Age-Related Topological Organization of Phase-Amplitude Coupling Between Postural Fluctuations and Scalp EEG During Unsteady Stance

Yi-Ching Chen, Yi-Ying Tsai, Wei-Min Huang, Chen-Guang Zhao, and Ing-Shiou Hwang

***Abstract*—Through phase-amplitude analysis, this study investigated how low-frequency postural fluctuations interact with high-frequency scalp electroencephalography (EEG) amplitudes, shedding light on age-related mechanic differences in balance control during uneven surface navigation. Twenty young (24.1 ± 1.9 years) and twenty older adults (66.2 ± 2.7 years) stood on a training stabilometer with visual guidance, while their scalp EEG and stabilometer plate movements were monitored. In addition to analyzing the dynamics of the postural fluctuation phase, phase-amplitude coupling (PAC) for postural fluctuations below 2 Hz and within EEG sub-bands (theta: 4-7 Hz, alpha: 8-12 Hz, beta: 13-35 Hz) was calculated. The results indicated that older adults exhibited significantly larger postural fluctuation amplitudes(p <0.001) and lower mean frequencies of the postural fluctuation phase (p=0.005) than young adults. The PAC between postural fluctuation and theta EEG (FCz and bilateral temporal-parietal-occipital area), as well as that between postural fluctuation and alpha EEG oscillation, was lower in older adults than in young adults (p <0.05). In contrast, the PAC between the phase of postural fluctuation and beta EEG oscillation, particularly in C3 (p = 0.006), was higher in older adults than in young adults. In summary, the postural fluctuation phase and phase-amplitude coupling between postural fluctuation and EEG are sensitive indicators of the age-related decline in postural adjustments, reflecting less flexible motor state transitions and adaptive changes in error monitoring and visuospatial attention.**

***Index Terms*—Balance, cross-frequency modulation, elderly, electroencephalography, visual feedback.**

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I. Introduction

AGE-RELATED declines in posture stability elevate the risk of accidental falls and injury-related mortality among older adults. Improving the postural balance of older adults through training on uneven surfaces, like a stabilometer, is crucial. This imposes an additional cognitive load to monitor sensory feedback and direct motor responses during postural training [1], [2], as postural training necessitates heightened attentional resources and cognitive flexibility to adeptly coordinate limb movements in response to balance constraints [3], [4]. For degenerative changes in executive function, visuospatial cognition, and sensorimotor processing [5], [6], older adults often demonstrate increased postural sway and a reduction in the complexity of their postural responses compared to younger adults [7], [8], [9]. Although age-related changes in postural responses have been widely reported [10], [11], [12], the phase aspects of postural response variations in older adults have long been overlooked. The phase components of postural response, obtained through the Hilbert transform [13], signify the timing relationship between different spectral components of postural adjustments relative to a perturbation event in time. Phase information, being sensitive to subtle yet rapid changes in responses, may well characterize the dynamic motor transitions within the postural control system.

EEG's high temporal resolution allows researchers to capture rapid changes in brain activity essential for maintaining postural balance. This includes intricate band-specific and localized processes for motor planning, execution, and feedback processing, which are crucial for adapting to and meeting balance constraints. EEG studies have pinpointed specific cortical regions, particularly sensorimotor areas, that show heightened activation during balance maintenance and in response to postural threats [14], [15]. Monitoring changes in

EEG oscillatory activity in the theta (4-7 Hz), alpha (8-12 Hz), and beta bands (13-35 Hz) can be useful to explore the neural mechanisms underlying cognitive, sensorimotor, and integrative processes pertinent to postural regulation. Increases in theta power in the frontal area may reflect the facilitation of attentional resource allocation with visual cues for postural adjustments [16], [17]. The alpha power over visual areas reflects the processing of visual feedback related to body and environmental contexts [4], [18]. Beta synchronization in the sensorimotor cortex may coincide with postural recovery, or the transition from active postural adjustment to static posture maintenance [19], [20]. However, to date, the direct communication between high-frequency brain oscillations and low-frequency postural behaviors has not been comprehensively characterized. This is despite evidence suggesting that cortical potentials in the beta band can be time-locked to peaks in postural fluctuation trajectories during standing balance [15], [21].

Phase-amplitude analysis between the phase component of postural fluctuations and band-specific oscillations is a promising approach to bridge the methodological gap in understanding brain-behavior coupling during postural regulation. Phase-amplitude analysis delves into the intricate interaction between the amplitude of one frequency component and the phase of another. Strong coupling between phase and amplitude indicates a coordinated relationship between oscillatory patterns [22]. The traditional application of phase-amplitude analysis in neuroscience is to characterize the coordinated cortical activity across different spectral bands among distributed cortical areas [23], [24], [25]. This study introduces a novel extension of phase-amplitude analysis, aiming to investigate brain-behavioral coupling between postural fluctuations and band-specific scalp EEG in young and older adults, as they could differently respond to postural training with support-surface perturbations during upright stance. The topological distribution of phase-amplitude coupling (PAC) between postural fluctuation phase and EEG activity is hypothesized to be age-dependent, reflecting distinct neurocognitive and sensorimotor processes involved in managing postural perturbations. During stabilometer stance, it is hypothesized that: 1) PAC in the theta band is lower for older adults due to diminished attentional resource allocation for postural control; 2) PAC in the alpha band is lower for older adults who rely more on visual feedback for stance regulation; 3) PAC in the beta band is lower for older adults who are less adept at adjusting to unstable postural responses.

