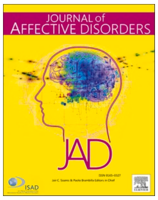
Journal of Affective Disorders 400 (2026) 121185

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad



Research paper

Altered cognitive performance and frontal alpha asymmetries during emotion–cognition interactions in older adults with a history of depression and/or anxiety

Fan Peng [a,*], Ada W.S. Leung [a,b], Anthony Singhal [a,c,**]

[a] Neuroscience and Mental Health Institute, University of Alberta, Edmonton, Alberta, Canada
[b] Department of Occupational Therapy, University of Alberta, Edmonton, Alberta, Canada
[c] Department of Psychology, University of Alberta, Edmonton, Alberta, Canada

ARTICLE INFO

Keywords:
Emotion-cognition interaction
Frontal alpha asymmetry
Occipital alpha
Aging
Depression/anxiety
EEG

ABSTRACT

Emotion–cognition interactions are essential for mental health in aging, yet their disruption in older adults with a history of affective disorders is poorly understood. We compared older adults reporting a history of depression and/or anxiety (History Group; $n = 20$) to those without such history (No-History Group; $n = 28$) using an emotional oddball task to examine behavioral performance and frontal and occipital alpha asymmetry patterns. Behaviorally, the history group exhibited generally delayed reaction times across all target types and reduced accuracy specifically in targets following fearful distractors, compared with the no-history group, indicating increased susceptibility to emotional interference associated with affective history. EEG analysis (electrodes F3/F4 for frontal and O1/O2 for occipital) revealed distinct frontal alpha asymmetries: the history group exhibited a left visual field bias in frontal alpha following fearful distractors, alongside a right-frontal dominance across target types. In contrast, the No-history group showed balanced bilateral frontal activation across target conditions. No group differences emerged in occipital alpha asymmetry, suggesting comparable visual processing strategies. These findings indicate that a history of depression and/or anxiety is associated with altered cognitive task performance and distinct frontal neural signatures in older adults, highlighting the potential value of emotion regulation interventions for supporting cognitive flexibility in this population.

1. Introduction

Mental health disorders are increasingly prevalent among older adults, with depression and anxiety representing two of the most common and burdensome conditions (World Health Organization, 2023). These disorders frequently co-occur and are associated with substantial impairments in daily functioning, emotional well-being, and quality of life (Ochsner and Gross, 2005). Beyond their immediate clinical impact, both depression and anxiety are linked to long-term disruptions in emotion regulation and cognitive control (Joormann and Gotlib, 2010; Kenwood et al., 2022). Accumulating evidence suggests that even a history of affective disorders may be linked to long-lasting neural consequences that increase vulnerability to cognitive decline and dementia in later life (Byers and Yaffe, 2011; Gimson et al., 2018; Ownby et al., 2006). However, how depression and anxiety histories interact with age-related brain reorganization at the neural level remains poorly understood.

Emotion and cognition rely on dynamically interacting, lateralized neural networks. Left-hemisphere engagement is typically associated with approach-related and goal-directed processing, whereas right-hemisphere engagement supports withdrawal-related and monitoring processes (Bartolomeo and Seidel Malkinson, 2019; Harmon-Jones and Gable, 2018). Electroencephalography (EEG) provides a temporally sensitive method to examine these dynamics. In particular, alpha-band oscillations (8–12 Hz) are widely interpreted as an inverse index of cortical excitability, with higher alpha reflecting inhibition (Jensen and Mazaheri, 2010). Frontal alpha asymmetry (FAA), defined as the relative difference in alpha power between left and right frontal regions, has been proposed as a neural marker of individual differences in affective and motivational tendencies (Davidson, 1992, 1998; Coan and Allen,

---

* Corresponding author.
** Correspondence to: A. Singhal, Department of Psychology, University of Alberta, Edmonton, Alberta, Canada.
E-mail addresses: fpeng3@ualberta.ca (F. Peng), asinghal@ualberta.ca (A. Singhal).

https://doi.org/10.1016/j.jad.2026.121185
Received 29 October 2025; Received in revised form 31 December 2025; Accepted 10 January 2026

2004; Harmon-Jones and Gable, 2018). Within this framework, reduced left-frontal alpha is associated with approach-related motivation, whereas reduced right-frontal alpha is linked to withdrawal and avoidance.

Prior research has linked affective disorders to atypical hemispheric asymmetries. Lesion studies associate left-frontal damage with depressive symptoms, suggesting reduced left-hemisphere engagement and relative right-hemisphere predominance (Robinson and Szetela, 1981). Neuroimaging and electrophysiological studies have similarly demonstrated greater right-frontal engagement in depression, reflecting difficulty disengaging from negative stimuli rather than nonspecific hyperactivity (Allen et al., 2004; Pereira and Khan, 2016; Yang et al., 2024). Similarly, anxiety, particularly anxious arousal and panic, has been linked to heightened right-hemisphere activation (Heller et al., 1995, 1997). Furthermore, generalized anxiety disorder has been associated with higher activation in the right temporal lobe (Li et al., 2023) and decreased activity in the left frontal and temporal lobes (Ding et al., 2025).

Despite this theoretical grounding, the diagnostic utility of FAA remains debated. Meta-analyses report negligible effects for resting-state FAA when comparing heterogeneous depressed samples with healthy controls (Luo et al., 2025; van der Vinne et al., 2017). Rather than undermining the relevance of FAA, these findings are increasingly interpreted as reflecting diagnostic heterogeneity: FAA appears to reflect underlying motivational and regulatory mechanisms rather than categorical diagnoses, with asymmetry patterns varying as a function of symptom profile, arousal, and contextual demands (Cantisani et al., 2015; Harmon-Jones and Gable, 2018; Horato et al., 2022). Patients with comorbid depression and anxiety often exhibit greater resting right-lateralized frontal asymmetry (Engels et al., 2010) than those with pure depression (Thibodeau et al., 2006).

A limitation of the existing FAA research is its reliance on resting-state EEG. Resting-state measures lack explicit motivational or emotional engagement and are limited in their ability to reveal compensatory strategies in older adults (Campbell and Schacter, 2017; Coan et al., 2006). In contrast, stimulus-evoked FAA during emotionally demanding tasks provides a constrained and mechanistically informative measure of emotion–cognition interactions (Coan and Allen, 2004; Allen and Reznik, 2015), allowing asymmetries to be examined in response to well-defined emotional and cognitive demands.

Emotionally salient stimuli, especially fear, preferentially capture attention, biasing right-hemisphere processing (Bourne and Vladeanu, 2011; Menon and Uddin, 2010). While adaptive for threat detection, this can impair performance when tasks rely on right-hemisphere networks, such as visuospatial attention and attentional reorienting (Hartikainen et al., 2010; Vossel et al., 2014). Altered hemispheric lateralization associated with depression and anxiety may exacerbate interference and reduce cognitive efficiency (Rogers, 2021).

These affective biases may interact with normative age-related changes. According to the Posterior-to-Anterior Shift in Aging (PASA) model, older adults increasingly recruit frontal executive control networks to compensate for posterior sensory decline (Davis et al., 2008). Aging is also associated with a positivity effect, characterized by preferential processing of positive information and linked to greater left-frontal engagement (Mather, 2016; Mather and Carstensen, 2005). These patterns suggest an age-related shift toward frontal and left-lateralized control processes. Alterations in hemispheric asymmetry may therefore contribute to emotional and cognitive difficulties observed in older adults with depression or anxiety (Ding et al., 2021). However, it remains unclear whether these adaptive shifts persist in older adults with a history of depression or anxiety, who may retain a right-hemisphere withdrawal bias.

To disentangle these mechanisms, it is essential to distinguish between executive and sensory processes. FAA primarily reflects frontal executive regulation and motivational orientation, whereas Occipital Alpha Asymmetry (OAA) indexes selective visual attention, with reduced contralateral occipital alpha indicating enhanced engagement (Worden et al., 2000; Thut et al., 2006). Comparing frontal and occipital asymmetries allows assessment of whether group differences arise from altered sensory selection, compensatory control, or their interaction.

The present study employed an emotional oddball task, a paradigm probing emotion–cognition interactions. Participants detected infrequent targets amid frequent or emotional distractors, with hemivisual field presentation enabling lateralized processing assessment (Bourne, 2006; Briggs and Martin, 2009). Because attentional orienting and vigilance are primarily supported by right-hemisphere networks (Corbetta and Shulman, 2002), this design captures competition between fear and goal-directed attention across hemispheres. We pooled depression and anxiety groups as they share a core neural signature—right-frontal dominance—linked to “anxious arousal” (Bruder et al., 1997; Nitschke et al., 1999; Thibodeau et al., 2006). This grouping aligns with the task's focus on immediate vigilance to examine generalized patterns of negative affect.

We examined stimulus-evoked hemispheric asymmetries in older adults with and without a history of depression and/or anxiety. Based on theoretical frameworks of FAA, affective lateralization, and PASA, we tested the following hypotheses:

1. Older adults with a history of depression and/or anxiety will exhibit reduced task efficiency relative to participants without such history, reflected in slower reaction times and lower accuracy, particularly for targets following fearful distractors.
2. Participants with a history of depression and/or anxiety will show altered hemispheric and visual field–related alpha asymmetries relative to participants without such history.
3. Across the sample, frontal alpha asymmetry effects will be more pronounced than occipital alpha asymmetry effects, consistent with age-related reliance on frontal executive control networks.

2. Methods

2.1. Participants

Twenty-two older adults (6 male) with a history of depression and/or anxiety and thirty older adults (11 male) without such history were recruited from several senior centers in Edmonton. All participants were right-handed. Written informed consent was obtained prior to participation, and the experimental protocol was approved by the Health Research Ethics Board at the University of Alberta. Four participants were excluded: two due to failure to demonstrate normal cognitive functioning based on screening assessments and two due to excessive artifacts in EEG recordings. The final sample consisted of 28 participants in the history group (ages 62–73; M = 68.92, SD = 2.88) and 20 participants in the no-history group (ages 60–75; M = 69.26, SD = 3.25). Demographic information of participants in both groups is shown in Table 1.

