The relationship between emotional regulation and hemispheric lateralization in depression: a systematic review and a meta-analysis

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INTRODUCTION

Emotional regulation refers to the ability to manage our behavioral response in the face of challenging and stressful everyday situations. It is a complex process responsible for starting or inhibiting reactions triggered by various stimuli. However, individual levels of affective traits and structural changes in specific regions of the brain have been associated with the ability to regulate emotions [1]. The persistence of negative affect [2], differences in hemispheric brain activation and changes in functions needed for executive control are characteristics that have been verified in some psychiatric disorders [3]. However, depressive disorder (DD) and anxiety disorders (AD) share many symptoms associated with cognitive deficits and emotional regulation [4]. Both anhedonia, a striking feature in depression [5], and the impulsivity present in generalized anxiety disorder [6] seem to be a consequence of the increase in negative emotions, suggesting that the neurobiology of emotional regulation is fundamental to the psychopathology.

Patients with anxiety disorder often have depressive symptoms and vice versa [7]. The investigation of similarities and differences between depression and anxiety, points to patterns of similar neural responses, especially in the frontal region of the brain [8]. In addition, some studies reinforce that the dysregulation of pathophysiological mechanisms, such as the neurotransmitter systems norepinephrine and serotonin, interfere with the symptoms of depression and anxiety [9]. And that the elevation of secretion of the corticotrophine-releasing hormone is recurrent in these disorders [10]. Through electroencephalography, it is also possible to observe that anxious and depressive patients present cortical activity dysfunction in analogous brain regions [11]. Thus, recent studies suggest that these disorders are highly comorbid [12].

Among the brain structures that possibly participate in emotional regulation strategies, the importance of the dorsolateral prefrontal cortex (dIPFC) has been emphasized because the behavioral flexibility of individuals who suffer damage in this region is compromised evidenced by difficulties observed in performing tasks involving working memory and decision-making [13]. In addition, correlates of neural dysfunctions, such as indecision, difficulty concentrating, prostration and lack of perspective for the future, may be observed among patients with symptoms of depression and patients with left
dlPFC injury [3]. This corroborates our hypothesis that this brain region is involved in cognitive and emotional processes. In addition, electroencephalographic findings support the idea that alpha band oscillations (8–13 Hz) in the right and left dlPFC are related to these same processes [3].

In line with these findings, some studies have sought ways to measure the activity of the dlPFC in depressive conditions using EEG as a tool for data collection. Through the difference between the logarithm of the absolute spectral values in the alpha band, in the electrodes located in the frontal region of the right and left hemispheres (ln μV² RH—in μV² LH), the frontal alpha asymmetry index (FAA) is obtained, where alpha potency is inversely proportional to cortical activity and negative values suggest left hypoactivation, while positive values indicate right hypoactivation [14]. Although FAA is susceptible to variation, it has been widely disseminated as a possible biological marker in depression [15].

It has been shown that the decrease in the severity of depressive symptoms corresponds to increased dorsolateral cortical activity [16] and that cognitive ability derives from cortical activation of this same region [17]. In addition, it was observed that the power of the alpha rhythm was negatively correlated with cortical activity in the dlPFC [14]. Since all these aspects are intrinsic to the dlPFC, the objective of this study was to relate emotional regulation to hemispheric lateralization in depression and to offer a meta-analysis that verifies the existence of hemispheric lateralization attributed to the FAA. We calculated the effect size for nine studies that evaluated oscillations in the alpha band [15, 18–25], using FAA with reference to electrodes F4 and F3 and their respective standard deviations as EEG measures for the comparison of subjects who comprised the depression group versus participants in the control group.

METHODS
A systematic search was conducted in the PubMed and Cochrane databases, covering articles published from 2009 until February 2020. The search protocol was developed based on Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA), and it was previously registered in PROSPERO (CRD42020150815) [26]. To avoid publication bias, non-English language studies and those in the gray literature (for example, conference abstracts) were included. A broad but highly structured search strategy was used based on the PICOS framework. The study population was subjects with a depressive or anxiety disorder, the intervention/exposure was EEG measurements, the comparison was to a control group, the outcome was the evaluated hemispheric lateralization attributed to the FAA and the study design included any type of design.