II. Methods

A. Ethical Approval

The study's data partly included findings from a prior investigation conducted by Chen et al. [16], [24], which explored the influence of varying visual feedback on regional activity and inter-regional connectivity during stabilometer stance. The study was approved by an authorized institutional human research review board at the National Chung Cheng University Hospital (No. B-ER-105-032). Prior to the experiment, all subjects read and signed personal consent forms, in accordance with the Declaration of Helsinki. However, the current analysis focuses on novel aspects, including phase-amplitude analysis and the dynamics of the postural fluctuation phase, which have not been previously documented. This study provides comprehensive methodological details for these analyses.

B. Participants

Twenty healthy young adults (9 males, 11 females, age: 24.1 $\pm$ 1.9 years) and twenty older adults (8 males, 12 females, 66.2 $\pm$ 2.7 years) from local community participated in this study. The young subjects were free of neurological and/or musculoskeletal disorders. The older adults were recreationally active without known neurological, cognitive, degenerative conditions, or severe cardiovascular conditions that could affect their balance. The subjects self-reported their right limb as the dominant limb. All experimental procedures complied with the Declaration of Helsinki and approved by the Clinical Research Ethics Board (No. B-ER-105-032) of a university hospital. Participants provided written informed consent.

C. Experimental Procedure and Measurements

Participants in both groups were instructed to maintain an upright posture as steadily as possible on the stabilometer, which measured 50 cm $\times$ 58 cm for the area, with a 25 cm radius and a height of 18.5 cm, for a duration of 60 seconds. Each participant performed three trials of stabilometer stance, with a resting interval of 3 minutes between trials. The postural task was conducted with visual guidance, including an online trajectory display of the stabilometer plate's movements and a target line representing ground level on a computer monitor (Fig. 1). Participants primarily controlled movements of their ankle joints to compensate for fluctuating movements of the stabilometer surface. Before the experiments, participants completed 1-2 practice trials to familiarize themselves with the task before experimental data collection. During stabilometer stance, the fluctuating movement of the stabilometer surface was recorded with an inclinometer (Model FAS-A, LORD MicroStrain, USA) mounted by the side of the stabilometer. Synchronized with the inclinometer, cortical activities during stabilometer stance were measured with a NuAmps amplifier (NeuroScan Inc., EI Paso, USA) and Ag-AgCl scalp electrodes in accordance with the International 10-20 system. Scalp EEG signals were localized at various cortical areas (Fp1/2, Fz, F3/4, F7/8, FT7/8, FCz, FC3/4, Cz, C3/4, CPz, CP3/4, Pz, P3/4, T3/4, T5/6, TP7/8, Oz, and O1/2). Reference electrodes were placed on each side of the mastoid process (A1/A2), and the ground electrode was placed on the forehead. To mitigate eye movement and blink artifacts, horizontal electrooculography (EOG) data were collected with electrodes at the outer canthus of the left and right eyes. For offline vertical EOG assessment, two electrodes were placed infra- and supra-orbitally at the right eye. Electrode impedances were maintained below 5 kΩ. The EEG data were recorded with a band-pass filter (cut-off frequencies: 0.1-70 Hz) and a 60 Hz notch filter. Signal synchronization was accomplished with the AD controller under the LabView platform (Labview v.8.5, National Instruments, USA). Sampling rate of the EEG and stabilometer plate movement were 1 kHz.

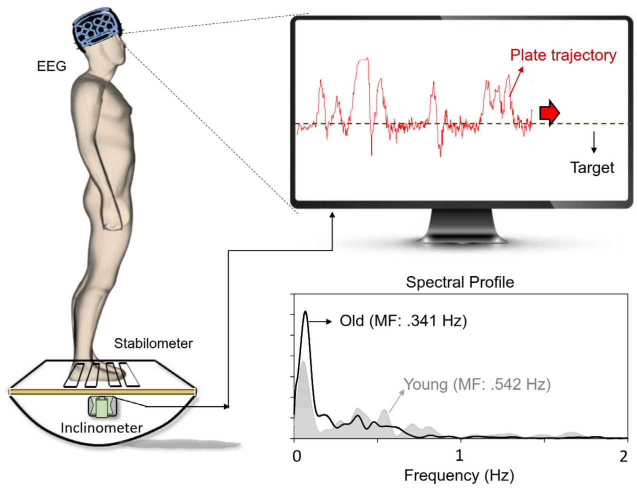

Fig. 1. System setup of physiological measures during stabilometer stance. Subjects maintained upright posture on a stabilometer, guided by real-time visual feedback on a computer screen displaying both angular plate movement and a horizontal target line. Below, spectral profiles and mean frequency (MF) of postural fluctuations for typical subjects across different age groups were showcased. Notably, these postural fluctuations predominantly comprised low-frequency components under 2 Hz.

