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## Functional role of frontal electroencephalogram alpha asymmetry in the resting state in patients with depression: A review

Yu-Hong Xie, Ye-Min Zhang, Fan-Fan Fan, Xi-Yan Song, Lei Liu

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### Abstract

Depression is a psychological disorder that affects the general public worldwide. It is particularly important to make an objective and accurate diagnosis of depression, and the measurement methods of brain activity have gradually received increasing attention. Resting electroencephalogram (EEG) alpha asymmetry in patients with depression shows changes in activation of the alpha frequency band of the left and right frontal cortices. In this paper, we review the findings of the relationship between frontal EEG alpha asymmetry in the resting state and depression. Based on worldwide studies, we found the following: (1) Compared with individuals without depression, those with depression showed greater right frontal EEG alpha asymmetry in the resting state. However, the pattern of frontal EEG alpha asymmetry in the resting state in depressive individuals seemed to disappear with age; (2) Compared with individuals without maternal depression, those with maternal depression showed greater right frontal EEG alpha asymmetry in the resting state, which indicated that genetic or experience-based influences have an impact on frontal EEG alpha asymmetry at rest; and (3) Frontal EEG alpha asymmetry in the resting state was stable, and little or no change occurred after antidepressant treatment. Finally, we concluded that the contrasting results may be due to differences in methodology, clinical characteristics, and participant characteristics.

**Key Words:** Depression; Frontal electroencephalogram alpha asymmetry; Frontal asymmetry; Resting state; Neurological indicator

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**Core Tip:** Researchers have paid more attention to the functional role of frontal electroencephalogram alpha asymmetry (FAA) in the resting state in individuals with depression. In this paper, we review the findings of the relationship between FAA in the resting state and depression. Individuals with clinical depression showed greater right FAA in the resting state. The pattern of FAA in the resting state in individuals with clinical depression seemed to disappear with age. Individuals with maternal depression showed greater right FAA in the resting state. There was little or no change in FAA in the resting state after antidepressant treatment.

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## INTRODUCTION

Depression is a leading cause of disability worldwide and contributes greatly to the global burden of disease. It is characterized by persistent sadness and a lack of interest or pleasure in previously rewarding or enjoyable activities, affecting daily life and even suicide in extreme cases[1]. Currently, depression affects more than 350 million people worldwide, and the growth rate of patients with depression has been approximately 18% in the past decade[1]. There are currently 95 million people suffering from depression in China, and approximately 280000 people commit suicide each year, 40% of whom suffer from depression[2]. The diagnosis rate of depression among adolescents in 2020 was 24.6%; the proportion of major depression was 7.4%[3]. The diagnosis of depression is usually carried out using clinical interviews conducted around the diagnostic classification system, such as the 11th edition of the International Classification of Diseases (ICD-11) and the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V). These diagnostic criteria are usually based on oral reports from patients or their families and direct observations by clinicians because each disease type has its own symptoms, including behavioural, cognitive, emotional, or physical disorders. However, these diagnostic methods incorporate a “yes or no” approach in the diagnosis of depression, and self-reporting and clinical observation methods are highly subjective, which leads to errors in the diagnosis of depression. Therefore, there is currently a lack of objective examination methods for depression.

Objective measurement of depression has always been a focus of researchers, and the measurement methods of brain activity have gradually received increasing attention[4]. Among these methods, frontal electroencephalogram (EEG) alpha asymmetry is a promising measurement method[5]. In the past 30 years, research on the relationship between frontal EEG alpha asymmetry and mood, personality, and neuropsychological diseases has developed rapidly. There are two types of frontal EEG alpha asymmetry: frontal EEG alpha asymmetry in the resting state and frontal EEG alpha asymmetry during tasking conditions with emotional challenges[6]. The frontal EEG alpha asymmetry at rest is associated with various trait-like individual differences. It is also called trait frontal EEG alpha asymmetry. Frontal EEG alpha asymmetry in tasking conditions is related to the operation that is designed to affect the individual's emotional state and is labelled state frontal EEG alpha asymmetry[7]. According to previous studies, researchers have paid more attention to the functional role of frontal EEG alpha asymmetry in the resting state in individuals with depression[8].

