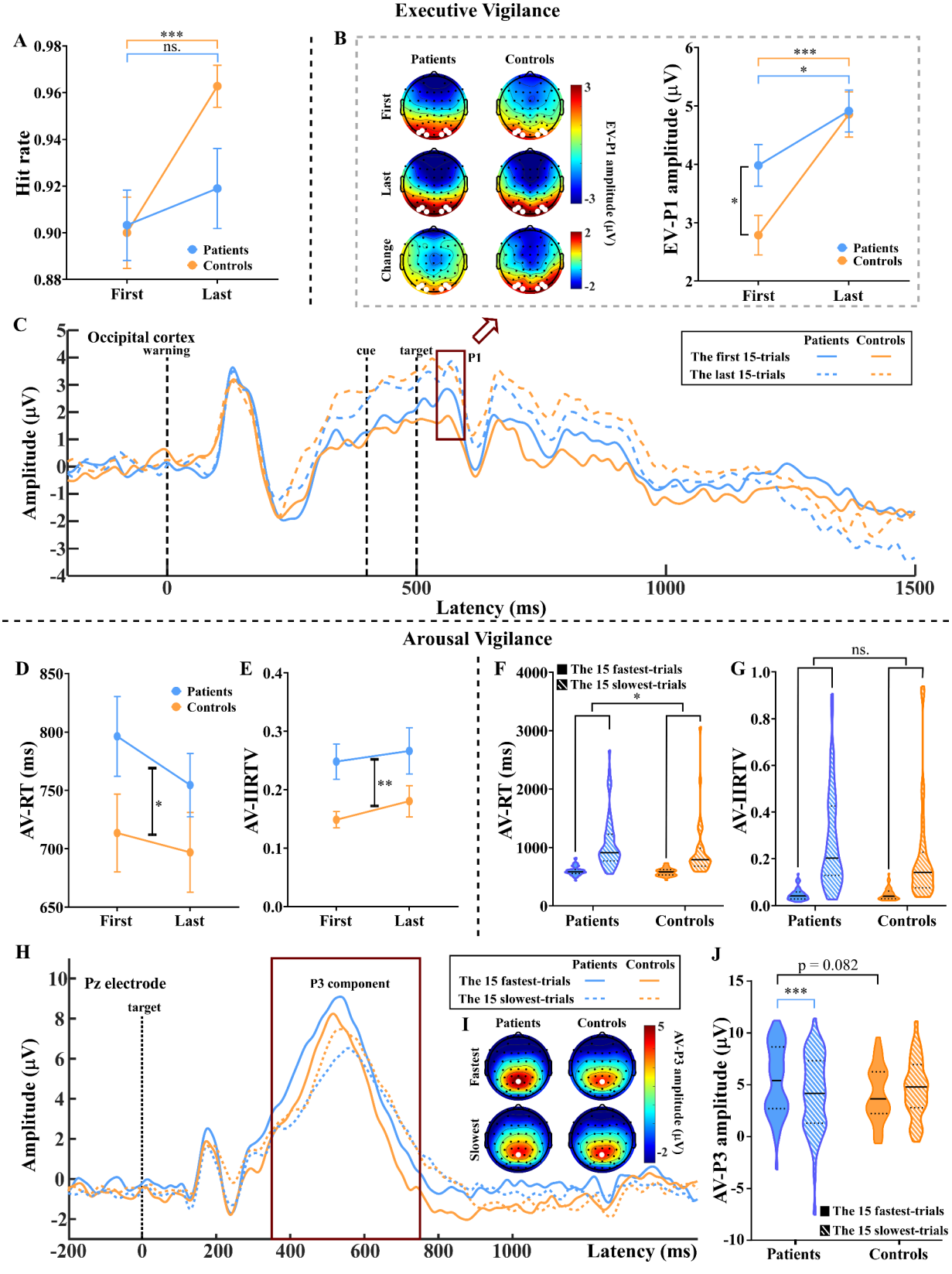

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Fig. 5 Comparisons of executive vigilance and arousal vigilance systems between patients and healthy controls. (**A**) Hit rate for the first 15-executive vigilance trials (time 1) and the last 15-executive vigilance trials (time 2) with a significant increase in controls but not in patients across times; (**B**) Left panel, topographies of peak amplitude of P1 at time 1 and time 2, as well as their differences for executive vigilance trials; Right panel, group × time interaction effect on the peak amplitude of P1 for executive vigilance trials, revealing a larger increase in P1 peak amplitude for healthy controls than that for patients across times and a larger P1 peak amplitude for patients than that for healthy controls at time 1; (**C**) ERP waveforms at the primary visual cortex (i.e., PO5, PO7, O1, PO6, PO8 and O2 electrodes) at time 1 and time 2 for executive vigilance trials; (**D**) Group difference in RT for arousal vigilance trials across times; (**E**) Group difference in IIRTV for arousal vigilance trials across times; (**F**) Group difference in RT for arousal vigilance trials across attended (fastest 15-arousal vigilance trials) and unattended (slowest 15-arousal vigilance trials) states; (**G**) No group difference in IIRTV for arousal vigilance trials across states; (**H**) ERP waveforms at Pz electrode under two states for both groups; (**I**) Topographies of averaged amplitude of P3 under two states for both groups; (**J**) Group × state interaction effect on averaged amplitude of P3 for arousal vigilance trials, revealing a significant state difference in patients and a higher amplitude trend in patients under attended-state. ns., not significant; $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$. EV, executive vigilance; AV, arousal vigilance; RT, reaction time; IIRTV, intra-individual reaction time variability

capacity to allocate cognitive resources for movement planning and execution [32, 41].

Enhanced α-band activity at the visual cortex (Fig. 3G-I) might be another neural basis for the impairment in general attention. Alpha-band activity is considered to suppress cortical excitability by attenuating neural spike timing and firing rate [27, 42–44]. Thus, the continuously increased α-band activity in patients with migraine may result in facilitated cortical inhibition in the visual cortex during target detection and processing. Abnormal resting-state α-band activity has been widely investigated in migraine [45–47]. Besides, α-band activity represents top-down regulation to the visual cortex during tasks. Lisicki et al. [33] recorded feedback volleys from higher-order visual areas (i.e., V2 to V4) to the primary visual cortex, inducing a larger α-band power in patients with migraine in interictal phase than that in healthy controls. This phenomenon may reflect an increased top-down inhibition to against sensory overload as a protective (or compensatory) mechanism [33].