D. Data Analysis

The angular plate movements in degrees were initially processed using a zero-phase low-pass filter with a cut-off frequency of 4 Hz. Postural fluctuations were obtained from these angular plate movements after eliminating linear trends. To ensure data consistency, the initial and final 2 seconds of postural fluctuations in each trial were excluded from subsequent analysis. Subsequently, the time series of postural fluctuations, amounting to 56 seconds, were decomposed into amplitude and phase components using the Hilbert transform (Fig. 2(A)). The stability of postural sway during stabilometer stance was indexed with root mean square (RMS) of the postural fluctuation amplitude. In addition, this study focused on the dynamics of the postural fluctuation phase. The size and regularity of the phase components of postural fluctuations were quantified using RMS and sample entropy (SampEn). SampEn, which ranges from 0 to 2, serves as an indicator of complexity of the postural fluctuation phase, with higher values denoting greater complexity. The mathematical formula of sample entropy was

$$SampEn\,(m, r, N) = -\log\left(\frac{\sum_{i=1}^{N-m} A_i}{\sum_{i=1}^{N-m} B_i}\right) \quad (1)$$

where $r = 15\%$ of the standard deviation of the force channel, m is the length of the template ($m = 2$) [27], and N is the number of data points in the time series. A_i is the number of matches of the ith template of length $m + 1$ data points, and B_i is the number of matches of the ith template of length m data points. The complexity measure was performed, and each time scale represented 10 ms. Furthermore, the spectral profiles of phase components of postural fluctuations were estimated with a fast Fourier transform and the Welch method (utilizing a Hanning window with a window length of 20 seconds and 20% overlapping segments). Mean frequency (MF) was determined from the spectra of the postural fluctuation phase (Fig. 2(A)). The purpose of examining the phase dynamics of postural fluctuations was to characterize the temporal aspects of postural adjustments, which might provide additional insights into sensorimotor integration or neural processing related to postural regulation.

All EEG data were preprocessed with band-pass filtering using a zero-phase finite impulse response (FIR) filter (Cut-off frequencies: 1 and 60 Hz (60 dB/octave)). Blinks were detected by generating a bipolar vertical electrooculography (EOG) channel, created by subtracting activity recorded from the infraorbitally-placed electrode from that recorded from the superorbitally-placed electrode. Subsequently, eye movement or blink artifacts were eliminated from the EEG signals using linear regression analysis based on bipolar vertical and horizontal electrooculogram channels, implemented in the NeuroScan 4.3 software program (NeuroScan Inc., El Paso, TX, USA). Akin to the processing of stabilometer plate data, the first and last 2 seconds of EEG signals were excluded from further analysis. For all EEG channels, band-specific EEG signals within theta (4-7 Hz), alpha (8-12 Hz), and beta (13-35 Hz) frequency bands were filtered using a zero-phase FIR band-pass filter (60 dB/octave) (Fig. 2(B)). EEG components potentially contaminated by low-frequency movement artifacts (< 4 Hz) and muscle activity (> 35 Hz) from the head and neck were excluded from the analysis.

We conducted phase-amplitude analysis (PAC) on the postural fluctuation phase and the amplitude of sub-band EEG across all recording electrodes using the modulation index (MI) [28] (Fig. 2(B)). The phases of postural fluctuations were divided into 18 bins based on their phases ($\Delta\varphi = \pi/9$), and the amplitude of each sub-band EEG (theta (4-7 Hz), alpha (8-12 Hz), and beta (13-35 Hz)) was averaged within each phase bin [29]. Subsequently, the mean amplitude across all sub-band oscillations was averaged for each of the 18 phase bins, resulting in a band-specific phase-amplitude plot for each experimental trial. This study employed the modulation index (MI) due to its robustness against confounding factors such as signal-to-noise ratio and data length [29], [30]. MI utilizes the Kullback-Leibler distance and Shannon entropy to quantify the divergence of a phase-amplitude plot. It is mathematically formulated as

$$MI = \frac{\log(N) + \sum_{j=1}^{N} P(j) \log|P(j)|}{\log(N)} \quad (2)$$

where P(j) is the amplitude for a given bin j; N is the number of bins (N = 18), and log(N) represents the entropy of a uniform distribution.