A search for articles published from 2009 in the PubMed and Cochrane databases was performed using the following keywords: executive function OR executive control OR cognition OR prefrontal cortex OR dorsolateral prefrontal cortex OR dlPFC activity OR lobe OR cerebral cortex OR cerebral mantle OR pallium OR prefrontal lobe OR cortex OR brain imaging OR EEG OR neuroimaging OR cognitive reappraisal OR working memory OR inhibitory control OR emotional regulation OR decision making OR cognitive reevaluation AND emotion OR sadness OR prostration OR melancholy OR heartbreak OR adversity OR distress OR anguish OR bitterness OR suffering OR euphoria OR joy OR happiness OR pleasure OR satisfaction OR impulsiveness OR contentment OR behavior OR self-control OR attention OR stress OR nervousness OR frenzy OR mood OR disorder OR anxiety OR depression OR irritability AND anxiety disorder OR generalized anxiety disorder OR panic disorder OR mood disorders OR depressive disorder OR separation anxiety disorder OR agoraphobia OR phobia AND brain imaging OR neuroimaging electroencephalogram.

The following data were collected: year of publication, first author, number of participants, age, mental disorder, EEG measurements, and standard deviation. In the statistical analysis of the data, only the following variables were used: number of participants, EEG measure (international system 10–20), and standard deviation.

For the EEG measurement, we considered the frontal alpha asymmetry index (FAA), calculated from the difference between the logarithm of the absolute spectral values in the alpha rhythm, observed from the F4 and F3 electrodes, fixed to the scalp of the frontal region of the right and left hemispheres (ln μV² RH—in μV² LH) = (F4 – F3). There was no standard deviation imputation for any article. Only 1 article had the standard deviation calculated, where the equation was $Sd = (P^t/N)^{group}$ ($P$ = difference between means; $t$ = test; $N$ group = number of participants from each group).

Statistical analyses of the variables were carried out with the aid of RevMan 5, Cochrane software. Using a random effects model and observing the mean differences between the intervention and control groups, we consider the 95% confidence interval for the obtained results. The heterogeneity was attributed to the different sample sizes among the groups of participants in the selected studies.

RESULTS
A total of 7038 potential articles for initial screening were published as of 2009. Of this total, 307 duplicates were identified through the Mendeley reference manager and with manual confirmation of the titles. After the exclusion of duplicates, 6731 articles remained, of which 6686 were rejected based on the reading of titles and abstracts and 45 were selected for the full reading of the texts. Then, 27 other studies were excluded because they did not meet the eligibility criteria. Therefore, 18 articles were included in this review. Of these, 9 were homogeneous enough for meta-analysis (Fig. 1). Another 9 studies could not be statistically analyzed due to the absence of the alpha band frontal asymmetry index (FAA) measure for electrodes F4 and F3 (Fig. 2).

Articles that did not present FAA measurements related to F4 and F3 electrodes were excluded from the meta-analysis during data extraction (Table 1). To minimize the heterogeneity among the studies included in the analysis, for the studies with FAA measurements in different regions and brain waves, only the measurements referring to the alpha wave in the frontal region were collected. However, specifically from the F4 and F3 electrodes, as already mentioned earlier in the topic methodology (Table 2).

SYSTEMATIC REVIEW
All studies that were part of this literature review investigated how brainwave activity was associated with emotional regulation and human cognition. By using EEG measurements in their experiments, it was possible to verify whether brainwave oscillations would be a potential biomarker in depression and anxiety when compared to the control group [27].

Researchers have sought to understand the cause of these oscillations, especially in the frontal and prefrontal regions, in individuals with symptoms of depression [28, 29]. In anxiety disorders, significant oscillations of cortical activity have been observed in all brain regions, especially in the areas in the left hemisphere [30].

Highly concerned individuals tended to show greater changes in the beta band in multiple brain regions [31]. In depressive subjects, beta oscillations are concentrated in the central frontoparietal region, which would explain the attention deficit proportional to the severity of symptoms [32].