Frontal EEG alpha asymmetry at rest shows differences in activation of the alpha frequency band of the left and right frontal cortices[9,10]. Related studies have shown that there is an inverse relationship between the activity of the alpha wave and the activity of the cerebral cortex. For example, research has shown that when the underlying cortex system is active, the alpha wave tends to decrease[11,12]. Frontal EEG alpha asymmetries are usually calculated by subtracting the EEG power in the right frontal cortices from the EEG power in the left frontal cortices. However, different researchers may use different methods of calculation. The first equation uses the channel "F4" and "F3" to refer to the levels of alpha power on the right and left frontal areas of the scalp, respectively, around the F4 and F3 positions on the 10-20 electrode placement system. These equations are used to compute frontal alpha asymmetry (FAA) by determining the difference or ratio between alpha power values at F3 and F4. There are two commonly used equations to calculate FAA in the literature. The majority of developmental studies employ the difference between the natural logarithm (ln) of absolute power at F4 and F3, which is expressed as  $\ln(F4) - \ln(F3)$ [13]. The second equation commonly used for computing frontal alpha asymmetry (FAA) involves taking the ratio of the difference between the alpha power levels in the left and right frontal hemispheres to their sum, expressed as  $(F4 - F3)/(F3 + F4)$ . This approach is believed to normalize the difference value[14]. A less common third method is to log-transform the ratio, yielding  $[\ln(F4) - \ln(F3)] / [\ln(F3) + \ln(F4)]$ [15]. Another approach to FAA calculation involves using relative frontal alpha power, which is determined by computing the percentage of alpha band power relative to

the total power across all frequency bands[16]. Relative power may have advantages over absolute power in evaluating paediatric populations due to its improved test-retest reliability[17] and ability to detect changes in the frequency composition of EEGs during development[18]. Recently, Harrewijn *et al* [19] introduced a fourth method to calculate FAA, which involves computing the difference between the natural logarithm of the relative power in both hemispheres, expressed as  $\ln[\text{rel}(F4)] - \ln[\text{rel}(F3)]$ .

To explore whether frontal EEG alpha asymmetry in the resting state is a reliable and useful index for understanding depression, this article reviews the research on the relationship between frontal EEG alpha asymmetry in the resting state and depression. First, we reviewed the pattern of frontal EEG alpha asymmetry in the resting state of clinically depressed individuals. Second, we reviewed the pattern of frontal EEG alpha asymmetry in the resting state of children inherited from generation to generation (parents suffering from depression). Third, we discussed whether frontal EEG alpha asymmetry in the resting state can be used as an effective indicator of depression intervention. Finally, we summarized the reasons for the inconsistent results.

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## CLINICAL RESEARCH

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**Table 1** provides the comparison of methods in studies on the pattern of frontal EEG alpha asymmetry in the resting state of clinically depressed individuals. Many studies have investigated the pattern of frontal EEG alpha asymmetry in the resting state in depressed individuals compared with nondepressed individuals. For example, Henriques and Davidson revealed that participants who have depression at early ages have greater right frontal lobe activation than nondepressed participants[20]. Furthermore, many studies have found that there was less left frontal lobe activation in participants with depression [21-34], in previously depressed subjects[28], and in a sample of individuals with a history of childhood-onset depression compared to that in healthy controls[35]. However, there were also some contradictory results over the years. For example, asymmetry differences were not found between depressed individuals and nondepressed individuals[36-38]. In addition, Quinn *et al*[39] examined the pattern of frontal EEG alpha asymmetry in the resting state of depressed patients with nonmelancholia, depressed patients with melancholia and control participants. The results found that compared with depressed patients with melancholia and healthy participants, depressed patients with nonmelancholia showed larger left frontal lobe activation. Smith *et al*[32] examined the frontal EEG alpha asymmetry patterns in patients with lifetime depression, current depression, and healthy controls in the resting state. The results indicated that reduced relative activity in the left frontal brain region may be associated with an increased risk of major depressive disorder (MDD). These findings extend previous research by demonstrating that the sources of surface asymmetry associated with a history of depression are likely linked to asymmetry in the dorsal-lateral frontal regions of the brain. Furthermore, decreased motivation for activating motor scripts in the premotor regions and the precentral gyrus may be pertinent to depression, and decreased left frontal brain activity can predict a lifetime history of depression[33].