The observed modulation index (MI) values underwent a standardization process to normalize the calculated MI with a distribution of shuffled coupling values, as outlined by Hülsemann et al. (2019) [29]. Shuffled coupling values were obtained by computing the MI value between permuted time series of the phase components of postural fluctuations and amplitude time series of sub-band EEG for each EEG channel. This shuffling procedure was iterated 250 times in this study. The observed MI was standardized to the distribution of shuffled coupling values to obtain a standardized modulation

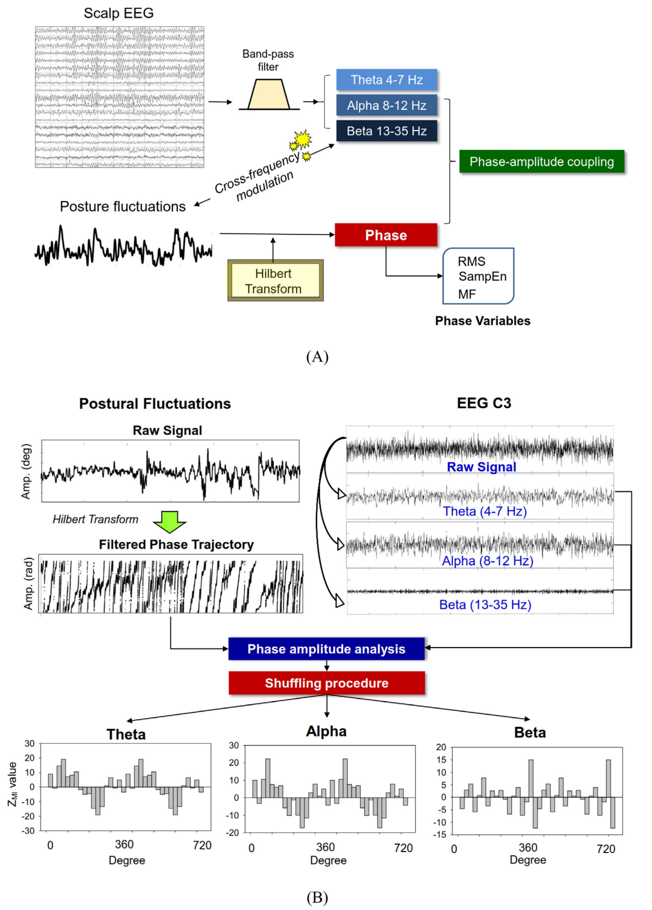

Fig. 2. (A) Flowchart illustrating the signal processing steps to obtain phase-amplitude between postural fluctuations and scalp EEG. The phase component of the postural fluctuations was extracted with the Hilbert transform. The scalp EEG was conditioned with band-pass filter to sub-band EEG (theta: 4-7 Hz, alpha: 8-12 Hz, and beta: 13-35 Hz). Cross-frequency modulation between low-frequency postural fluctuations and high-frequency EEG is characterized with band-specific phase-amplitude coupling (PAC). (B) Schematic illustration depicting the quantification of PAC from a typical dataset. PAC is calculated between the phase component of postural fluctuations and the amplitude of different sub-band EEG signals. Shuffling procedure is used to standardize the PAC with the Z-value of PAC (Z_{MI}) in eighteen phase bins.

index (Z_{MI}) using the following formula:

$$Z_{MI} = MI_{observed} - \frac{\mu MI_{shuffled}}{\sigma MI_{shuffled}} \quad (3)$$

where MI denotes the PAC coupling value, μ denotes the mean, and σ denotes the standard deviation (SD). Data analysis was conducted offline in Matlab R2019a (The Mathworks Inc., Natick, USA).

E. Statistical Analysis

The postural and standardized PAC variables from the three experimental trials were averaged for each subject. An independent t-test was employed to compare the root mean square (RMS) of postural fluctuation amplitude between young and older adults. Additionally, multivariate Hotelling's T-squared statistics were applied to evaluate the age effect on variables such as RMS, sample entropy (SampEn), and mean frequency (MF) of the phase components of postural fluctuations. Subsequently, post-hoc tests were conducted using independent t-tests, with significance determined using the Holm's stepdown test to ascertain the level of significant difference. The Holm's test, unlike the Bonferroni test, avoids over-correction. For all post-hoc hypotheses ($H = \cap_{i=1}^{m}$), the Holm's test did not reject elementary H_i if $p_i \leq i*0.05/m$ for ordered unadjusted p values ($p_1 \leq \ldots \leq p_m$). The type 1 error rate using the Holm's test was exactly 0.05. Pearsons correlation analysis was conducted to assess the significance of the correlation between the RMS of postural fluctuation amplitude and all variables of the postural fluctuation phase, which were found to be age-dependent. The Z_{MI} values of all EEG electrodes from both young and older adults were compared using independent t-tests to identify age-dependent regions of interest across different EEG spectral bands. For the theta, alpha, and beta bands, the Z_{MI} values within the regions of interest were pooled for further analysis. Independent t test was applied to assess the age effect (young vs. older) on the pooled Z_{MI} of the region of interest. All statistical analyses were performed in IBM SPSS Statistics (v19). The level of significance was 0.05.