The studies demonstrated similar results regarding oscillations in different brain waves; however, there are specificities to
consider: Iseger [33], for example, stated that alpha band changes can serve as a potential biomarker in the treatment of depression, but only in men. In addition, the prefrontal cortex presented higher alpha power in individuals monitored only in the open-eyes condition [34]. Schizophrenic patients differed from depressive patients in delta power values, which would favor the correct diagnosis between the two disorders [29].

Although the 9 articles excluded [27 – 35] contributed significantly to our literature review, restrictions on alpha band FAA measures made it impossible to include them in our statistical analyses.

META-ANALYSIS

The studies used in the statistical synthesis investigated the existence of hemispheric lateralization of the alpha wave in depression. The FAA (F4 – F3) was calculated from the difference between the logarithms of the absolute spectral values of the alpha wave from the F4 and F3 electrodes. In the assembly of the electrodes, the Cz reference was considered, where F4 was located on the right scalp and F3 was located on the left scalp. For negative FAA, we have the value of F3 > F4, which means higher alpha power in the left front hemisphere (left alpha lateralization) and decrease of cortical activity in this region, as they are inversely proportional magnitudes. In the same way, when FAA is positive, we have that F4 > F3 and alpha power focuses on the right front hemisphere (right alpha side), with consequent reduction of cortical activity on the right [14].

Some studies included in the meta-analysis showed divergent outcomes. This was possibly due to individual characteristics of the study, such as how many participants and the age of the participants. The studies that showed the lowest numbers of participants, i.e., depression group, N = 12/control group, N = 7 in ref. [18] and depression group, N = 14/control group, N = 14 in ref. [23], did not show a relationship between frontal alpha asymmetry and depression in elderly individuals. In ref. [21], a study with a higher number of participants (depression group, N = 111; mean age = 38.5, and control group, N = 526; mean age = 35.1), the decrease in cortical activation was reflected in the patterns of FAA. Thus, alpha asymmetry is a highly variable phenomenon, and the sample size of participants influences the reliability of the results. Therefore, even if there are no statistically significant differences in FAA, further studies are encouraged [15].

Other important individual characteristics are highlighted below: Dharmadhikari [19] suggested FAA as a biological marker in depression but admitted its limitations due to a sample of nonrandomized participants. Gollan [20] observed important asymmetric differences in the alpha wave and associated them with behavioral activation. Frontal alpha asymmetry also indicated hypoactivity in the left frontal hemisphere [22], as well as an overall increase in alpha power in depressive disorders [24]. Patients with severe depression presented higher indices of frontal alpha asymmetry, even in the absence of suicidal ideation [25].

Although our meta-analysis does not demonstrate significant statistical results regarding the role of FAA as a hemispheric lateralization parameter, we observed a slight tendency toward left lateralization in the depression group (Fig. 2).

In the selected studies, there were a total of 1061 participants. Of these, 326 belonged to the depression group, and 735 belonged to the control group. A random effects meta-analysis...
Table 1. Summary of studies included in the systematic review.

| Study         | Diagnosis | Total sample (%) | Intervention group | Control group | EEG measure | Rhythm (Hz)          |
|---------------|-----------|------------------|--------------------|---------------|-------------|----------------------|
|               |           | Female | Male | Subjects | Mean age | Subjects | Mean age |           |               |             |             |
| 1 Begić et al., 2011 | Depression | 68 | 32 | 33 | 55.1 | 30 | 35.9 | ASP | Delta, Theta, Alpha, Beta |
| 2 Cook et al., 2014 | Depression | 41 | 59 | 121 | 40.6 | 47 | 37.9 | ASP/RSP | Theta |
| 3 Hinrikus et al., 2009 | Depression | 100 | 0 | 18 | 35 | 18 | 35 | SASI | Theta, Alpha, Beta |
| 4 Iseger et al., 2017 | Depression | 58 | 42 | 1008 | 38 | 336 | 37 | ASP | Theta, Alpha |
| 5 Kan et al., 2015 | Depression | 37.5 | 62.5 | 4 | 23.3 | 4 | 23.3 | ASP | Alpha |
| 6 Liu et al., 2019 | Anxiety | 55 | 45 | 40 | 19.8 | 40 | 19.5 | ASP | Beta |
| 7 Olbrich et al., 2014 | Depression | 100 | 0 | 60 | 39.4 | 60 | 37.6 | ASP | Delta, Theta, Alpha, Beta |
| 8 Li et al., 2017 | Depression | 55.5 | 45 | 33 | 32.8 | 32 | 29.5 | PSI | Delta, Theta, Alpha, Beta |
| 9 Wang et al., 2016 | Anxiety | 53 | 47 | 64 | 46.41 | 30 | 45.41 | D2 | Theta, Alpha, Beta |