2.2. Experimental procedure

All participants first completed an assessment session that included cognitive functioning, depressive symptoms, anxiety symptoms, handedness, and demographic information. The session lasted no more than 20 min per participant, and no participants requested a break. Following the screening session, participants completed an EEG session involving oddball trials, which were split into four blocks of 305 trials each. Fearful distractor trials were pseudorandomized to avoid presenting more than two consecutive trials of the same valence. Each trial began with a jittered fixation lasting between 400 and 1250 ms, followed by the display of a stimulus (standard, distractor, or target) for 1250 ms. To prevent participants from predicting the stimuli, the interval between infrequent stimuli (distractors and targets) was randomized using an exponential distribution, with a median interval of 8 s and a range of 6 to

Table 1
Demographic information.

Characteristic	History Group ($n = 20$)	No-History Group ($n = 28$)
Age (*years*) *M (SD)*	68.92 (2.88)	69.26 (3.25)
Sex, *n* (%)		
Female	14 (70.0)	17 (60.7)
Male	6 (30.0)	11 (39.3)
Education Level, *n* (%)		
Secondary	3 (15.0)	7 (25.0)
Undergraduate Degree	11 (55.0)	18 (64.3)
Postgraduate	6 (30.0)	3 (10.7)
Marital Status, *n* (%)		
Married	7 (35.0)	17 (60.7)
Divorced	7 (35.0)	5 (17.9)
Single	6 (30.0)	5 (17.9)
Other	0 (0.0)	1 (3.6)
Living Status, *n* (%)		
With family	9 (45.0)	19 (67.9)
Living alone	11 (55.0)	9 (32.1)
Number of Children, *n* (%)		
None	8 (40.0)	9 (32.1)
1–2	9 (45.0)	14 (50.0)
3 or more	2 (10.0)	5 (17.9)
Ethnicity, *n* (%)		
White	17 (85.0)	23 (82.1)
Asian	3 (15.0)	3 (10.7)
Black	0 (0.0)	1 (3.6)
Other	0 (0.0)	1 (3.6)

Note: **History**: participants with a depression and/or anxiety history. **No-History**: participants without depression and/or anxiety histories. **n**: sample size. **SD**: standard deviation. **Postgraduate**: Master's or Ph.D. **Degree**: Bachelor's or Diploma programs.

Table 2
Scores for cognitive & mental health screening measures.

Measure	History Group ($n = 20$)	No-History Group ($n = 28$)
MoCA Score		
Mean	27.57	27.88
Range	≥26	≥26
GAD-7 Score (*Anxiety*), *n* (%)		
0–4 (Minimal)	13 (65)	28 (100)
5–9 (Mild)	4 (20)	0 (0)
10–14 (Moderate)	2 (10)	0 (0)
15–21 (Severe)	1 (5)	0 (0)
BDI-II Score (*Depression*), *n* (%)		
0–13 (Minimal)	7 (35)	28 (100)
14–19 (Mild)	4 (20)	0 (0)
20–28 (Moderate)	7 (35)	0 (0)
29–63 (Severe)	2 (10)	0 (0)
History of Affective Disorder(s), *n* (%)		
Anxiety	6 (30)	–
Depression	11 (55)	–
Anxiety & Depression	4 (20)	–

Note: ***History****: participants with a depression and/or anxiety history.* ***No-History****: participants without depression and/or anxiety histories.* **n**: sample size. **MoCA**: Montreal Cognitive Assessment. *Normal: Total Score ≥ 26/30.* **GAD-7**: General Anxiety Disorder 7-Item. *No to minimal anxiety symptoms: Total Score 0–4. Mild anxiety symptoms: Total Score 5–9. Moderate anxiety symptoms: Total Score 10–14. Severe anxiety symptoms: Total Score 15–21.* **BDI-II**: Beck Depression Inventory-Version 2. *Minimal or None Depression: Total Score 0–13. Mild Depression: Total Score 14–19. Moderate Depression: Total Score 20–28. Severe Depression: Total Score 29–63.* **Affective Disorder(s):** Diagnosis of past or current affective disorder(s).

10 s, interspersed with standard stimuli (Peng et al., 2025). Participants were instructed to use one hand to indicate the location of standard stimuli and oddball targets by pressing distinct buttons, and the other hand to respond to emotional distractors (fearful or neutral). Hand assignments were alternated between blocks. Within each group, half of the participants used their left hand to respond to circles (standard stimuli and oddball targets) in the first two blocks, and their right hand in the last two, while the other half followed the opposite order. Participants were instructed to respond as quickly and accurately as possible. All EEG assessments were conducted between 11:00 a.m. and 3:00 p.m. to minimize potential circadian influences on EEG activity and cognitive performance.

2.3. Screening assessments

The assessment battery consisted of the following measures. Cognitive functioning was screened using the Montreal Cognitive Assessment (MoCA), and only individuals scoring ≥26 were included (Nasreddine et al., 2005). Mental health was assessed using the Generalized Anxiety Disorder 7-item scale (GAD-7; ≥5 indicating clinically relevant anxiety; Spitzer et al., 2006) and the Beck Depression Inventory–II (BDI-II; ≥13 indicating clinically significant depressive symptoms; Beck et al., 1996). The no-history group included only participants who reported no history of depression or anxiety and scored below both clinical thresholds. Handedness was assessed using the Edinburgh Handedness Inventory (Oldfield, 1971), and only right-handed participants were included to control for hemispheric lateralization. Individuals with medical, neurological, or sensory conditions known to affect task performance, emotional, or cognitive processing, including epilepsy, stroke, dementia, head trauma, substance abuse, or color vision deficiency, were excluded. Descriptive statistics for all measures are reported in Table 2.

2.4. Task and stimuli

Participants performed a modified emotional oddball paradigm (Peng et al., 2025), with frequent stimuli [75% (912 trials)] and infrequent distracters and oddball targets [25% (308 trials)]. The paradigm consisted of four blocks, with frequent stimuli and oddball targets presented in either the left or right hemivisual field. The frequent stimuli and targets were identical in size and centrally positioned within each visual field. The right visual field was the frequent field in the first two blocks, and the left visual field in the last two. Infrequent distracters included fearful images (68 trials), neutral images (68 trials), and positive images (4 trials). Oddball targets were categorized by the preceding infrequent stimulus: *Target After Fear* (56 trials), *Target After Neutral* (60 trials), and *Target After Target* (52 trials). Neutral images paired with fearful ones were matched for visual qualities. Positive images served as a contextual and attentional anchoring function rather than a primary analytic focus and were therefore excluded from analyses. Distracter images were selected from the International Affective Picture System (IAPS) based on normative valence and arousal ratings, supplemented with in-house images from prior studies (Singhal et al., 2012; Wang et al., 2008).

In this task, participants used one hand to respond to circular visual stimuli, with separate buttons assigned to standard and oddball targets. Response hand assignments were counterbalanced: half participants began by using their left hand to respond to targets during the initial two blocks, switching to their right hand for the final two; the other half followed the opposite sequence. Responses to image-based distractors were made with the non-target-response hand. Emotional salience of distractors was manipulated across blocks: participants were asked to respond only to fearful images in Blocks 1 and 4, and only to neutral images in Blocks 2 and 3. This design encouraged consistent attentional engagement with images.

2.5. Electroencephalography (EEG) recording and analyses

EEG data were collected using a 256-channel high-density HydroCel Geodesic Sensor Net, with signals amplified at a gain of 1000 and sampled at 250 Hz. Electrode impedance was maintained below 50 kΩ (Ferree et al., 2001), and signals were initially referenced to the vertex electrode (Cz). The EEG recordings were bandpass filtered between 0.1 and 50 Hz. Following filtering, data were re-referenced using the average reference approach (Hagemann et al., 2001). To address artifacts, independent component analysis (ICA) was conducted via EEGLAB (Delorme and Makeig, 2004). Components reflecting common sources of noise—such as ocular and muscular activity—were identified through inspection of their spatial maps, time series, and spectral properties, and subsequently removed.

Post-cleaning, time-frequency analysis was carried out using the Better OSCillation detection method (BOSC), which employs a wavelet-based strategy designed to reduce the influence of transient artifacts on power estimates (Caplan et al., 2001; Whitten et al., 2011). We selected a 6-cycle width for the wavelet decomposition because this parameter provides an optimal trade-off between temporal and spectral resolution for the alpha band (8–12 Hz), allowing us to capture transient changes in power without the excessive temporal smoothing associated with higher cycle counts (Cohen, 2014). To quantify sustained oscillatory activity, we first estimated the background noise spectrum by fitting a linear regression to the log-log power spectrum at each electrode, excluding the alpha band peak to model the characteristic *1/f* power drop-off. Oscillatory episodes were defined as periods in which the signal simultaneously exceeded a power threshold set at the 95th percentile of the estimated background distribution and persisted for at least three consecutive cycles. Finally, *P*episode was calculated as the proportion of time within each epoch that the signal met both criteria, ensuring that subsequent asymmetry analyses were driven by sustained neural oscillations rather than broadband artifacts.

Trials were categorized based on target type (target following fearful, neutral, or prior target stimuli) and participant group (history vs. no-history). The analysis window encompassed −300 ms to 1500 ms relative to stimulus onset, with event timings corrected by 36 ms to account for known hardware delay. Two regions of interest were predefined for further analysis: frontal clusters (F3 [41, 36, 40] and F4 [224, 223, 214]), linked to alpha activity modulation during emotion regulation and executive control (Harmon-Jones and Gable, 2018; Thibodeau et al., 2006), and occipital clusters (O1 [116, 108, 115] and O2 [150, 151, 159]), associated with visual processing and spatial attention (Worden et al., 2000; Kelly et al., 2006). The 8–12 Hz alpha band was selected based on existing literature (e.g., Klimesch, 2012; Jensen and Mazaheri, 2010), which highlights its role in cognitive and emotional processing.