TABLE I
DIFFERENCES IN POSTURAL FLUCTUATION AMPLITUDE DURING STABILOMETER STANCE BETWEEN YOUNG AND OLDER ADULTS

	Young	Old	Statistics
RMS_Amp. (deg.)	1.410 ± 0.710	2.797 ± 1.250	t_{38} = -4.475, $p < 0.001$

III. RESULTS

Table I outlines the outcomes of an independent t-test conducted on the root mean square (RMS) of postural fluctuation amplitude, comparing young and older adults. The results indicated that older adults displayed a significantly greater RMS of postural fluctuation amplitude compared to young adults ($p < 0.001$), indicating an aging-related decline in balance control during stabilometer stance. Table II presents a comparison of Hotelling's T-squared statistics concerning the phase variables (RMS, SampEn, and MF) of postural fluctuations between young and older adults. The analysis revealed that these variables were age-dependent (Wilks' $\Lambda = 0.752$, $p = 0.015$). Post-hoc analysis further demonstrated that the RMS ($p = 0.045$), SampEn ($p = 0.020$), and mean frequency (MF) ($p = 0.005$) of the postural fluctuation phase in older adults were smaller than those observed in younger adults. Pearson's correlation analysis indicated a negative correlation between postural fluctuation amplitude measured by RMS and the MF of the postural fluctuation phase (r = -0.328, p = 0.039). However, there was no significant correlation observed between SampEn and RMS of the postural fluctuation phase with postural fluctuation amplitude ($p > 0.05$), as detailed in Table III.

Figure 3(A) depicts the aggregated topological distribution of phase-amplitude coupling (PAC) between the postural fluctuation phase and amplitude of theta oscillation (4-7 Hz) in both young and older adults during stabilometer stance. Regarding the Z_{MI}, there was a tendency for greater PAC

TABLE II
COMPARISON OF VARIABLES POSTURAL FLUCTUATION PHASES DURING STABILOMETER STANCE BETWEEN YOUNG AND OLDER ADULTS

	Young	Old	Pos-hoc test
RMS (radian)	1.771 ± 0.124	1.702 ± 0.083	t_{38} = 2.076, p=0.045
SampEn	0.154 ± 0.056	0.117 ± 0.037	t_{38} = 2.428, p=0.020
MF (Hz)	0.630 ± 0.148	0.512 ± 0.100	t_{38} = 2.953, p=0.005
Hotelling's Statistics	Wilks' Λ = 0.752 ,	p = 0.015	

TABLE III
PEARSON CORRELATION BETWEEN POSTURAL FLUCTUATION AMPLITUDE AND VARIABLES OF POSTURAL FLUCTUATION PHASE

	RMS_phase	SampEn_phase	MF_phase
RMS_Amp.	r= -0.058, p=0.723	r= -0.286, p=0.073	r= -0.328, p=0.039

between the postural fluctuation phase and theta oscillation amplitude in young adults compared to older adults. Independent t-tests revealed regions of interest with age-related differences in theta PAC, including the FCz and bilateral temporal-parietal-occipital (TPO) areas (TP7, T5, CPz, TP8, P4, T6, O1, and O2) (p < 0.05). Figure 3(B) compares the pooled ZMI of the PAC of the theta band in the regions of interest (FCz and TPO) between young and older adults. The results of independent t-tests indicated that the PAC in the theta band of FCz (t_{38} = 2.094, p = 0.043) and TPO (t_{38} = 2.887, p = 0.006) for older adults was smaller than that observed in young adults. Figure 4(A) illustrates the aggregated topological distribution of PAC between the postural fluctuation phase and amplitude of alpha oscillation (8-12 Hz) during stabilometer stance for both young and older adults. Similar to theta PAC, alpha PAC was visually greater in young adults than in older adults. Independent t-tests revealed age-related differences in PAC in the T5, O1, CPz, and right parietal-temporal-occipital (RPT) (TP8, P4, and T6) areas (p < 0.05). Figure 4(B) compares the pooled ZMI within the regions of interest (T5O1, CPz, and RPT) of the PAC in the alpha band between young and older adults. The results of independent t-tests indicated that the PAC of T5O1 (t_{38} = 3.023, p = 0.004), CPz (t_{38} = 2.216, p = 0.033), and RPT (t_{38} = 2.714, p = 0.009) in the alpha band for older adults was smaller than that observed in young adults.

Figure 5(A) displays the combined topological distribution of PAC between the postural fluctuation phase and amplitude of beta oscillation (13-35 Hz) during stabilometer stance for both young and older adults. In contrast to the PAC observed in the theta and alpha bands, the PAC between the postural fluctuation phase and amplitude of EEG beta oscillation appeared visually stronger in older adults than in young adults. Independent t-statistics revealed significant age-related differences in beta PAC, specifically in the C3 electrode (t_{38} = −2.905, p = 0.006), where beta PAC was greater in older adults than in young adults.

IV. DISCUSSION

At the behavioral level, this study identified age-related differences in the phase dynamics of postural fluctuation (RMS, SampEn, and MF) on uneven surfaces utilized for postural training. Notably, a lower MF of the postural fluctuation phase was associated with a larger postural fluctuation size during stabilometer stance. Regarding cortical-behavioral aspects, age-related variances in PAC between postural fluctuation phase and scalp EEG were observed across frequency bands. Young adults displayed higher PAC in the theta and alpha bands compared to older adults, particularly in the temporal-parietal-occipital areas. Conversely, older adults exhibited higher PAC in the beta band in the left primary motor cortex compared to young adults.