Furthermore, some studies have examined whether frontal EEG alpha asymmetry in the resting state is a stable measurement index in depressed individuals. Allen *et al*[11] investigated the short-term stability of frontal EEG alpha asymmetry in the resting state in female patients with depression. The results showed that in an 8-wk and 16-wk interval, the score of frontal EEG alpha asymmetry at resting state had high internal consistency and stability, and changes in asymmetry scores were not related to changes in clinical depression. Moreover, Vuga *et al*[40] compared the long-term stability (1 to 3 years) of frontal EEG alpha asymmetry at rest between depression patients and healthy individuals. The results demonstrated that for depression patients and healthy individuals, frontal EEG alpha asymmetry at rest was moderately stable, in which gender, the history of individual depression, the severity of depression characteristics in the post-test, and the degree of change in the severity of depression were not related to the stability of frontal EEG alpha asymmetry. In addition, Gold *et al*[41] aimed to investigate the extent to which frontal EEG alpha asymmetry at rest was an effective and reliable neurological indicator for diagnosing the severity of depression in adults. The results showed that in a 3-month interval, the correlation between the scores of frontal EEG alpha asymmetry in the resting state and psychiatric examination was mostly small and not statistically significant. Similarly, McFarland *et al*[42] also found that frontal EEG alpha asymmetry in the resting state did not predict the course of depression at six months. Therefore, these results concluded that frontal EEG alpha asymmetry in the resting state was stable over time, and its potential as a predictive biomarker for depressive symptoms remains unclear.

Most people with depression suffer from depression in childhood[43]. The study of frontal EEG alpha asymmetry in the resting state in children with major depression may reveal the biological relevance of the early development of the underlying chronic disease. Kentgen *et al*[44] investigated frontal EEG alpha asymmetry at rest in adolescents with depression compared to developing controls. The results showed that there was no significant difference in frontal EEG alpha asymmetry in the resting state between adolescents with depression and developing controls. Feldmann *et al*[45] aimed to extend previous findings and assess frontal EEG alpha asymmetry in the resting state in a major depressive

**Table 1** The comparison of methods in studies on asymmetry pattern at resting state of clinically depressed individuals

Ref.	Sample		Age (yr)		% female	Diagnosis of depression	EEG detail			
	Experimental group	Control group	Experimental group	Control group			Reference montage	EO/EC	Recording length (min)	Alpha range (Hz)
Henriques <i>et al</i> [20], 1991	EG: 15 depressed subjects	CG: 13 healthy controls	M = 40.40 (depressed subjects)	M = 40.61 (HCs)	60.7	SADS	CZ; AE; AR	E0+EC	1	8-13
Baehr <i>et al</i> [21], 1998	EG: 13 MDD	CG: 11 healthy controls	M = 43.50 (MDD)	M = 44.20 (HCs)		DSM-IV; BDI	Cz	EC	5	8-13
Beeney <i>et al</i> [22], 2014	EG: 13 MDD	CG: 21 healthy controls	M = 32.12 (MDD)	M = 27.78 (HCs)	100	DSM-IV; SCID	AL	E0+EC	8	8-13
Bruder <i>et al</i> [23], 1997	EG: 44 MDD (19 with and 25 without an anxiety disorder)	CG: 26 healthy controls	MDD with an anxiety disorder: M = 36.70; MDD without an anxiety disorder: M = 41.30	M = 32.90 (HCs)	50	DSM-III-R	NR	E0+EC	6	7.8-12.5
Cantisani <i>et al</i> [24], 2016	EG: 20 depressed subjects	CG: 19 healthy controls	M = 43.30 (depressed subjects)	M = 41.05 (HCs)	53.8	SCID; DSM-IV-TR	CA		6	8-12.5
Dharmadhikari <i>et al</i> [25], 2019	EG: 24 MDD	CG: 17 healthy controls	M = 34.82 (MDD)	M = 29.52 (HCs)	63.4	DSM-IV	auricle	EC	10	8-13
Gordon <i>et al</i> [26], 2010	EG: 92 MDD	CG: 1908 healthy controls			58.7	DSM-IV	AR	E0+EC	4	8-13
Gotlib[27], 1998	EG: 16 currently depressed, 31 previously depressed	CG: 30 never depressed				SCID	Cz	E0+EC	8	8-13
Jaworska <i>et al</i> [28], 2012	EG: 53 MDD	CG: 43 healthy controls				DSM-IV-TR; SCID-IV-I/P; HAM-D; MADRS	AM; CZ; AR	E0+EC	6	8-13
Kemp <i>et al</i> [29], 2010	EG: 15 MDD	CG: 15 healthy controls	M = 39.90 (MDD)	M = 42.40 (HCs)	60	MINI; DASS	AM	EC	2	8-13
Koo <i>et al</i> [30], 2019	EG: 20 MDD	CG: 20 healthy controls	M = 51.05 (MDD)	M = 47.15 (HCs)		ICD-10/DSM IV	AE	EC	10	8-13
Roh <i>et al</i> [31], 2020	EG: 44 MDD without Suicidal ideation, 23 MDD with suicidal ideation	CG: 60 healthy controls	M = 39.30 (MDD); M = 37.48 (MDD with SI)	M = 34.83 (HCs)	85	DSM-IV	ECC	EO	3	8-12
Stewart <i>et al</i> [32], 2010	EG: 143 MDD	CG: 163 healthy controls	M = 19.10 (MDD)		69	DSM-IV	CSD; Cz; LM	E0+EC	8	8-13
Cai <i>et al</i> [34], 2020	EG: 24 MDD	CG: 22 healthy controls				DSM-IV; MINI	Cz	EC	5	8-14
Nusslock <i>et al</i> [35], 2018	EG: 37 depression, 18 anxiety + depression	CG: 69 healthy controls				SCID	AM	E0+EC	6	8-13
Brzezicka <i>et al</i> [36], 2017	EG: 26 MDD	CG: 26 healthy controls	M = 28.00 (MDD)	M = 24.90 (HCs)			CSD	EC	5	8-13