ASP absolute spectral power values, RSP relative spectral power, SASI spectral asymmetry index, PSI phase synchronization index, D2 correlation dimension.

Table 2. Summary of studies included in the meta-analysis.

| Study          | Total sample (%) | Diagnosis measure | Subj. | Depression group | Control group | Alpha (Hz) | Eye |
|----------------|------------------|-------------------|-------|------------------|---------------|------------|-----|
|                | Female | Male | BDI | FAA | SD | Subj. | Mean age | FAA | SD | 8–13 | C |
| 1 Carvalho et al. (2011) | 63 | 37 | 12 | 71 | 0.05 | 0.21 | 7 | 72 | 0.003 | 0.18 | C |
| 2 Dharmadhikari et al. (2019) | 63 | 37 | 24 | 34.8 | -0.23 | 2.13 | 17 | 29.5 | 0.13 | 1.23 | C |
| 3 Gollan et al. (2014) | 62.5 | 37.5 | 37 | 35.6 | 0.43 | 0.73 | 35 | 35.6 | 0.16 | 0.27 | O/C |
| 4 Grin-Yatsenko et al. (2009) | 58 | 42 | 111 | 38.5 | -0.04 | 0.13 | 526 | 35.1 | -0.006 | 0.04 | 7.5–14 | O/C |
| 5 Jaworska et al. (2012) | 54 | 46 | 53 | 40.4 | -0.02 | 0.23 | 43 | 36.5 | 0.05 | 0.25 | 10–12 | C |
| 6 Kaiser et al. (2016) | 100 | 0 | 14 | 80.5 | 0.212 | 0.20 | 14 | 78.6 | 0.213 | 0.12 | 8–13 | C |
| 7 Kemp et al. (2010) | 60 | 40 | 15 | 39.9 | -0.02 | 0.13 | 15 | 42.4 | 0.09 | 0.17 | 8–13 | O/C |
| 8 Roh et al. (2020) | 85.5 | 14.5 | 44 | 38.3 | 0.05 | 0.23 | 60 | 34.8 | 0.03 | 0.22 | 8–13 | O/C |
| 9 Segrave et al. (2011) | 100 | 0 | 16 | 42.1 | -0.08 | 0.20 | 18 | 40.7 | -0.02 | 0.25 | 8–13 | O/C |

Reference (C2); FAA, F4–F3; C close, O open.
showed low heterogeneity ($Q_t = 11.00$, df = 8, $p = 0.20$, $I^2 = 27\%$) and an average effect of the studies equal to $-0.03$ (CI = $[-0.07$; $0.01]$). The heterogeneity, although not significant, was attributed to the different sample sizes of participants in the selected studies (Fig. 2).

**DISCUSSION**

This study evaluated the relationship between emotional regulation with hemispheric lateralization in depressive patients. In addition, cognitive deficits underlying depression were described. From a neurobiological perspective, it was found that structural and/or metabolic alterations were associated with dysfunction in cognitive-emotional processing. Recent studies corroborated these findings, stating that cortical hypoxia observed in lesions specific to the dlPFC has underlying sequelae, such as increased symptoms of depression and cognitive deficit [36].