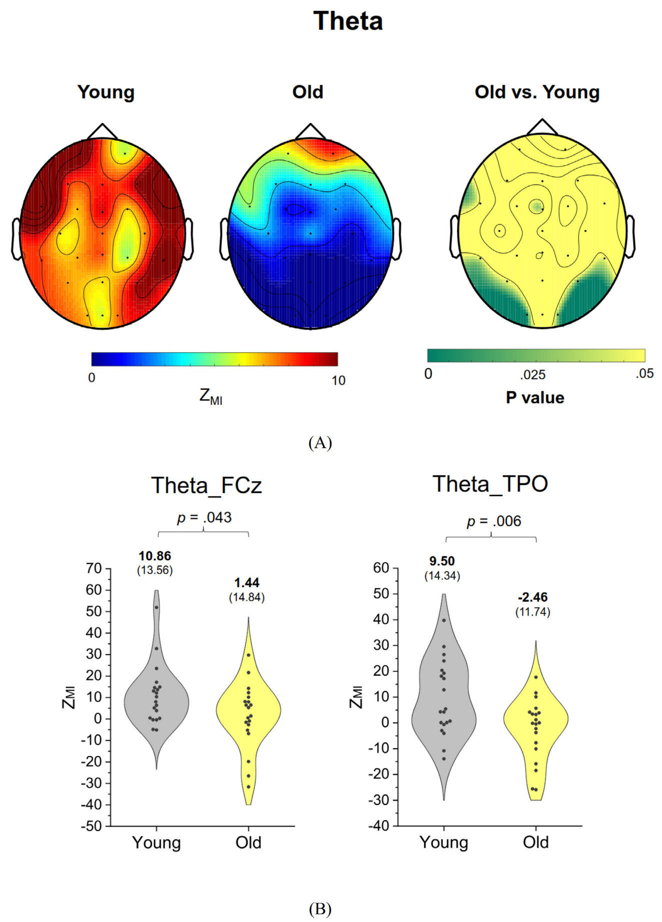

Fig. 3. Scalp maps illustrating the contrasting topological distribution of phase-amplitude coupling (PAC) in the theta band (4-7 Hz), which involves the phase component of postural fluctuations and band-specific EEG activity, between young and older populations. (A) Pooled topological distributions of PAC observed in both young and aged populations, along with age-related differences in PAC. These differences are depicted through the topological distribution of P values, derived from the results of independent t-tests. (B) Mean and standard deviations of pooled standardized z-values are provided for regions of interest, specifically the FCz and bilateral temporal-parietal-occipital area (TPO).

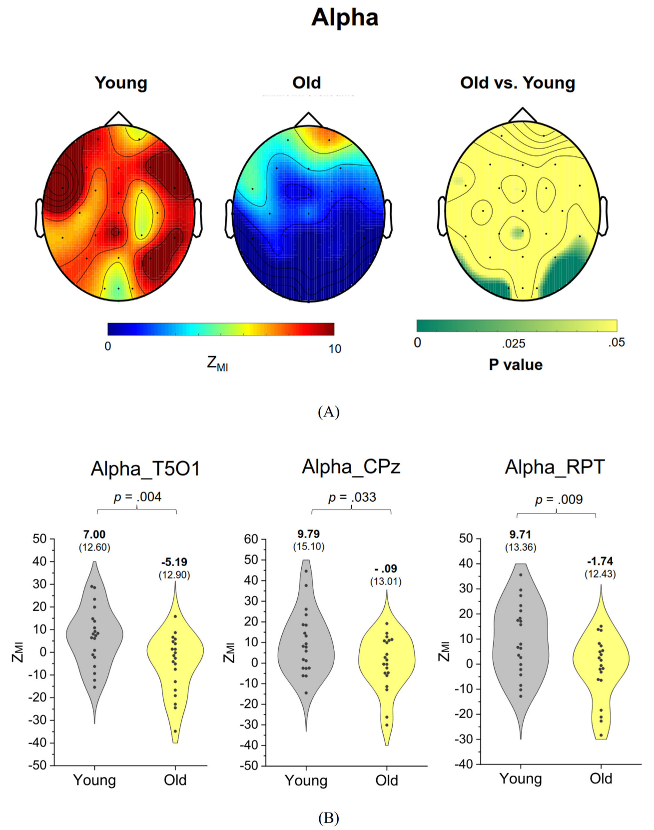

Fig. 4. The scalp maps illustrate the contrasting topological distribution of phase-amplitude coupling (PAC) in the alpha band (8-12 Hz), which involves the phase component of postural fluctuations and band-specific EEG activity, between young and older populations. (A) Pooled topological distributions of PAC observed in both young and aged populations, along with age-related differences in PAC. These differences are depicted through the topological distribution of P values, derived from the results of independent t-tests. (B) Mean and standard deviations of pooled standardized z-values are provided for regions of interest, specifically the T5O1, CPz, and right parietal-temporal area (RPT).