Jang <i>et al</i> [37], 2020	EG: 20 MDD, 18 patients with schizophrenia	CG: 16 healthy controls	M = 42.60 (MDD); M = 32.00 (MDD with schizophrenia)	M = 37.75 (HCs)	48.1	DSM-IV; MINI	BM	E0+EC	5	8-12
Segrave <i>et al</i> [38], 2011	EG: 16 MDD	CG: 18 healthy controls	M = 40.75 (MDD)	M = 42.11 (HCs)	100	DSM-IV	Cz; CA	E0+EC	6	8-13
Quinn <i>et al</i> [39], 2014	EG: 117 MDD	CG: 120 healthy controls				MINI; DSM-IV	AR; ER	EC	2	8-13
Smith <i>et al</i> [99], 2018	EG:143 lifetime MDD, 62 current MDD	CG: 163 healthy control			69	BDI; SCID	Cz	E0+EC	8	8-13
Vuga <i>et al</i> [40], 2006	EG: 49 childhood onset MDD	CG: 50 healthy controls	19-34	19-39	66.7	DSM-IV	Cz	E0+EC	6	7.5-12.5; alpha 1 (7.5-10.5); alpha 2 (10.5-12.5)
Gold <i>et al</i> [41], 2013	EG: 79 adults with depression		M= 35.60		78.5	DSM-III-R; SCID; MADRS	ALM	EC	5	8-12
McFarland <i>et al</i> [42], 2006	EG: 67 MDD		M = 34.64		65.7	SCID	LE	E0+EC	6	8-13
Kentgen <i>et al</i> [44], 2000	EG: 25 right-handed female outpatients	CG: 10 healthy controls			100	DSM-IV	NR	E0+EC	6	7.8-12.5
Feldmann <i>et al</i> [45], 2018	EG: 16 adolescents with depression	CG: 34 healthy controls	M = 16.08	M = 15.67	72.6	ICD-10; BDI	Cz; Mastoids; AE	E0+EC	8	7.5-13
Grünewald <i>et al</i> [46], 2018	EG: 20 adolescents (12-17 yr) with unipolar depression (12 with first episode, 8 with recurrent depression)	CG: 31 healthy controls	M = 14.85 (unipolar depression)	M = 14.16 (HCs)	60.8	DSM-IV; ICD-10	AR		5	7.5-13.5
Deslandes <i>et al</i> [89], 2008	EG: 22 depressed subjects	CG: 14 healthy controls	M = 71.60 (depressed subjects)	M = 72.40 (HCs)	94	DSM-IV	ER	E0+EC	8	8-13
Kaiser <i>et al</i> [50], 2018	EG: 14 depression, 11 anxiety + depression	CG: 14 healthy controls	M = 78.60 (anxiety + depression); m = 80.50 (depression)	M = 80.90 (HCs)	100	GDS; HADS	RLMB	E0+EC	4	6.9-12.9
Carvalho <i>et al</i> [51], 2011	EG: 12 depressed subjects, 8 remitted subjects	CG: 7 non-depressed elderly subjects			66.7	DSM-IV	Earlobes	EC	8	8-12.9