2.6. Data analyses

Behavioral outcomes were assessed using accuracy and reaction time (RT). Prior to analysis, RTs were filtered to exclude incorrect responses and anticipatory outliers (< 175 ms), consistent with established thresholds (Peng et al., 2025; Singhal et al., 2012). Data were analyzed using Linear Mixed-Effects Models (LMMs) (Raudenbush and Bryk, 2002) to account for the hierarchical structure of the data (trials nested within subjects). The model included Group (history vs. no-history) and Condition (*Target-After-Fear*, *Target-After-Neutral*, *Target-After-Target*) as fixed effects, along with their interaction. To account for baseline variability between participants, we included a random intercept for each subject (u0j). The model is defined as:

$$\mathrm{Yij} = \beta 0 + \beta 1(\mathrm{Groupj}) + \beta 2(\mathrm{Conditioni}) + \beta 3(\mathrm{Groupj} \times \mathrm{Conditioni}) + \mathrm{u0j} + \varepsilon \mathrm{ij}$$

Reaction time distributions were inspected for skewness. To validate the robustness of the results, models were initially fitted using log-transformed RTs. As the pattern of significance and effect sizes remained consistent between the transformed and raw data, raw RT values (milliseconds) are reported in the final text and figures to facilitate interpretation. Accuracy scores were analyzed as continuous variables. Statistical parameters (Estimates, Standard Errors) were derived from the fitted models. Significance for fixed effects was determined using *t*-tests with Satterthwaite approximations for degrees of freedom. Significant main effects and interactions ($p < .05$) were decomposed using post-hoc pairwise comparisons, corrected with the Bonferroni method to control the error rate.

EEG alpha-band activity (8–12 Hz) was analyzed using mixed-effects ANOVAs to assess the effects of Group (history vs. no-history), Visual Field (left vs. right), and Hemisphere (left vs. right) on oscillatory power. Group was treated as a between-subjects factor, and Visual Field and Hemisphere as within-subject factors. Greenhouse-Geisser corrections were applied where the assumption of sphericity was violated. Significance was set at $p < .05$.

3. Results

3.1. Group differences in behavioral performance in emotion-cognition interactions

The analysis of behavioral performance in emotion-cognition interactions revealed significant differences between older adults with a history of depression and/or anxiety (the history group) and older adults without such history (the no-history group). In terms of RTs (see Table 3 for descriptive results), the history group performed significantly slower RTs than the no-history group (*Estimate* = 16.734, *SE* = 8.200, *t* = 2.041, *p* = .047; see Fig. 1a). For accuracy (see Table 3 for descriptive results), a significant Group × Condition interaction (*Estimate* = −0.010, *SE* = 0.003, *t* = −3.072, *p* = .003) indicated that the history group demonstrated lower accuracy than the no-history group specifically in the *Target-After-Fear* condition (see Fig. 1b). The negative estimate suggests that accuracy declined more steeply in the history group following fearful stimuli compared to no-history group.

3.2. Hemifield and hemispheric asymmetries in frontal alpha during emotion-cognition interactions

Behaviorally, both groups showed similar visual field effects: participants responded significantly faster to LVF than RVF targets ($p < .001$; Fig. 1c and Table 3).

For EEG data, distinct patterns of frontal alpha hemifield asymmetries were observed between the history and no-history groups, reflecting differential patterns of frontal activation during emotion–cognition interactions. In the history group, a left visual field (LVF) bias emerged in *target-after-fear* condition (see Fig. 2a and 3a), indicated by significantly greater frontal activation—reflected by reduced alpha power—in response to targets following fearful stimuli presented in the LVF ($p = .041$; Table 3). Conversely, the no-history group exhibited a right visual field (RVF) bias in *target-after-fear* condition, showing increased frontal activation for right-field targets ($p = .038$; Table 3, Fig. 2a and 3b).

Furthermore, there were differences in hemispheric asymmetries across two groups. Across all target conditions, the history group demonstrated consistently greater right than left frontal activation (*Target After Fear*: $p = .012$; *Target After Neutral*: $p = .011$; *Target After Target*: $p = .004$), suggesting a stable right hemisphere dominance (see Table 4 and Fig. 2b and 3c). The no-history group, by comparison, showed no significant hemispheric asymmetry, indicating more balanced frontal activation during emotion–cognition interactions (see Fig. 2b and 3d).

Additionally, analysis of hemifield-by-hemisphere interactions revealed group-specific differences. In the history group, right frontal activation was selectively enhanced for *Target-After-Fear* presented in the LVF ($p < .001$), whereas the left frontal region responded similarly to both visual fields, demonstrating reduced lateralization (see Fig. 2c and Table 5). The no-history group showed comparable visual field biases in both hemispheres ($p = .003$ and 0.071 for left and right frontal regions), reflecting a bilaterally distributed attentional response without strong hemispheric specialization.

Table 3
Reaction time (ms), accuracy and *P*episode for alpha-band oscillations across participants separated by target visual field and target condition (*Mean(SD)*).

Group	Variable	Target Visual Field		Target Condition		
		Left Field	Right Field	Target-After-Fear	Target-After-Neutral	Target-After-Target
History	Reaction time	507.61 (118.70)	595.25 (114.90)	622.44 (113.25)	585.02 (104.20)	459.35 (72.91)
(*n* = 20)	Accuracy	0.957 (0.084)	0.963 (0.091)	0.929 (0.078)	0.963 (0.038)	0.988 (0.019)
	Frontal Alpha	0.112 (0.087)	0.145(0.118)	0.128 (0.077)	0.111 (0.081)	0.068 (0.035)
	Occipital Alpha	0.058 (0.034)	0.060 (0.038)	0.059 (0.031)	0.055 (0.030)	0.040 (0.028)
No-History	Reaction time	463.49 (114.54)	534.96 (114.39)	559.18 (107.58)	528.49 (118.76)	397.29 (60.43)
(*n* = 28)	Accuracy	0.965 (0.043)	0.969 (0.096)	0.955 (0.060)	0.964 (0.058)	0.982 (0.050)
	Frontal Alpha	0.136 (0.084)	0.106 (0.068)	0.122 (0.077)	0.108 (0.050)	0.079 (0.035)
	Occipital Alpha	0.079 (0.040)	0.065 (0.035)	0.072 (0.036)	0.070 (0.031)	0.060 (0.027)

Note: **History**: participants with a depression and/or anxiety history. **No-History**: participants without depression and/or anxiety histories.

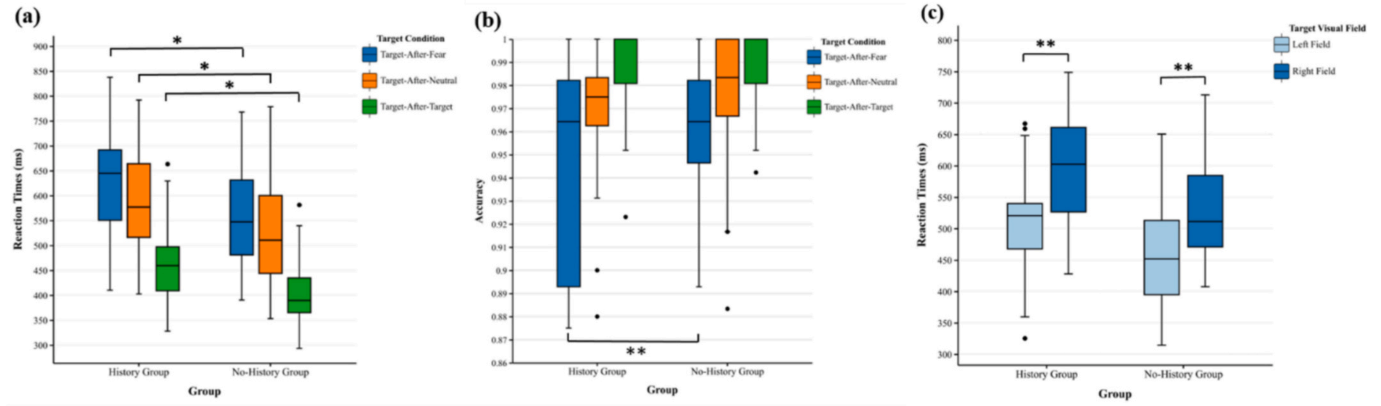


Fig. 1. Behavioral performance between groups and visual fields. (a) Comparisons of reaction times in target conditions between two groups. (b) Comparison of accuracy in target conditions between two groups. (c) Comparison of reaction times in target-after-fear condition between two groups. (*History group:* participants with a depression and/or anxiety history. *No-History group*: participants without depression and/or anxiety histories. **: $p < .01$ significance. *: $p < .05$ significance.)

3.3. Hemifield asymmetries in occipital alpha in visuospatial attention

No significant hemifield asymmetries in occipital alpha power were observed in either group during the *Target-After-Fear* and *Target-After-Neutral* conditions, indicating balanced visuospatial processing when targets followed emotionally salient or neutral distractors. However, in the *Target-After-Target* condition, both groups exhibited significantly greater occipital activation—evidenced by reduced alpha power—in response to right visual field targets ($p = .028$; Table 5 and Fig. 2d), suggesting a shift toward right-field attentional bias when no emotional distractor preceded the target (Fig. 3e and f).

4. Discussion

This study investigated the neural mechanisms underlying emotion-cognition interactions and visuospatial attention in older adults with a history of depression and/or anxiety compared to those without such history. By examining frontal and occipital alpha asymmetries, we were able to reveal group-specific differences in attentional control and emotion regulation that may reflect broader changes in neural processing during aging. The findings highlight how frontal alpha and occipital alpha power demonstrating visual field biases, hemispheric asymmetry, and the effects of emotional distraction in directing attention, shedding light on how emotion regulation and cognitive processing are altered in aging populations, particularly in those with a history of depression and/or anxiety.

4.1. Behavioral performance impairment in older adults with depression and/or anxiety histories

Our behavioral findings align with prior research showing that older adults with depression and/or anxiety histories exhibit cognitive vulnerabilities (Freire et al., 2017; Potter and Steffens, 2007; Steffens and Potter, 2008). While depression and anxiety are established risk factors for cognitive decline (Camacho-Conde and Galán-López, 2020), the present study focuses on characterizing the functional impact of these histories. We demonstrated impairments in real-time task performance, specifically in an emotion–cognition interaction task that required participants to regulate responses to emotional stimuli while directing visuospatial attention. This captures functional impairments in emotion regulation and attentional flexibility which are two core processes critical to maintaining cognitive health in late life.