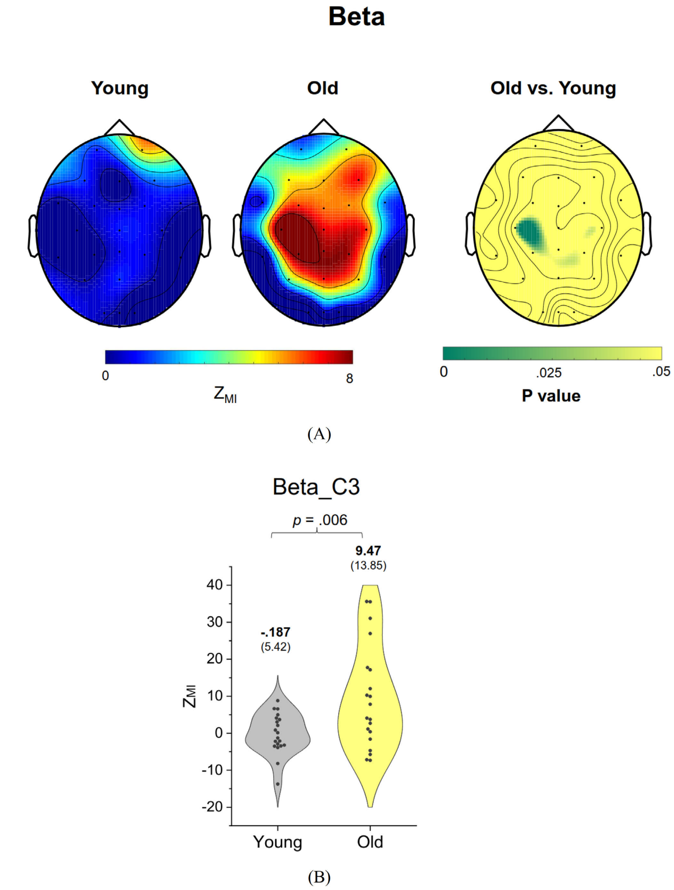

Fig. 5. Scalp maps illustrating the contrasting topological distribution of phase-amplitude coupling (PAC) in the beta band (13-35 Hz), which involves the phase component of postural fluctuations and band-specific EEG activity, between young and older populations. (A) Pooled topological distributions of PAC observed in both young and aged populations, along with age-related differences in PAC. These differences are depicted through the topological distribution of P values, derived from the results of independent t-tests. (B) Mean and standard deviations of pooled standardized z-values are provided for regions of interest, specifically the C3 area.

A. Age-Related Phase Dynamics of Postural Fluctuations

In addition to the decline in postural stability during stabilometer stance (Table I), the phase dynamics of postural fluctuation revealed several interesting aspects of age-related adaptations in postural strategy (Table II). Within the framework of intermittent control mechanisms [31], [32], postural fluctuations reflect intermittent corrective responses aimed at counteracting perturbations and maintaining balance at the target level. The intermittent nature of postural regulation (0.2-2 Hz) represents a compromised mechanism to cope with noisy inputs [33] and delayed sensory feedback [34] within closed-loop systems, while conserving energy and minimizing effort. Functionally, intermittent postural control operates in a state-dependent manner to refine postural trajectories with reference to visual cues [4], [35]. As a result, the decrease in RMS and SampEn of the postural fluctuation phase implies that the older adults adjusted their postural phase in a more passive and simplified manner; this implication conceptually aligns with the loss of postural complexity with aging [7], [8]. However, the reduced responsiveness to balance contexts does not directly contribute to postural instability with aging ($p > 0.05$) (Table III). The only metric of stance instability was MF of the postural fluctuation phase ($p = 0.039$) (Table III). In terms of mean frequency of postural fluctuation phase (Table II), the older adults who displayed less frequent phase shifts in postural fluctuation experienced more destabilization on the stabilometer, probably because they made fewer intermittent adjustments to maintain balance. The ineffective postural strategies adopted by older adults at the behavioral level stem from compensatory reorganization of cortical regions following degenerative changes in the sensorimotor system.

B. Age-Related phase-amplitude Postural Fluctuation Phase and Scalp EEG

The traditional approach uses coherence spectra to characterize the degree of linear dependency between brain activity and body kinematics (or muscular activity) at similar frequency bands [36], [37], [38]. Traditional cortico-peripheral coupling characterized by coherence analysis is limited when it comes to examining cross-frequency modulation, such as between postural fluctuations (<1 Hz) and EEG activity (> 4 Hz). As phase-amplitude analysis, commonly used to represent inter-regional cortical connectivity [28], [29], [30], can specify how the phase of a lower-frequency signal modulates the amplitude of a higher-frequency signal. Phase-amplitude coupling (PAC) offers a sophisticated method to explore the nonlinear nature of brain-posture interactions. By leveraging PAC, this approach illuminates new dimensions of central regulation of postural fluctuations (or postural intermittency) which have recently been found to be time-locked to the amplitude of cortical potentials at various bands. Zaback et al. solely targeted the Cz electrode and successfully recorded event-related potentials synchronized with the local peaks of the time series of the center of pressure in the anterior-posterior direction. The authors also highlighted that event-related potentials became more prominent as cortical engagement in balance control increased, particularly under challenging postural tasks [15]. Extending that previous work [9], [26], [39], this study further revealed the topological organization of phase-amplitude coupling (PAC) between postural fluctuations and cortical oscillations to be band-specific and age-dependent during unsteady stance (Figs. 3-5). The phases of postural fluctuations synchronize with cortical oscillations in the theta, alpha, and beta bands across various cortical areas involved in postural neural networks (Figs. 3, 4, and 5).