The vigilance system was another attentional system found to be impaired in patients with migraine. Vigilance refers to the ability of maintaining the rapid detection of infrequent stimuli from the external environment, which is a key distinction from the concept of alerting [15, 48]. Although we cannot completely rule out the influence of the learning network, presented by increased hit rate in healthy participants but not in patients, further ERP analysis supports the existence of impairment in the maintenance of vigilance in patients with migraine. Specifically, P1 amplitude for executive vigilance-trials was increased across times both in patients and healthy controls, while the significant group difference in P1 amplitude observed in time 1 did not show in time 2 (Fig. 5B). Healthy participants were able to allocate more attentional resources to maintain a high level of performance in the later time of the experiment, whereas patients with migraine were unable to sustain the same level of resource allocation, despite demonstrating hypersensitive to external stimuli in the early time of the experiment. This suggested that the ability of patients with migraine to sustain executive vigilance over time may be impaired.

Similarly, the present study also identified disabilities in maintaining arousal vigilance, revealed by P3 component. Parietal P3 is an indicator of attentional resource allocation to target [49, 50] and has been shown to be reduced during mind wandering relative to on-task periods in patients with migraine [51]. Previous studies have reported mixed results with enlarged [51, 52], reduced [53, 54], and nonabnormal [53] P3 amplitude in patients with migraine, compared to healthy controls, which were affected by task types and stages of migraine attack. Our results exhibited a higher trend in P3 amplitude in patients with migraine, which may illustrate a higher demand of attentional resources to maintain attended state, compared to healthy controls.

While differences in behavioral and evoked EEG responses have been widely investigated in migraine research, previous studies often relied on univariate analyses, lacking an integration and comprehension of complex and high-dimensional features. Machine learning offers an opportunity to handle such datasets, capture non-linear interactions between features, and distinguish patients from healthy controls, which is particularly meaningful for clinical use. A few studies have developed classification models often using somatosensory evoked responses [55, 56]. These studies showed good model performance, as somatosensory and pain processing share similar networks. In contrast, cognitive task evoked responses have received less attention in the literature. Using an XGB classifier, our binary classification model developed with attentional related behavioral indicators and EEG signals, highlighting the potential of using attentional features as biomarkers for migraine diagnosis.