Attributed to the prefrontal cortex, executive control refers to the human capacity to manipulate information, inhibit behaviors, change the attentional focus, and solve everyday problems, among other competencies [17]. Thus, abnormalities in the neural circuits of this brain region have important consequences on cognition [36]. Curiously, in depressive disorder, there are common clinical complaints about the decline of skills granted with hemispheric lateralization in depressive patients [13] and F7 and F8 [15, 19–22, 25]. Only the data from F4 and F3 were adopted because they are the most common electrodes in the literature for the analysis of frontal alpha asymmetry. Another limitation was the exclusion of the asymmetries related to delta, theta and beta waves [27, 28, 32, 33, 35]. Our decision to explore only the alpha wave was based on findings that correlated certain cognitive and behavioral characteristics to specific frequency bands. In fact, alpha waves have often been associated with information processing, and alpha wave oscillations have significant implications for executive control [38]. Thus, it is necessary to consider future studies that reconsider the aspects not addressed in this work.

In conclusion, the effect of our study does not confirm hemispherical lateralization in depressed individuals, it was found that emotional regulation and cognitive processes share similar neural circuits. Therefore, we encourage further research on this complex relationship, especially studies dedicated to the search for quantitative biological markers in depression. The increase in biomarker data for depression will represent a concrete and tangible tool, providing greater accuracy in the diagnosis of depressive disorder and ensuring patients an early and adequate treatment.

**REFERENCES**

1. Mocaíber I, Oliveira L, Pereira M, Pinheiro W, Ventura P, Figueira I et al. Neurobiologia da regulação emocional: implicações para a terapia cognitivo-comportamental. Psicol Estud. 2008;13:531–8. https://doi.org/10.1590/S1413-73722008000300014.
2. Davis E, Folland-Ross L, Gotlib I. Neural correlates of top-down regulation and generation of negative affect in major depressive disorder. Psychiatry Res: Neuroimaging. 2018. https://doi.org/10.1016/j.pscychresns.2018.04.001.
3. Henriques J, Davidson R. Left frontal hypoactivity in depression. J Abnorm Psychol. 1991;100:535–45. https://doi.org/10.1037/0021-843X.100.4.535.
4. Jenkins L, Stange J, Bessette K, Chang Y, Corwin S, Skerritt K et al. Differential engagement of cognitive control regions and subgenual cingulate based upon presence or absence of comorbid anxiety with depression. J Affect Disord. 2016. https://doi.org/10.1016/j.jad.2015.10.013.
5. Heller A, Johnstone T, Shackman A, Light S, Peterson M, Kolden G et al. Reduced capacity to sustain positive emotion in major depression reflects diminished maintenance of fronto-striatal brain activation. PNAS. 2009;106:22445–50. https://doi.org/10.1073/pnas.0910651106.
6. Ferreira-Garcia R, Faria CGF, Nardi AE, Freire RC. Negative affect mediates impulsivity in generalized anxiety disorder psychopathology. Psychopathology. 2019;52:327–33.
7. Crane NA, Jenkins LM, Dion C, Meyers KK, Weldon AL, Gabriel LB et al. Comorbid anxiety increases cognitive control activation in major depressive disorder. Depress Anxiety. 2016;33:967–77. https://doi.org/10.1002/da.22541.
8. Fava M, Rush AJ, Alpert JE, Carmin CN, Balasubramani GK, Winiweter SR et al. What clinical and symptom features and comorbid disorders characterize outpatient with anxious major depressive disorder: a replication and extension. Can J Psychiatry. 2006;51:823–35. https://doi.org/10.1177/070674370605101304.
9. Ressler KJ, Nemeroff CB. Role of serotoninergic and noradrenergic systems in the pathophiology of depression and anxiety disorders. Depress anxiety. 2000;12:2–19. https://doi.org/10.1002/1520-6394(200012)1:2.1.50.
10. Holboer F, Izing M. Central CRH system in depression and anxiety—evidence from clinical studies with CRH1 receptor antagonists. Eur J Pharmacol. 2008;583:350–7. https://doi.org/10.1016/j.ejphar.2007.12.032.
11. Heller AS, Johnstone T, Peterson MJ, Kolden GG, Kalin NH, Davidson RJ. Increased prefrontal cortex activity during negative emotion regulation as a predictor of depression symptom severity trajectory over 6 months. JAMA Psychiatry. 2013;70:1181–9. https://doi.org/10.1001/jamapsychiatry.2013.23430.
12. Hamilton JP, Chen MC, Waugh CE, Jooreman J, Gotlib IH. Distinctive and common neural underpinnings of major depression, social anxiety, and their comorbidity. Soc Cogn Affect Neurosci. 2015;10:552–60. https://doi.org/10.1093/scan/nsu084.
13. Sotres-Bayon F, Cain CK, LeDoux JE. Brain mechanisms of fear extinction: historical perspectives on the contribution of prefrontal cortex. Biol Psychiatry. 2000;66:329–36. https://doi.org/10.1001/jbiopsy.2005.10.012.
14. Allen JJ, Urry HL, Hitt SK, Coan JA. The stability of resting frontal electroencephalographic asymmetry in depression. Psychophysiology. 2004;41:269–80. https://doi.org/10.1111/j.1469-8986.2003.00149.x.