Importantly, these impairments were particularly pronounced in emotionally salient contexts. A significant Group × Condition interaction for accuracy highlighted that the history group performed particularly worse in the *Target-After-Fear* condition, suggesting that fearful distractors disproportionately disrupt cognitive performance in older adults with depression and/or anxiety histories. These findings align with models positing that emotional interference impairs cognitive control and attentional resources, especially in vulnerable populations (Carstensen et al., 2006; Pessoa, 2009). The impaired accuracy following fearful stimuli in the history group indicates deficits in emotion-cognition interactions, emphasizing the need for interventions targeting emotion regulation and cognitive control in older adults with depressive and/or anxiety histories.

4.2. Frontal alpha hemifield asymmetries in emotion-cognition interactions between groups

In our study, frontal alpha hemifield asymmetries revealed distinct patterns of emotion–cognition interactions between history and no-history groups. When responding to targets following fearful

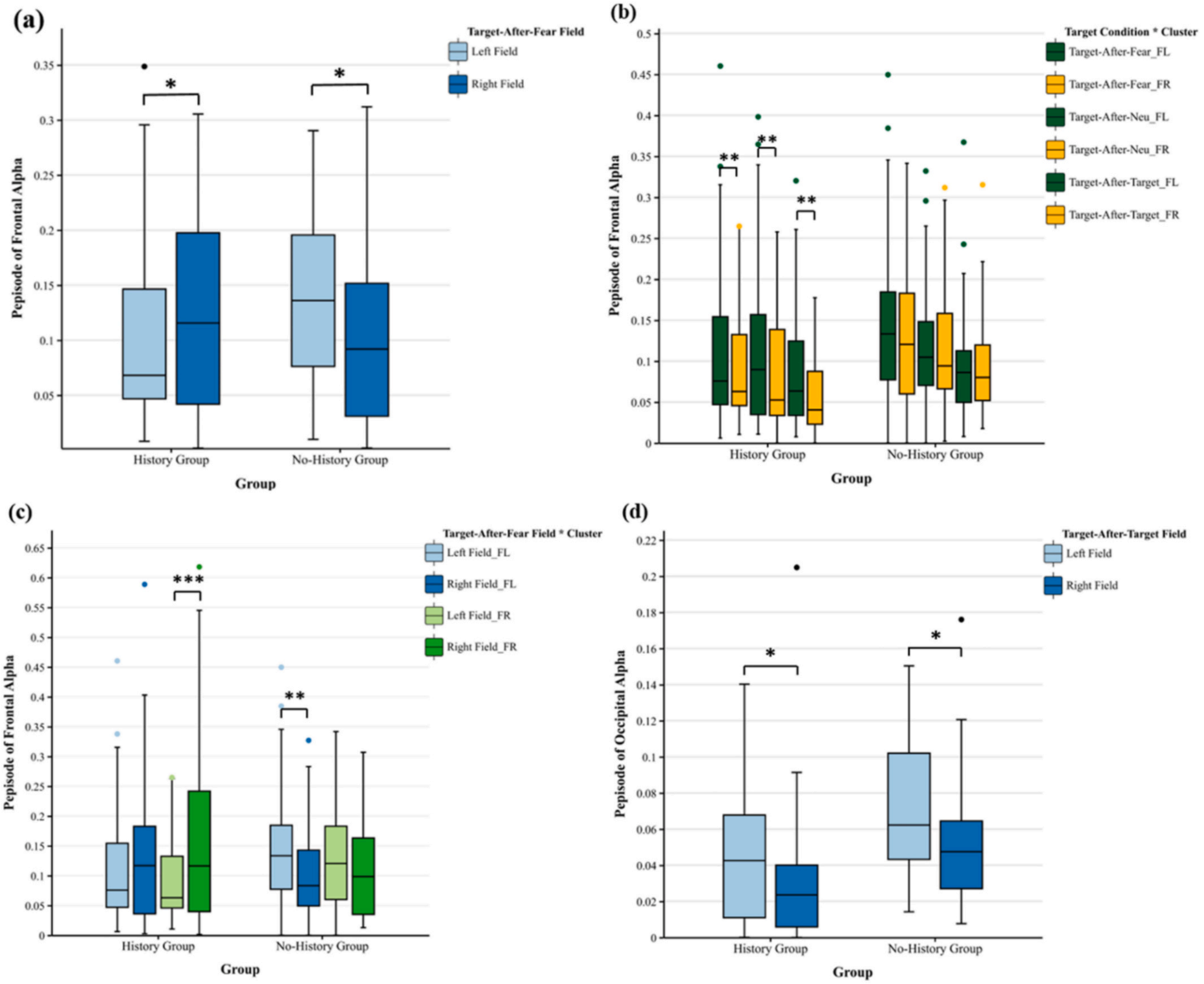


Fig. 2. Alpha oscillations between visual fields and hemispheres. (a) Averaged frontal alpha activity patterns in response to targets following fearful stimuli presented in the left versus right visual field (visual field bias) for both groups. (b) Frontal alpha asymmetries across all target conditions in the history group. (c) Contribution of right frontal alpha to visual field bias during the target-after-fear condition in the history group. (d) Similar averaged occipital alpha activity in response to target-after-target presented in the left versus right visual field for both groups. (*History group:* participants with a depression and/or anxiety history. *No-History group*: participants without depression and/or anxiety histories. ***: $p < .001$ significance. **: $p < .01$ significance. *: $p < .05$ significance).

distractors, the history group demonstrated a LVF bias, while the no-history group exhibited a RVF bias. Specifically, the history group showed greater frontal activation (lower alpha power) to LVF targets ($p = .041$), whereas the no-history group showed increased frontal activation to RVF targets ($p = .038$). Because LVF information projects primarily to the right hemisphere and RVF information to the left (Thut et al., 2006), these patterns indicate that the history group engaged right frontal regions more strongly, while the no-history group relied more on left frontal activation during target processing following fearful distractors. This distinction may reflect differences in affective and motivational tendencies between groups. According to Davidson's model, left frontal regions are associated with approach motivation and positive affect, while right frontal regions support withdrawal motivation and negative affect (Davidson, 1992; Davidson, 2004). Thus, the no-history group's RVF bias likely reflects adaptive engagement of left-lateralized networks that facilitate goal-directed attention and regulation (Harmon-Jones and Gable, 2018). By contrast, the history group's LVF bias suggests greater reliance on right frontal mechanisms linked to sensitivity to negative cues and withdrawal-oriented processing (Coan and Allen, 2004; Suslow et al., 2020; Thibodeau et al., 2006). These findings highlight how differences in hemispheric engagement reflect emotion regulation strategies, with the history group showing patterns indicative of disrupted affective control.

4.3. Right hemisphere dominance in frontal alpha in older adults with depression and/or anxiety histories

The frontal alpha asymmetry patterns observed in the history group across all conditions are indicative of higher activation of the right frontal region, suggesting a predominant role of the right hemisphere in emotion-cognition interactions (Hartikainen, 2021). Rather than a beneficial compensatory mechanism, this increased right hemisphere activation likely reflects a predisposition toward negative emotion processing (Davidson and Irwin, 1999). Sustained right-frontal hyperactivation in depression and anxiety has been linked to impaired engagement of left-hemisphere regulatory networks, resulting in reduced flexibility of emotion regulation and cognitive control (Rotenberg, 2004; Thibodeau et al., 2006; Engels et al., 2007; Coan and