On the other hand, several studies have utilized correlation analysis to explore the relationship between attentional deficits and clinical characteristics in migraine [12, 57] but few research has quantified the influence of these attentional deficits. In the present study, attentional deficits were able to predict the headache frequency using XGB regression modeling. However, we acknowledge that there are noticeable discrepancies between the predicted and actual values, even though the correlation between them was statistically significant (Fig. 6C). The

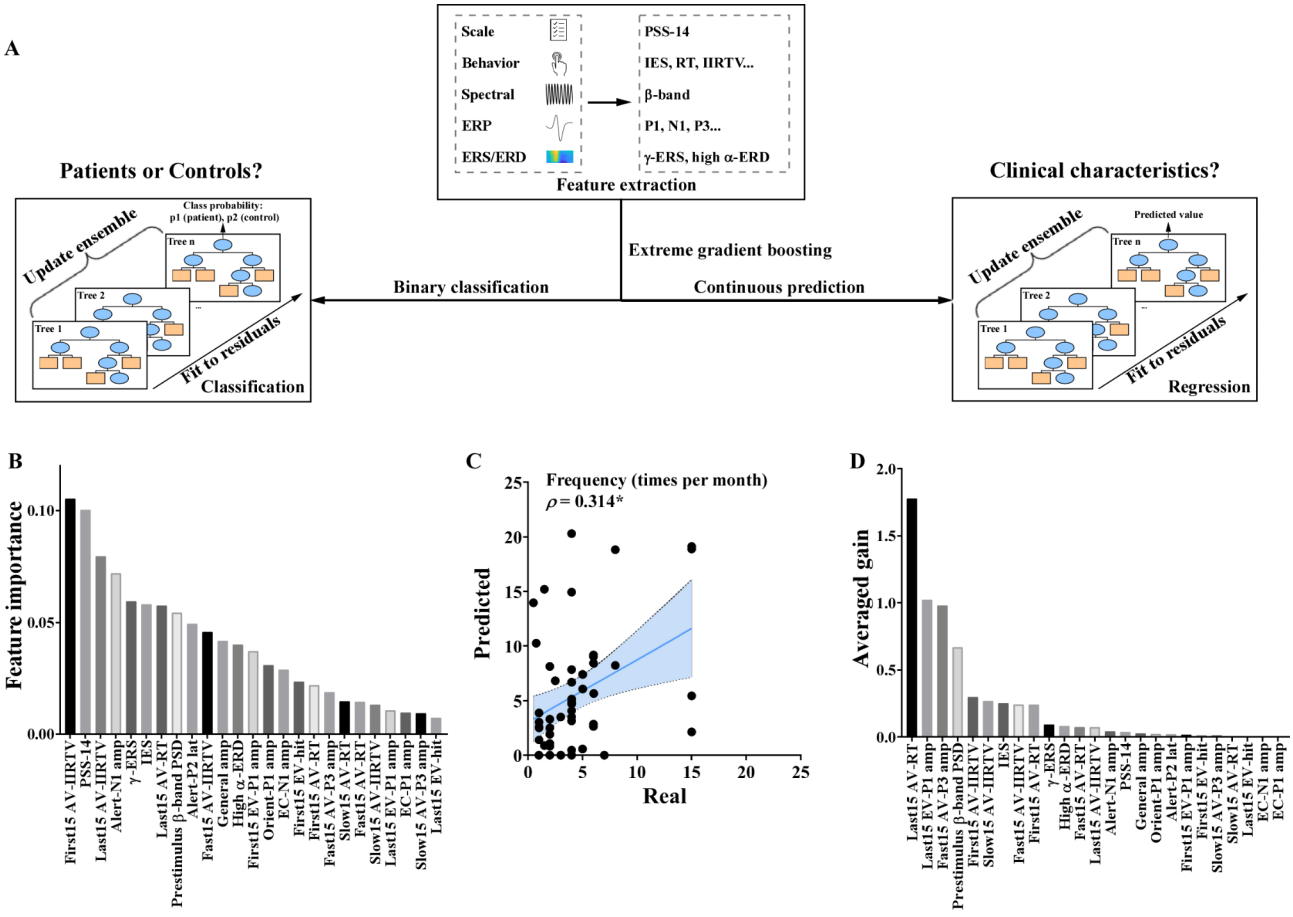

Fig. 6 Classification and regression models. (**A**) Feature selection for developing gradient boosting (XGB) classification model to distinguish patients from healthy controls and regression model to predict clinical characteristics of patients; (**B**) Importance ranking for each feature in the XGB classification model; (**C**) Correlation between real headache frequency and predicted headache frequency estimated by the XGB regression model using leave-one-out cross-validation; (**D**) Average reduction in loss for each feature across all splits and trees in regression model for predicting headache frequency. $^{*}p < 0.05$. PSS-14, Perceived Stress Scale-14; IES, inverse efficiency score; RT, reaction time; IIRTV, intra-individual reaction time variability; ERS, event-related synchronization; AV, arousal vigilance; amp, amplitude; PSD, power spectral density; lat, latency; EV, executive vigilance; EC, executive control