15. Segrave R, Cooper N, Thomson R, Croft R, Sheppard D, Fitzgerald P. Individualized Alpha activity and frontal asymmetry in major depression. Clin EEG Neurosci. 2011;42:45–52. https://doi.org/10.1177/155005941104200110.

16. Cheon EJ, Koo BH, Choi JH. The efficacy of neurofeedback in patients with major depressive disorder: an open labeled prospective study. Appl Psychophysiol Biofeedback. 2016;41:103–10. https://doi.org/10.1007/s10848-015-9315-8.

17. Miller E, Cohen J. An integrative theory of prefrontal cortex function. Rev Neurosci 2001;24:167–202. https://doi.org/10.1146/annurev.neuro.24.1.167.

18. Carvalho A, Moraes H, Silveira H, Ribeiro P, Piedad R, Deslandes A, et al. EEG frontal asymmetry in the depressed and remitted elderly: is it related to the trait or to the state of depression? J Affect Disord. 2011;129:143–8. https://doi.org/10.1016/j.jad.2010.08.023.

19. Dharmadhikari AS, Jaiswal SV, Tandle AL, Sinha D, Jog N. Study of frontal alpha asymmetry in mild depression: a potential biomarker or not? J Neurosci Rural Pract. 2019;10:250–55. https://doi.org/10.4103/jnpr.jnpr_293_18.

20. Gollan J, Hoxda D, Chihade D, Pfieger M, Rosebrock L, Cacioppo J. Frontal alpha EEG asymmetry before and after behavioral activation treatment for depression. Biol Psychol. 2014. https://doi.org/10.1016/j.biopsycho.2014.03.003.

21. Grin-Yatsenko V, Baas I, Ponomarev V, Kropotov J. EEG power spectra at early stages of depressive disorders. J Clin Neurophysiol. 2009;26:401–6. https://doi.org/10.1097/WNP.0b013e3181c298fe.

22. Jaworska N, Blier P, Fusee W, Knott V. Alpha power, alpha asymmetry and anterior electroencephalographic spectral asymmetry index for detection of depression. Med Princ Pract. 2019;10:250–55. https://doi.org/10.4103/jnpr.jnpr_293_18.

23. Kaiser A, Doppelmayr M, Igleseder B. Electroencephalogram alpha asymmetry in geriatric depression valid or vanished? Z Gerontol Geriat. 2016;51. https://doi.org/10.1111/j.1469-8986.2003.00149.x.

24. Miller E, Cohen J. An integrative theory of prefrontal cortex function. Rev Neuropsychiatry 2001;24:167–202. https://doi.org/10.1146/annurev.neuro.24.1.167.

25. Roh S, Kim J, Kim S, Kim Y, Lee S. Frontal alpha asymmetry and anterior electroencephalographic asymmetry in depression. J Nerv Ment Dis. 2010;198:55–61. https://doi.org/10.1097/NMD.0b013e3181c298fe.