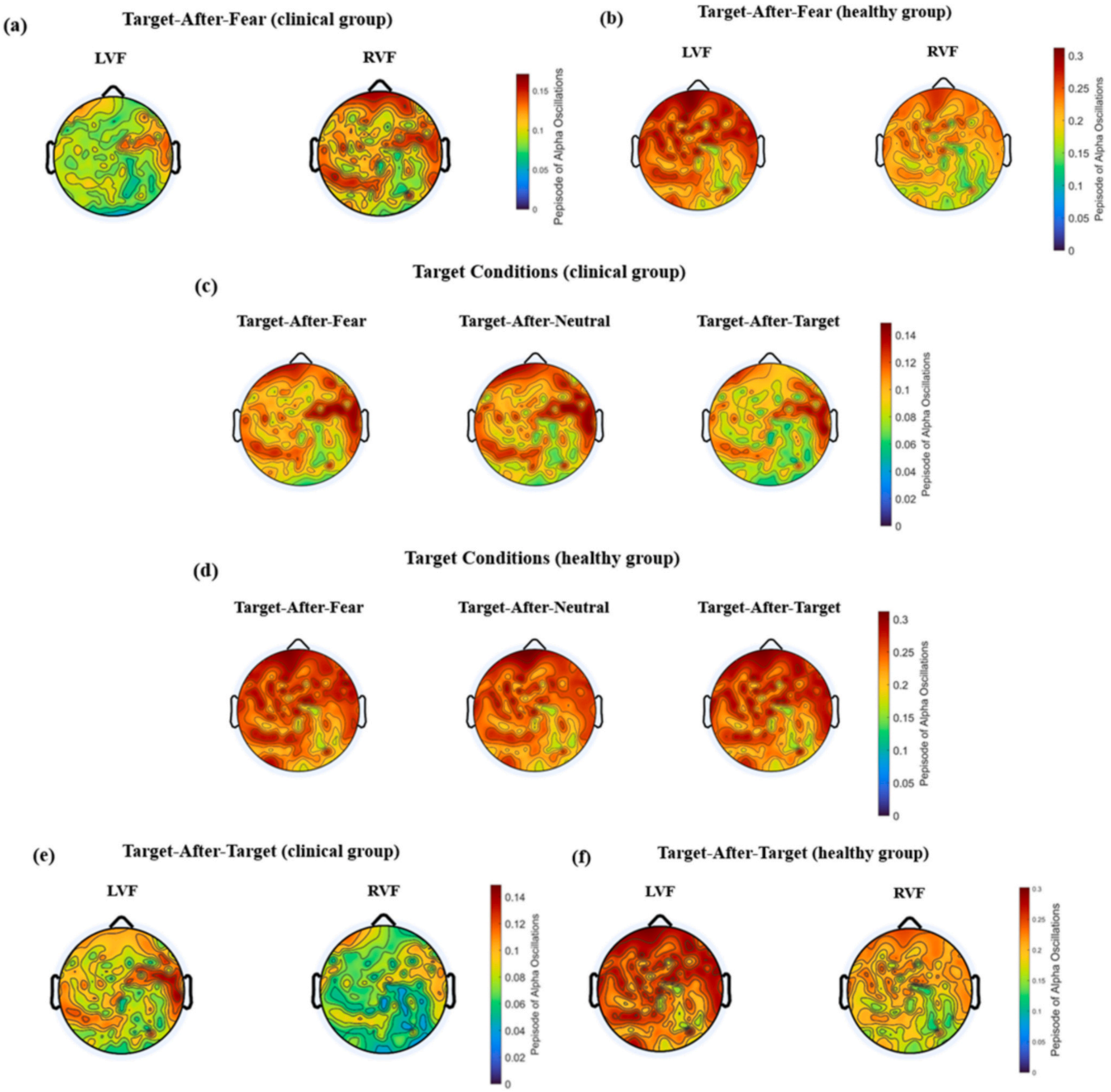


Fig. 3. Topographic distribution of alpha oscillations. (a) History group during the Target-After-Fear condition for left (LVF) and right (RVF) visual fields. (b) No-History group during the Target-After-Fear condition for LVF and RVF. (c) History group in target conditions. (d) No-History group in target conditions. (e) History group during the Target-After-Target condition for LVF and RVF. (f) No-History group during the Target-After-Target condition for LVF and RVF. (*History group:* participants with a depression and/or anxiety history. *No-History group*: participants without depression and/or anxiety histories.)

Table 4

P_{episode} for alpha-band oscillations across participants separated by target condition, and cluster location (*Mean(SD)*).

Group	Electrode cluster	Target-After-Fear	Target-After-Neutral	Target-After-Target
History	L. Frontal	0.133 (0.083)	0.118 (0.084)	0.073 (0.052)
($n = 20$)	R. Frontal	0.124 (0.104)	0.104 (0.073)	0.063 (0.038)
	L. Occipital	0.059 (0.041)	0.054 (0.030)	0.040 (0.026)
	R. Occipital	0.059 (0.045)	0.056 (0.038)	0.040 (0.029)
No-History	L. Frontal	0.120 (0.096)	0.106 (0.075)	0.080 (0.056)
($n = 28$)	R. Frontal	0.121 (0.087)	0.111 (0.073)	0.080 (0.048)
	L. Occipital	0.074 (0.045)	0.072 (0.047)	0.062 (0.039)
	R. Occipital	0.070 (0.041)	0.069 (0.044)	0.058 (0.036)

Note: **History**: participants with a depression and/or anxiety history. **No-History**: participants without depression and/or anxiety histories. **L**: left. **R**: right.

Table 5

P_{episode} for alpha-band oscillations across participants separated by cluster location, target condition and target visual field (*Mean(SD)*).

Group	Electrode cluster	Target-After-Fear		Target-After-Neutral		Target-After-Target	
		Left Field	Right Field	Left Field	Right Field	Left Field	Right Field
History	L. Frontal	0.126 (0.070)	0.140 (0.096)	0.122 (0.087)	0.114 (0.081)	0.082 (0.054)	0.064 (0.049)
($n = 20$)	R. Frontal	0.097 (0.078)	0.151 (0.126)	0.091 (0.062)	0.117 (0.084)	0.060 (0.031)	0.066 (0.044)
	L. Occipital	0.055 (0.034)	0.063 (0.047)	0.055 (0.025)	0.053 (0.034)	0.046 (0.021)	0.034 (0.031)
	R. Occipital	0.062 (0.046)	0.056 (0.045)	0.059 (0.043)	0.052 (0.032)	0.048 (0.036)	0.031 (0.022)
No-History	L. Frontal	0.141 (0.105)	0.100 (0.076)	0.119 (0.081)	0.092 (0.069)	0.095 (0.074)	0.064 (0.038)
($n = 28$)	R. Frontal	0.130 (0.087)	0.112 (0.087)	0.114 (0.072)	0.108 (0.074)	0.093 (0.062)	0.063 (0.035)
	L. Occipital	0.082 (0.044)	0.065 (0.046)	0.080 (0.048)	0.063 (0.045)	0.071 (0.041)	0.052 (0.036)
	R. Occipital	0.076 (0.045)	0.065 (0.037)	0.077 (0.047)	0.061 (0.041)	0.068 (0.038)	0.048 (0.034)

Note: **History**: participants with a depression and/or anxiety history. **No-History**: participants without depression and/or anxiety histories. **L**: left. **R**: right.

Allen, 2004). This asymmetry was particularly pronounced in the *Target-After-Fear* condition, where the history group exhibited higher frontal activation to LVF compared to RVF targets. This pattern reflects the increased processing demands of orienting attention to the left visual field, which is predominantly supported by the right hemisphere. This bias is consistent with prior research indicating that individuals with depression and anxiety exhibit heightened vigilance toward emotionally salient stimuli, a process preferentially mediated by the right hemisphere (Davidson, 1998; Thibodeau et al., 2006). Critically, behavioral performance in the history group was poorer than in the no-history group, with reduced accuracy and slower reaction times. Together, the observed right-hemispheric dominance likely reflects a carryover effect of the threat-related information that interferes with subsequent cognitive efficiency (Hu et al., 2014).

4.4. Bilateral frontal recruitment in older adults without depression/anxiety histories

The no-history group exhibited a pattern of bilateral frontal recruitment, characterized by balanced activation with no significant hemispheric asymmetry. This finding aligns with the HAROLD model (Hemispheric Asymmetry Reduction in Older Adults; Cabeza, 2002), which suggests that healthy aging involves more bilateral neural engagement to support cognitive function. Neuroimaging and electrophysiological studies support this view, showing that older adults typically recruit both hemispheres during attentional tasks, reflecting reduced lateralization compared to younger adults (Casagrande et al., 2021; Dumas, 2015; Learmonth et al., 2017). Unlike the right-lateralized pattern observed in the history group, this bilateral engagement in older adults without depression or anxiety histories likely represents an adaptive recruitment of neural networks during cognitive processing following fearful distraction (Cabeza, 2002; Dolcos et al., 2002; Park and Reuter-Lorenz, 2009).

Importantly, in the *Target-After-Fear* condition, the no-history group showed higher frontal activation to RVF compared to LVF targets, mirroring the neural asymmetry pattern seen in younger adults using the same paradigm (Peng et al., 2025). This RVF bias is consistent with findings from visual neglect research, which indicate that the left hemisphere primarily processes RVF stimuli while the right hemisphere contributes to processing of both visual fields (Heilman and Van Den Abell, 1980; Reuter-Lorenz et al., 1990). However, despite this neural similarity, older adults in the no-history group exhibited unbalanced reaction times, with slower responses to RVF targets compared to LVF targets. In contrast, younger adults show comparable reaction times across visual fields (Peng et al., 2025), suggesting a subtle reduction in neural efficiency with age. Although older adults can recruit additional frontal resources to process RVF stimuli, as evidenced by lower frontal alpha power, this compensatory engagement does not fully preserve the balanced behavioral performance characteristic of younger populations. Future studies could compare frontal asymmetry patterns between mentally healthy younger and older adults to further clarify how age-related changes in neural recruitment relate to cognitive efficiency across visual fields.

4.5. Occipital alpha asymmetry: visuospatial attention

Occipital alpha power is commonly regarded as a marker of visuospatial attention, with lower alpha power indicating greater cortical activation to relevant stimuli (Thut et al., 2006). In this study, no significant hemifield asymmetries in occipital alpha were observed during the *Target-After-Fear* and *Target-After-Neutral* conditions, despite frontal asymmetries. However, in the absence of emotional distractors (*Target-After-Target* condition), both groups exhibited significantly lower alpha power in response to RVF targets. This suggests right-field attentional bias emerges when processing demands are purely visuospatial and aligns with the view that the occipital cortex primarily processes sensory information (Corbetta and Shulman, 2002; Heilman and Van Den Abell, 1980).

Notably, this occipital hemifield asymmetry mirrors the frontal alpha pattern in the no-history group, where RVF biases were more pronounced. This alignment implies that, in healthy aging, there is a preserved coordination between frontal and occipital regions: the frontal cortex directs attentional resources to support emotion regulation and cognitive control (Corbetta and Shulman, 2002; Dolcos and McCarthy, 2006; Kaplan and Berman, 2010), while the occipital cortex processes the spatial input (Heilman and Van Den Abell, 1980; Yang et al., 2024). Conversely, the finding that occipital asymmetry was present in both groups, whereas frontal asymmetry differed, suggests that early visual processing remains intact even when frontal control is compromised.

4.6. Implications for clinical and healthy aging populations

This study provides preliminary insights into how a history of depressive and/or anxiety disorders may relate to patterns of frontal alpha asymmetry, hemispheric lateralization, and visual field processing in older adults. The history group exhibited relatively greater right-hemisphere activation, which could suggest increased reliance on right-lateralized networks for emotion-cognition interactions. In contrast, the no-history group showed more bilateral frontal activation, potentially reflecting adaptive neural recruitment associated with healthy aging. These observations contribute to understanding how affective history may interact with normative age-related brain changes, although caution is warranted in interpreting these findings, given the sample size and cross-sectional design.

While it is tempting to draw clinical implications, these results should be considered exploratory. They suggest that interventions promoting balanced hemispheric engagement could be beneficial, but specific recommendations remain speculative. Potential approaches, supported by prior research, include bilateral movement-based activities (e.g., dance, tai chi, dual-task stepping) that may engage both hemispheres (Serrien et al., 2006; Hackney and Earhart, 2008), EEG-based neurofeedback targeting frontal alpha asymmetry to modulate motivational processes (Harmon-Jones and Gable, 2018; Mennella et al., 2017), and mindfulness or breath-based practices, such as alternate nostril breathing (e.g., Pranayama), hypothesized to influence interhemispheric coordination (Bhaskar et al., 2020; Campanelli et al., 2020). These strategies warrant careful investigation in future studies before any clinical recommendations can be made.