small size with large inter-individual variability may have been one of the impediments to the regression model training. Thus, future studies should increase the sample size to further investigate the causality instead of correlations between the attentional deficits and clinical characteristics during migraine development. Nonetheless, the regression model in our study captured meaningful relationships, which is valuable for understanding general trends and identifying important features. In this model, arousal vigilance features played crucial roles in the regression model (Fig. 6D), indicating the importance of long-term monitoring of vigilance function in migraine sufferers.

Although our study illustrated the importance of attentional deficits in patients with migraine, an area rarely explored before, there were several limitations. Study recruitment was limited to patients with migraine without aura who were during the interictal phase, which restricts the generalizability of our findings to other attack periods. Also, attentional dysfunctions for different migraine subtypes (e.g., episodic and chronic) need to be contrasted in future studies to enhance precision diagnosis and precision medicine. Nonetheless, reanalysis showed consistent results after excluding patients with a frequency of 15 attacks per month (i.e., chronic migraine; see Table S1). Besides, while blinding was implemented during data preprocessing and feature extraction, the lack of blinding during data acquisition may introduce some bias. However, given that both patients and controls followed standardized instructions and procedures, we believe that the absence of blinding for the examiner is unlikely to significantly influence the results. In addition, although we employed LOOCV, which is well-suited for small datasets by maximizing the use of available data while still providing a robust validation framework [58, 59], the generalizability of the models remains limited without external independent validation [39]. Multi-center data collection will help validate the generalizability

of the findings across diverse populations. Furthermore, the specificity of these attentional deficits to migraine remains in question, which hinders the recognition of the related features as migraine diagnostic or predicting biomarkers [39]. Including patients with other types of chronic pain, particularly various headache types, will help verify whether the identified attentional deficits are selective to migraine.

Conclusion

Our study demonstrated significant attentional deficits in patients with migraine, primarily characterized by hypersensitivity to external stimuli, general attentional network impairments, and decrements in vigilance function. These deficits were effective in distinguishing patients with migraine from healthy controls and in predicting headache frequency. Increasing attention on the association between migraines and attentional deficits may help develop more comprehensive treatment therapies that focus not only on pain management but also on improving the overall quality of life for patients.

Abbreviations

ANT	Attentional Network Test
RT	Reaction Time
EEG	Electroencephalography
ANTI-Vea	Attentional Networks Test for Interactions and Vigilance–Executive and Arousal Components
PLA	People's Liberation Army
ASC	Allodynia Symptom Checklist
HIT-6	Headache Impact Test-6
MIDAS	Migraine Disability Assessment
PHQ-9	Patient Health Questionnaire-9
GAD-7	Generalized Anxiety Disorder-7
PSS-14	Perceived Stress Scale-14
PSQI	Ptiisburgh Sleep Quality Index
ACC	Accuracy
IES	Inverse Efficiency Score
IIRTV	Intra-Individual Reaction Time Variability
PSD	Power Spectral Density
ERP	Event-Related Potential
CNV	Contingent Negative Variation
ERS	Event-Related Synchronization
ERD	Event-Related Desynchronization
GLM	Generalized Linear Model
GEE	Generalized Estimate Equation
XGB	Extreme Gradient Boosting
LOOCV	Leave-One-Out Cross-Validation
SD	Standard Deviation

Supplementary Information

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Supplementary Material 1

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Author contributions

YXC: study conception and design, data analysis, data interpretation, writing the manuscript, review and editing. SYX: study conception and design, data acquisition, writing the manuscript, review and editing. LBZ: data analysis, review and editing. DSL, HS, RFW, RA, XXL, YYL, SHZ, DQZ, YS and SQW: data acquisition. LH: review and editing, funding acquisition, supervision. ZD: review and editing, funding acquisition, project administration, supervision. XJL: review and editing, funding acquisition, supervision. All authors read, reviewed and approved the final version of the manuscript.

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of the Chinese PLA General Hospital in accordance with the ethical principles of the Declaration of Helsinki (2023 – 460).

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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