26. Carvalho A, Moraes H, Silveira H, Ribeiro P, Piedade R, Deslandes A, et al. EEG frontal asymmetry in the depressed and remitted elderly: is it related to the trait or to the state of depression? J Affect Disord. 2011;129:143–8. https://doi.org/10.1016/j.jad.2010.08.023.

27. Dharmadhikari AS, Jaiswal SV, Tandle AL, Sinha D, Jog N. Study of frontal alpha asymmetry in mild depression: a potential biomarker or not? J Neurosci Rural Pract. 2019;10:250–55. https://doi.org/10.4103/jnpr.jnpr_293_18.

28. Gollan J, Hoxda D, Chihade D, Pfieger M, Rosebrock L, Cacioppo J. Frontal alpha EEG asymmetry before and after behavioral activation treatment for depression. Biol Psychol. 2014. https://doi.org/10.1016/j.biopsycho.2014.03.003.

29. Grin-Yatsenko V, Baas I, Ponomarev V, Kropotov J. EEG power spectra at early stages of depressive disorders. J Clin Neurophysiol. 2009;26:401–6. https://doi.org/10.1097/WNP.0b013e3181c298fe.

30. Jaworska N, Blier P, Fusee W, Knott V. Alpha power, alpha asymmetry and anterior electroencephalographic spectral asymmetry index for detection of depression. Med Princ Pract. 2019;10:250–55. https://doi.org/10.4103/jnpr.jnpr_293_18.

31. Kaiser A, Doppelmayr M, Igleseder B. Electroencephalogram alpha asymmetry in geriatric depression valid or vanished? Z Gerontol Geriat. 2016;51. https://doi.org/10.1111/j.1469-8986.2003.00149.x.

32. Li Y, Kang C, Wei Z, Gu X, Liu T, Zhou Y, et al. Beta oscillations in major depression —signalling a new cortical circuit for central executive function. Sci Rep. 2017;7:18021. https://doi.org/10.1038/s41598-017-18306-w.

33. Iegeyer T, Korgaonkarc M, Kenemansja J, Grieve S, Bakenenf C, Fitzgerald PG, et al. EEG connectivity between the subgenual anterior cingulate and prefrontal cortex in response to antidepressant medication. Eur Neuropsychopharmacol. 2017;27:301–12. https://doi.org/10.1016/j.euroneuro.2017.02.002.

34. Kan D, Lee P. Decrease alpha waves in depression: an electroencephalogram (EEG) study. In BioSignal Analysis Processing and Systems (ICBAPS), 2015 International Conference on, IEEE (2015), p. 156–161. https://doi.org/10.1109/ICBAPS.2015.7292237.

35. Begic D, Popovic-Knapic V, Grubisin J, Kosanovic-Rajacic B, Filipi6 C, Telerovic I, et al. Quantitative electroencephalography in schizophrenia and depression. Psychiatr Danubina. 2011;23:355–62.

36. Koenigs M, Huey E, Calamia M, Raymond V, Tran D, Grafman J. Distinct regions of prefrontal cortex mediate resistance and vulnerability to depression. J Neurosci. 2008;28:12341–8. https://doi.org/10.1523/jneurosci.2324-08.2008.

37. Lucass F, Braga O, Fonseca C, Frochtgarten L. Recomendações para o registro e interpretação do mapeamento topográfico do eletronecefalografo (EEG) e potenciais evocados sensoriais (PES). Braz J Epilepsy Clin Neurophysiol 1996;2:82–75. https://doi.org/10.1590/S0004-262X1999000100026.

38. Nenert R, Viswanathan S, Dubuc D, Visscher K. Modulations of ongoing alpha oscillations predict successful short-term visual memory encoding. Front Hum Neurosci. 2012;7:1–10. https://doi.org/10.3389/fnhum.2012.00127.

AUTHOR CONTRIBUTIONS
NH and LAQ designed the study. NH searched the databases, NH and LAQ made the extractions and AEN critically reviewed the manuscript.

COMPETING INTERESTS
The authors declare no competing interests.

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