Beyond depression and anxiety, these findings may inform broader questions about emotion-cognition interactions in late adulthood. Several conditions that emerge or persist in older adults, including posttraumatic stress disorder, bipolar disorder, somatic symptom disorder, and mild cognitive impairment with behavioral disturbances, often involve overlapping challenges in emotion regulation, attentional control, and hemispheric functioning (Keller et al., 2022). Investigating patterns of neural asymmetry and compensatory processing across disorders may support transdiagnostic approaches and help identify mechanisms that extend beyond disorder-specific symptomatology (Farras-Permanyer et al., 2019; Mather, 2016; Orlando and Filippini, 2024).

Future research should examine these neural patterns longitudinally and across diverse clinical and nonclinical populations, including individuals with comorbid conditions, to clarify how affective history and aging interact over time. Assessing whether early markers of frontal asymmetry predict cognitive or emotional outcomes, and whether interventions targeting neural plasticity, such as aerobic exercise, cognitive remediation, or neurofeedback, can modulate hemispheric balance, will be important next steps. Such work could inform evidence-based, circuit-level strategies for promoting emotional and cognitive functioning in older adults, without prematurely claiming direct clinical efficacy.

4.7. Limitations

While this study provides preliminary insights into how a history of depression and/or anxiety may influence emotion–cognition interactions and visuospatial attention in older adults, several limitations should be noted. First, the demographic characteristics of our sample warrant careful consideration. The participants in the present study, recruited from Edmonton, Canada, were predominantly highly educated. High education is a well-established factor contributing to cognitive reserve, which can buffer against age-related cognitive decline and support neural compensatory mechanisms. Consequently, the bilateral recruitment observed in our “no-history” group might reflect a “best-case” scenario of successful aging that is not generalizable to older adults with lower socioeconomic status or educational backgrounds. Furthermore, while not controlled for here, the high northern latitude introduces potential environmental variables (e.g., seasonal daylight fluctuations) that future studies should address through diverse geographic sampling.

Second, important clinical and pharmacological confounds were not fully controlled. The history group included participants with past or recurring depressive and/or anxiety symptoms, some of whom had previously used or were currently taking psychotropic medications (e.g., antidepressants, anxiolytics). While we excluded major neurological or psychiatric conditions known to strongly influence neural activity, other common age-related medical conditions, such as hypertension, dyslipidemia, and diabetes, were not systematically controlled. Given the potential effects of medications on both neural activation and behavioral performance, some of the observed differences may reflect pharmacological influences in addition to group-level neural characteristics. Future studies should examine medication effects explicitly or include unmedicated participants to clarify the contribution of depressive/anxiety history independent of treatment.

Third, the classification of the history group presents a methodological limitation. To ensure sufficient power, we combined participants with histories of depression and anxiety into a single group. While this reflects the high comorbidity and shared “general affective distress” (Clark and Watson, 1991), it obscures potential differences between subtypes. For example, anxiety subtypes show distinct lateralization: “anxious apprehension” (worry) is often left-lateralized (Heller et al., 1997), whereas “anxious arousal” (somatic vigilance) is right-lateralized (Nitschke et al., 1999). Depression may involve patterns of hypoactivation or withdrawal distinct from anxiety-related hyperarousal. Pooling these heterogeneous profiles could mask disorder-specific deficits or unique compensatory mechanisms. Future studies with larger samples should examine these diagnostic categories separately to clarify their contributions to neurocognitive aging.

Finally, this study employed a cross-sectional design within a controlled laboratory setting, which limits the ability to draw causal inferences or track how neural patterns in the history group evolve over time. Longitudinal studies are needed to determine whether the right-hemisphere dominance observed in older adults with a history of depression and/or anxiety reflects a stable trait, represents a long-lasting effect of past affective episodes, or emerges as a developing compensatory mechanism. Additionally, the experimental paradigm used static images, which may not fully capture the complexity of real-world emotional regulation. Processing dynamic, naturalistic emotional stimuli may engage neural resources differently than the controlled laboratory conditions implemented here.

4.8. Conclusion

This study demonstrates that older adults with a history of depression and/or anxiety exhibit distinct patterns of emotion–cognition interaction. Specifically, while the no-history group exhibited adaptive bilateral frontal recruitment to preserve accuracy, the history group displayed maladaptive right-hemisphere dominance. This lateralization coincided with impaired cognitive performance following fearful distractors, suggesting that past depressive and/or anxiety disorders leave a lasting impact on emotion regulation and cognitive processing. Consequently, frontal alpha asymmetry serves as a potential neural marker of vulnerability to cognitive and emotional dysregulation. These findings highlight the need for interventions targeting frontal asymmetry to mitigate these enduring risks in older adults with affective histories.

CRediT authorship contribution statement

Fan Peng: Writing – original draft, Visualization, Investigation, Formal analysis, Data curation, Conceptualization. **Ada W.S. Leung:** Writing – review & editing, Validation, Supervision, Resources, Methodology. **Anthony Singhal:** Writing – review & editing, Validation, Supervision, Resources, Methodology, Funding acquisition.

Consent to participant and for publication

All subjects provided written informed consent for their participation and anonymized publication of their data.

Ethics approval

All study materials and experimental procedures were in accordance with 1964 Declaration of Helsinki and its later amendments. This study was approved by the Health Research Ethics Board at the University of Alberta (Pro00130303).

Funding

This work was supported by the University of Alberta internal fund endowed to AS.

Declaration of competing interest

The authors declare no competing financial interests or personal relationships that could have influenced the work reported in this study.

Acknowledgement

The authors thank Mohamed Osman for their valuable assistance with data collection and organization.

Data availability

The data or codes for the experiments are available from the corresponding authors on request. The experiment was preregistered on ClinicalTrials.gov (NCT Number NCT06406595). Permission to use the Montreal Cognitive Assessment (MoCA) was obtained from the official website (https://mocacognition.com/, Certificate No. CAPENFA710874506-01).

References

Allen, J.J., Reznik, S.J., 2015. Frontal EEG asymmetry as a promising marker of depression vulnerability: summary and methodological considerations. Curr. Opin. Psychol. 4, 93–97. https://doi.org/10.1016/j.copsyc.2014.12.017.

Allen, J.J.B., Urry, H.L., Hitt, S.K., Coan, J.A., 2004. The stability of resting frontal electroencephalographic asymmetry in depression. Psychophysiology 41 (2), 269–280. https://doi.org/10.1111/j.1469-8986.2003.00149.x.

Bartolomeo, P., Seidel Malkinson, T., 2019. Hemispheric lateralization of attention processes in the human brain. Curr. Opin. Psychol. 29, 90–96. https://doi.org/10.1016/j.copsyc.2018.12.023.

Beck, A.T., Steer, R.A., Brown, G., 1996. Beck Depression Inventory–II (BDI-II). APA PsycTests. https://doi.org/10.1037/t00742-000.

Bhaskar, L., Tripathi, V., Kharya, C., Kotabagi, V., Bhatia, M., Kochupillai, V., 2020. High-frequency cerebral activation and interhemispheric synchronization following sudarshan kriya yoga AS global brain rhythms: the state effects. Int. J. Yoga 13 (2), 130–136. https://doi.org/10.4103/ijoy.IJOY_25_19.

Bourne, V.J., 2006. The divided visual field paradigm: methodological considerations. Laterality 11 (4), 373–393. https://doi.org/10.1080/13576500600633982.

Bourne, V.J., Vladeanu, M., 2011. Lateralisation for processing facial emotion and anxiety: contrasting state, trait and social anxiety. Neuropsychologia 49 (5), 1343–1349. https://doi.org/10.1016/j.neuropsychologia.2011.02.008.

Briggs, K.E., Martin, F.H., 2009. Affective picture processing and motivational relevance: arousal and valence effects on ERPs in an oddball task. Int. J. Psychophysiol. 72 (3), 299–306. https://doi.org/10.1016/j.ijpsycho.2009.01.009.

Bruder, G.E., Fong, R., Tenke, C.E., Leite, P., Towey, J.P., Stewart, J.E., McGrath, P.J., Quitkin, F.M., 1997. Regional brain asymmetries in major depression with or without an anxiety disorder: a quantitative electroencephalographic study. Biol. Psychiatry 41 (9), 939–948. https://doi.org/10.1016/S0006-3223(96)00260-0.

Byers, A.L., Yaffe, K., 2011. Depression and risk of developing dementia. Nat. Rev. Neurol. 7 (6), 323–331. https://doi.org/10.1038/nrneurol.2011.60.

Cabeza, R., 2002. Hemispheric asymmetry reduction in older adults: the HAROLD model. Psychol. Aging 17 (1), 85–100. https://doi.org/10.1007/s00221-012-3319-x.

Camacho-Conde, J.A., Galán-López, J.M., 2020. Depression and cognitive impairment in institutionalized older adults. Dement. Geriatr. Cogn. Disord. 49 (1), 107–120. https://doi.org/10.1159/000508626.

Campanelli, S., Tort, A.B.L., Lobão-Soares, B., 2020. Pranayamas and their neurophysiological effects. Int. J. Yoga 13 (3), 183–192. https://doi.org/10.4103/ijoy.IJOY_91_19.

Campbell, K.L., Schacter, D.L., 2017. Aging and the resting state: is cognition obsolete? Lang. Cogn. Neurosci. 32 (6), 661–668. https://doi.org/10.1080/23273798.2016.1227858.

Cantisani, A., Koenig, T., Horn, H., Müller, T., Strik, W., Walther, S., 2015. Psychomotor retardation is linked to frontal alpha asymmetry in major depression. J. Affect. Disord. 188, 167–172. https://doi.org/10.1016/j.jad.2015.08.018.

Caplan, J.B., Madsen, J.R., Raghavachari, S., Kahana, M.J., 2001. Distinct patterns of brain oscillations underlie two basic parameters of human maze learning. J. Neurophysiol. 86 (1), 368–380. https://doi.org/10.1152/jn.2001.86.1.368.