Notably, young adults predominantly utilized theta and alpha oscillations to regulate postural fluctuations (Figs.3 and 4), whereas older adults exhibited higher phase-amplitude coupling, particularly in the beta band at C3 (Fig. 5). According to the frontal aging hypothesis [40], older individuals might face increased challenges in interpreting spatial representations of postural errors [41], [42]. Regarding loss-related responses to visuo-spatial errors with aging, the reduced theta PAC in FCz (Fig. 3(B)) suggests that older adults may struggle to adjust the phases of postural fluctuations in response to visualized error feedback. This age-related difficulty in error detection and monitoring has previously been evidenced by the decreased activation and connectivity of mid-frontal theta oscillation during stabilometer stance [4], [16]. Therefore, the theta phase-amplitude decoupling in this study is assumed to reflect a reduced capacity for error-based anticipation and planning of corrective responses in postural phase against stance destabilization in older adults. Although visual feedback is mostly important for maintaining stance, it is undeniable that maintaining an upright posture while visually pursuing a target line relies on multi-sensory integration of the visual, vestibular, and somatosensory systems. For young adults, multi-sensory integration provides complementary information about the body's position and motion, with which the brain can cross-validate visual signals and enhance the reliability of perceptual estimates of postural orientation. In contrast to young adults, who make better use of multi-sensory integration, older adults prioritize the minimization of sensory ambiguity by favoring the least affected visual channel to compensate for other non-visual systems [9], [43]. The reliance on visual monitoring for older adults as a compensatory strategy for balance-correcting responses could also be compensatory for earlier degeneration of error detection within the mid-frontal system. Therefore, the increased demand on visuospatial processing could compromise the PAC between postural fluctuations and alpha oscillations in the posterior cortex for older adults during stabilometer stance (Figure 4(B)). This compromise would be consistent with the predictions of the cortical idling hypothesis, which associates states of diminished alpha power with enhanced neural activation [44], [45]. Zhou et al. reported that lower pre-stimulus alpha power in occipital-parietal areas improved perceptual sensitivity in a visual detection task, implying that posterior alpha oscillations play a role in downstream gating of excitability in visual regions [46], [47]. Beta oscillations in the sensorimotor cortex are functionally associated with motor execution and sensorimotor integration [48]. Beta synchronization represents cortical information processing for maintaining "the status quo" [49], allowing fine movement adjustments with reliable feedback processes and predictable outcomes [50]. A surge in beta power (or beta rebound) concurs with the recovery phase of the postural perturbation [19], [51]. Alternatively, beta desynchronization takes place for altering "the status quo" to allow for the transition of the motor state to adapt to an impending perturbation [20], [52]. During stabilometer stance, the high PAC in the beta band suggests that older adults inappropriately sustain motor states (i.e., response inhibition [14], [53]) under destabilization. This argument is behaviorally supported by the fewer attempts to correct postural deviations (lower MF of the postural fluctuation phase) (Table III) and greater postural sway (Table I) in the older adults. Such an inflexible postural strategy adopted by older adults is disadvantageous to postural stability, as it hinders the timely updating of postural responses and reduces adaptability to postural perturbations. The enhanced beta PAC observed at the C3 electrode might suggest that older adults predominantly utilize their right dominant lower limb to regulate stabilometer stance in a more rigid manner.

Despite the novel application of PAC in examining age-related effects on cortical control during stabilometer stance, extending these initial findings to other stance conditions should be approached with prudence. Further research on PAC, encompassing a broader range of stance situations, is necessary in the future.

V. Conclusion

This study unveils novel neural evidence highlighting age-related differences in the dynamics of the postural fluctuation phase and brain-behavior coupling on uneven surfaces. The downward spectral shift in postural fluctuation phases is associated with reduced stance stability among older adults navigating such surfaces. Additionally, phase-amplitude analysis reveals a weaker coupling between postural fluctuation phases and mid-frontal theta and alpha oscillations in the posterior area among older adults. Conversely, older adults exhibit stronger phase-amplitude coupling with beta EEG oscillations in the left primary motor cortex. These findings collectively imply age-related declines in active error monitoring and flexible motor state transitions in destabilization contexts. Furthermore, older adults exhibit a compensatory reliance on visual cues for postural regulation during stabilometer stance, particularly in the context of postural training.

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