Carstensen, L.L., Mikels, J.A., Mather, M., 2006. Aging and the intersection of cognition, motivation, and emotion. In: Birren, J.E., Schaire, K.W. (Eds.), Handbook of the Psychology of Aging, 6th ed. Elsevier, pp. 343–362. https://doi.org/10.1016/B978-012101264-9/50018-5.

Casagrande, M., Agostini, F., Favieri, F., Forte, G., Giovannoli, J., Guarino, A., Marotta, A., Doricchi, F., Martella, D., 2021. Age-related changes in hemispherical specialization for attentional networks. Brain Sci. 11 (9), 1115. https://doi.org/10.3390/brainsci11091115.

Clark, L.A., Watson, D., 1991. Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. J. Abnorm. Psychol. 100 (3), 316–336. https://doi.org/10.1037//0021-843x.100.3.316.

Coan, J.A., Allen, J.J., 2004. Frontal EEG asymmetry as a moderator and mediator of emotion. Biol. Psychol. 67 (1–2), 7–49. https://doi.org/10.1016/j.biopsycho.2004.03.002.

Coan, J.A., Allen, J.J., McKnight, P.E., 2006. A capability model of individual differences in frontal EEG asymmetry. Biol. Psychol. 72 (2), 198–207. https://doi.org/10.1016/j.biopsycho.2005.10.003.

Cohen, M.X., 2014. Analyzing Neural Time Series Data: Theory and Practice. MIT Press, Cambridge, MA. https://doi.org/10.7551/mitpress/9609.001.0001.

Corbetta, M., Shulman, G.L., 2002. Control of goal-directed and stimulus-driven attention in the brain. Nat. Rev. Neurosci. 3 (3), 201–215. https://doi.org/10.1038/nrn755.

Davidson, R.J., 1992. Anterior cerebral asymmetry and the nature of emotion. Brain Cogn. 20 (1), 125–151. https://doi.org/10.1016/0278-2626(92)90065-t.

Davidson, R.J., 1998. Anterior electrophysiological asymmetries, emotion, and depression: conceptual and methodological conundrums. Psychophysiology 35 (5), 607–614. https://doi.org/10.1017/s0048577298000134.

Davidson, R.J., 2004. What does the prefrontal cortex "do" in affect: perspectives on frontal EEG asymmetry research. Biol. Psychol. 67 (1–2), 219–233. https://doi.org/10.1016/j.biopsycho.2004.03.008.

Davidson, R.J., Irwin, W., 1999. The functional neuroanatomy of emotion and affective style. Trends Cogn. Sci. 3 (1), 11–21. https://doi.org/10.1016/s1364-6613(98)01265-0.

Davis, S.W., Dennis, N.A., Daselaar, S.M., Fleck, M.S., Cabeza, R., 2008. Que PASA? The posterior-anterior shift in aging. Cereb. Cortex 18 (5), 1201–1209. https://doi.org/10.1093/cercor/bhm155.

Delorme, A., Makeig, S., 2004. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J. Neurosci. Methods 134 (1), 9–21. https://doi.org/10.1016/j.jneumeth.2003.10.009.

Ding, F., Ying, Y., Jin, Y., Guo, X., Xu, Y., Yu, Z., Jiang, H., 2025. Reduced frontotemporal connectivity during a verbal fluency task in patients with anxiety, sleep, and major depressive disorders. Front. Neurol. 16, 1542346. https://doi.org/10.3389/fneur.2025.1542346.

Ding, Y.D., Yang, R., Yan, C.G., Chen, X., Bai, T.J., Bo, Q.J., Chen, G.M., Chen, N.X., Chen, T.L., Chen, W., Cheng, C., Cheng, Y.Q., Cui, X.L., Duan, J., Fang, Y.R., Gong, Q.Y., Hou, Z.H., Hu, L., Kuang, L., Li, F., Guo, W.B., 2021. Disrupted hemispheric connectivity specialization in patients with major depressive disorder: evidence from the REST-meta-MDD Project. J. Affect. Disord. 284, 217–228. https://doi.org/10.1016/j.jad.2021.02.030.

Dolcos, F., McCarthy, G., 2006. Brain systems mediating cognitive interference by emotional distraction. J. Neurosci. 26 (7), 2072–2079. https://doi.org/10.1523/JNEUROSCI.5042-05.2006.

Dolcos, F., Rice, H.J., Cabeza, R., 2002. Hemispheric asymmetry and aging: right hemisphere decline or asymmetry reduction. Neurosci. Biobehav. Rev. 26 (7), 819–825. https://doi.org/10.1016/s0149-7634(02)00068-4.

Dumas, J.A., 2015. What is normal cognitive aging? Evidence from task-based functional neuroimaging. Curr. Behav. Neurosci. Rep. 2 (4), 256–261. https://doi.org/10.1007/s40473-015-0058-x.

Engels, A.S., Heller, W., Mohanty, A., Herrington, J.D., Banich, M.T., Webb, A.G., Miller, G.A., 2007. Specificity of regional brain activity in anxiety types during emotion processing. Psychophysiology 44 (3), 352–363. https://doi.org/10.1111/j.1469-8986.2007.00518.x.

Engels, A.S., Heller, W., Spielberg, J.M., Warren, S.L., Sutton, B.P., Banich, M.T., Miller, G.A., 2010. Co-occurring anxiety influences patterns of brain activity in depression. Cogn. Affect. Behav. Neurosci. 10 (1), 141–156. https://doi.org/10.3758/CABN.10.1.141.

Farras-Permanyer, L., Mancho-Fora, N., Montalà-Flaquer, M., Bartrés-Faz, D., Vaqué-Alcázar, L., Peró-Cebollero, M., Guàrdia-Olmos, J., 2019. Age-related changes in resting-state functional connectivity in older adults. Neural Regen. Res. 14 (9), 1544–1555. https://doi.org/10.4103/1673-5374.255976.

Ferree, T.C., Luu, P., Russell, G.S., Tucker, D.M., 2001. Scalp electrode impedance, infection risk, and EEG data quality. Clin. Neurophysiol. 112 (3), 536–544. https://doi.org/10.1016/S1388-2457(00)00533-2.

Freire, A.C.C., Pondé, M.P., Liu, A., Caron, J., 2017. Anxiety and depression as longitudinal predictors of mild cognitive impairment in older adults. Can. J. Psychiatr. 62 (5), 343–350. https://doi.org/10.1177/0706743717699175.

Gimson, A., Schlosser, M., Huntley, J.D., Marchant, N.L., 2018. Support for midlife anxiety diagnosis as an independent risk factor for dementia: a systematic review. BMJ Open 8 (4), e019399. https://doi.org/10.1136/bmjopen-2017-019399.

Hackney, M.E., Earhart, G.M., 2008. Tai Chi improves balance and mobility in people with Parkinson disease. Gait Posture 28 (3), 456–460. https://doi.org/10.1016/j.gaitpost.2008.02.005.

Hagemann, D., Naumann, E., Thayer, J.F., 2001. The quest for the EEG reference revisited: a glance from brain asymmetry research. Psychophysiology 38 (5), 847–857. https://doi.org/10.1017/S0048577201001081.

Harmon-Jones, E., Gable, P.A., 2018. On the role of asymmetric frontal cortical activity in approach and withdrawal motivation: an updated review of the evidence. Psychophysiology 55 (1). https://doi.org/10.1111/psyp.12879.

Hartikainen, K.M., 2021. Emotion-attention interaction in the right hemisphere. Brain Sci. 11 (8), 1006. https://doi.org/10.3390/brainsci11081006.

Hartikainen, K.M., Ogawa, K.H., Knight, R.T., 2010. Trees over forest: unpleasant stimuli compete for attention with global features. Neuroreport 21 (5), 344–348. https://doi.org/10.1097/WNR.0b013e328336eeb3.

Heilman, K.M., Van Den Abell, T., 1980. Right hemisphere dominance for attention: the mechanism underlying hemispheric asymmetries of inattention (neglect). Neurology 30 (3), 327–330. https://doi.org/10.1212/wnl.30.3.327.

Heller, W., Etienne, M.A., Miller, G.A., 1995. Patterns of perceptual asymmetry in depression and anxiety; implications for neuropsychological models of emotion and psychopathology. J. Abnorm. Psychol. 104, 327–333. https://doi.org/10.1037//0021-843x.104.2.327.

Heller, W., Nitschke, J.B., Etienne, M.A., Miller, G.A., 1997. Patterns of regional brain activity differentiate types of anxiety. J. Abnorm. Psychol. 106 (3), 376–385. https://doi.org/10.1037//0021-843x.106.3.376.

Horato, N., Quagliato, L.A., Nardi, A.E., 2022. The relationship between emotional regulation and hemispheric lateralization in depression: a systematic review and a meta-analysis. Transl. Psychiatry 12 (1), 162. https://doi.org/10.1038/s41398-022-01927-9.

Hu, T., Zhang, D., Wang, J., Mistry, R., Ran, G., Wang, X., 2014. Relation between emotion regulation and mental health: a meta-analysis review. Psychol. Rep. 114 (2), 341–362. https://doi.org/10.2466/03.20.PR0.114k22w4.

Jensen, O., Mazaheri, A., 2010. Shaping functional architecture by oscillatory alpha activity: gating by inhibition. Front. Hum. Neurosci. 4, 186. https://doi.org/10.3389/fnhum.2010.00186.

Joormann, J., Gotlib, I.H., 2010. Emotion regulation in depression: relation to cognitive inhibition. Cognit. Emot. 24 (2), 281–298. https://doi.org/10.1080/02699930903407948.

Kaplan, S., Berman, M.G., 2010. Directed attention as a common resource for executive functioning and self-regulation. Perspect. Psychol. Sci. 5 (1), 43–57. https://doi.org/10.1177/1745691609356784.

Keller, M., Mendoza-Quiñones, R., Cabrera Muñoz, A., Iglesias-Fuster, J., Virués, A.V., Zvyagintsev, M., Edgar, J.C., Zweerings, J., Mathiak, K., 2022. Transdiagnostic alterations in neural emotion regulation circuits - neural substrates of cognitive reappraisal in patients with depression and post-traumatic stress disorder. BMC Psychiatry 22 (1), 173. https://doi.org/10.1186/s12888-022-03780-y.

Kelly, S.P., Lalor, E.C., Reilly, R.B., Foxe, J.J., 2006. Increases in alpha oscillatory power reflect an active retinotopic mechanism for distracter suppression during sustained visuospatial attention. J. Neurophysiol. 95 (6), 3844–3851. https://doi.org/10.1152/jn.01234.2005.

Kenwood, M.M., Kalin, N.H., Barbas, H., 2022. The prefrontal cortex, pathological anxiety, and anxiety disorders. Neuropsychopharmacology 47 (1), 260–275. https://doi.org/10.1038/s41386-021-01109-z.

Klimesch, W., 2012. Alpha-band oscillations, attention, and controlled access to stored information. Trends Cogn. Sci. 16 (12), 606–617. https://doi.org/10.1016/j.tics.2012.10.007.

Learmonth, G., Benwell, C.S.Y., Thut, G., Harvey, M., 2017. Age-related reduction of hemispheric lateralisation for spatial attention: an EEG study. NeuroImage 153, 139–151. https://doi.org/10.1016/j.neuroimage.2017.03.050.

Li, Q., Zhang, T., Meng, J., Wang, L., Hua, Q., Xie, X.H., Ji, G.J., Bai, T.J., Wang, K., 2023. Abnormal hemispheric specialization and inter-hemispheric functional cooperation in generalized anxiety disorder. Behav. Brain Res. 455, 114660. https://doi.org/10.1016/j.bbr.2023.114660.

Luo, Y., Tang, M., Fan, X., 2025. Meta analysis of resting frontal alpha asymmetry as a biomarker of depression. NPJ Mental Health Res. 4 (1), 2. https://doi.org/10.1038/s44184-025-00117-x.

Mather, M., 2016. The affective neuroscience of aging. Annu. Rev. Psychol. 67, 213–238. https://doi.org/10.1146/annurev-psych-122414-033540.

Mather, M., Carstensen, L.L., 2005. Aging and motivated cognition: the positivity effect in attention and memory. Trends Cogn. Sci. 9 (10), 496–502. https://doi.org/10.1016/j.tics.2005.08.005.

Mennella, R., Patron, E., Palomba, D., 2017. Frontal alpha asymmetry neurofeedback for the reduction of negative affect and anxiety. Behav. Res. Ther. 92, 32–40. https://doi.org/10.1016/j.brat.2017.02.002.

Menon, V., Uddin, L.Q., 2010. Saliency, switching, attention and control: a network model of insula function. Brain Struct. Funct. 214 (5–6), 655–667. https://doi.org/10.1007/s00429-010-0262-0.

Nasreddine, Z.S., Phillips, N.A., Bédirian, V., Charbonneau, S., Whitehead, V., Collin, I., Cummings, J.L., Chertkow, H., 2005. The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. J. Am. Geriatr. Soc. 53 (4), 695–699. https://doi.org/10.1111/j.1532-5415.2005.53221.x.

Nitschke, J.B., Heller, W., Palmieri, P.A., Miller, G.A., 1999. Contrasting patterns of brain activity in anxious apprehension and anxious arousal. Psychophysiology 36 (5), 628–637.

Ochsner, K.N., Gross, J.J., 2005. The cognitive control of emotion. Trends Cogn. Sci. 9 (5), 242–249.

Oldfield, R.C., 1971. The assessment and analysis of handedness: the Edinburgh inventory. Neuropsychologia 9 (1), 97–113. https://doi.org/10.1016/0028-3932(71)90067-4.

Orlando, I., Filippini, N., 2024. Aging modulates frontal lobes involvement in emotion regulation processing. J. Neurosci. Res. 102 (1), e25282. https://doi.org/10.1002/jnr.25282.

Ownby, R.L., Crocco, E., Acevedo, A., John, V., Loewenstein, D., 2006. Depression and risk for Alzheimer disease: systematic review, meta-analysis, and meta-regression analysis. Arch. Gen. Psychiatry 63 (5), 530–538. https://doi.org/10.1001/archpsyc.63.5.530.

Park, D.C., Reuter-Lorenz, P., 2009. The adaptive brain: aging and neurocognitive scaffolding. Annu. Rev. Psychol. 60, 173–196. https://doi.org/10.1146/annurev.psych.59.103006.093656.

Peng, F., Lambert, K.J.M., Bakhtiari, R., Singhal, A., 2025. Handedness modulates emotion-cognition interactions as revealed by ERPs and alpha oscillations. Sci. Rep. 15, 25908. https://doi.org/10.1038/s41598-025-08600-3.

Pereira, D.M., Khan, A., 2016. Hemispheric specialization of impaired disengagement of attention in depression: a tachistoscopic method. Laterality 21 (2), 161–176. https://doi.org/10.1080/1357650X.2015.1090444.

Pessoa, L., 2009. How do emotion and motivation direct executive control? Trends Cogn. Sci. 13 (4), 160–166. https://doi.org/10.1016/j.tics.2009.01.006.

Potter, G.G., Steffens, D.C., 2007. Contribution of depression to cognitive impairment and dementia in older adults. Neurologist 13 (3), 105–117. https://doi.org/10.1097/01.nrl.0000252947.15389.a9.

Raudenbush, S.W., Bryk, A.S., 2002. Hierarchical Linear Models: Applications and Data Analysis Methods, 2nd ed. Sage.

Reuter-Lorenz, P.A., Kinsbourne, M., Moscovitch, M., 1990. Hemispheric control of spatial attention. Brain Cogn. 12 (2), 240–266. https://doi.org/10.1016/0278-2626(90)90018-j.

Robinson, R.G., Szetela, B., 1981. Mood change following left hemispheric brain injury. Ann. Neurol. 9 (5), 447–453. https://doi.org/10.1002/ana.410090506.

Rogers, L.J., 2021. Brain lateralization and cognitive capacity. Animals 11 (7), 1996. https://doi.org/10.3390/ani11071996.

Rotenberg, V.S., 2004. The peculiarity of the right-hemisphere function in depression: solving the paradoxes. Prog. Neuro-Psychopharmacol. Biol. Psychiatry 28 (1), 1–13. https://doi.org/10.1016/S0278-5846(03)00163-5.

Serrien, D.J., Ivry, R.B., Swinnen, S.P., 2006. Dynamics of hemispheric specialization and integration in the context of motor control. Nat. Rev. Neurosci. 7 (2), 160–166. https://doi.org/10.1038/nrn1849.

Singhal, A., Shafer, A.T., Russell, M., Gibson, B., Wang, L., Vohra, S., Dolcos, F., 2012. Electrophysiological correlates of fearful and sad distraction on target processing in adolescents with attention deficit-hyperactivity symptoms and affective disorders. Front. Integr. Neurosci. 6, 119. https://doi.org/10.3389/fnint.2012.00119.

Spitzer, R.L., Kroenke, K., Williams, J.B., Löwe, B., 2006. A brief measure for assessing generalized anxiety disorder: the GAD-7. Arch. Intern. Med. 166 (10), 1092–1097. https://doi.org/10.1001/archinte.166.10.1092.

Steffens, D.C., Potter, G.G., 2008. Geriatric depression and cognitive impairment. Psychol. Med. 38 (2), 163–175. https://doi.org/10.1017/S003329170700102X.

Suslow, T., Hußlack, A., Kersting, A., Bodenschatz, C.M., 2020. Attentional biases to emotional information in clinical depression: a systematic and meta-analytic review of eye tracking findings. J. Affect. Disord. 274, 632–642. https://doi.org/10.1016/j.jad.2020.05.140.

Thibodeau, R., Jorgensen, R.S., Kim, S., 2006. Depression, anxiety, and resting frontal EEG asymmetry: a meta-analytic review. J. Abnorm. Psychol. 115 (4), 715–729. https://doi.org/10.1037/0021-843X.115.4.715.

Thut, G., Nietzel, A., Brandt, S.A., Pascual-Leone, A., 2006. Alpha-band electroencephalographic activity over occipital cortex indexes visuospatial attention bias and predicts visual target detection. J. Neurosci. 26 (37), 9494–9502. https://doi.org/10.1523/JNEUROSCI.0875-06.2006.

van der Vinne, N., Vollebregt, M.A., van Putten, M.J.A.M., Arns, M., 2017. Frontal alpha asymmetry as a diagnostic marker in depression: Fact or fiction? A meta-analysis. NeuroImage. Clinical 16, 79–87. https://doi.org/10.1016/j.nicl.2017.07.006.

Vossel, S., Geng, J.J., Fink, G.R., 2014. Dorsal and ventral attention systems: distinct neural circuits but collaborative roles. Neuroscientist 20 (2), 150–159. https://doi.org/10.1177/1073858413494269.

Wang, L., Huettel, S., De Bellis, M.D., 2008. Neural substrates for processing task-irrelevant sad images in adolescents. Dev. Sci. 11 (1), 23–32. https://doi.org/10.1111/j.1467-7687.2007.00661.x.

Whitten, T.A., Hughes, A.M., Dickson, C.T., Caplan, J.B., 2011. A better oscillation detection method robustly extracts EEG rhythms across brain state changes: the human alpha rhythm as a test case. NeuroImage 54 (2), 860–874. https://doi.org/10.1016/j.neuroimage.2010.08.064.

Worden, M.S., Foxe, J.J., Wang, N., Simpson, G.V., 2000. Anticipatory biasing of visuospatial attention indexed by retinotopically specific alpha-band electroencephalography increases over occipital cortex. J. Neurosci. 20 (6), RC63. https://doi.org/10.1523/JNEUROSCI.20-06-j0002.2000.

World Health Organization, 2023. Mental health of older adults. https://www.who.int/news-room/fact-sheets/detail/mental-health-of-older-adults.

Yang, X., Fiebelkorn, I.C., Jensen, O., Knight, R.T., Kastner, S., 2024. Differential neural mechanisms underlie cortical gating of visual spatial attention mediated by alpha-band oscillations. Proc. Natl. Acad. Sci. USA 121 (45), e2313304121. https://doi.org/10.1073/pnas.2313304121.