EC: Eyes closed; EO: Eyes open; EEG: Electroencephalogram; HCs: Healthy controls; MDD: Major depressive disorder.

Diagnosis of depression: BDI: Beck Depression Inventory; DASS: Depression, Anxiety and Stress Scales; DSM-III-R: Diagnostic and Statistical Manual of Mental Disorders, Third edition, Revised; DSM-IV: Diagnostic and Statistical Manual of Mental Disorders, Fourth edition; GDS: Geriatric depression scale; HAMD: Hamilton Rating Scale for Depression; HADS: Hospital anxiety and depression scale-depression; ICD-11: 11<sup>th</sup> edition of the International Classification of Diseases; MADRS: Montgomery and Asberg Depression Rating Scale; MINI: Mini International Neuropsychiatric Interview; SADS: The Schedule for Affective Disorders and Schizophrenia; SCID: Structured Clinical Interview for DSM III-R; SCID-IV-I/P: Structured Clinical Interview for DSM-IV, Axis I, Patient Version.

Reference montage: AE: The average across all 31 electrodes; AL: The average of all EEG leads; ALM: Averaged linked mastoids; AM: Averaged mastoids; AR: Average reference; BM: Both mastoids; CA: Common average montage; CA: Common average; CSD: Current source densities; ECC: The reference electrode was predefined; ER: Ear reference; LE: Linked ear; LM: Linked mastoids; NR: Nose reference; RLMB: The right and left mastoid bone.

adolescent sample while considering possible extraneous variables, such as comorbid anxiety and disease state. They repeated the results of Kentgen *et al*[44] and found that there was no significant difference between major depressive adolescents without comorbid anxiety disorder and healthy

controls. However, major depressive adolescents with comorbid anxiety disorder demonstrated greater right frontal lobe activation than healthy controls. The results show that frontal EEG alpha asymmetry in the resting state itself has nothing to do with adolescent depression and emphasize the importance of considering comorbid anxiety disorder when examining adolescents' asymmetry patterns[45]. In addition, Grunewald *et al*[46] analysed frontal EEG alpha asymmetry at rest in adolescents with depression and healthy controls. They found that adolescents with depression had less left-sided frontal alpha power, while healthy controls exhibited no asymmetry. For healthy controls, more left frontal alpha was associated with a higher depression score, which was not observed in adolescents with depression.

The diagnosis of depression in the elderly is often more difficult because the symptoms of depression may be confused by factors such as the individual's own physical condition[47]. In addition, depression in the elderly is connected with cognitive deficits and physical disability, which increases the difficulty of distinguishing depression from dementia[48]. At present, many studies have explored the relationship between frontal EEG alpha asymmetry in the resting state and depression in young people, but few studies have focused on frontal EEG alpha asymmetry in the resting state in elderly individuals with depression. Recently, Deslandes *et al*[49] found that depressed elderly patients showed relatively greater right frontal activity than healthy elderly patients; however, the difference was not significant. This was consistent with the results of Kaiser *et al*[50], who found that there was no significant difference in frontal EEG alpha asymmetry in the resting state between depressed elderly individuals and healthy elderly individuals. Carvalho and Hopko analysed frontal EEG alpha asymmetry in the resting state in depressed, remitted and nondepressed elderly subjects. There was no difference in frontal EEG alpha asymmetry at rest among the groups. Moreover, the results showed no evidence of a relationship between frontal EEG alpha asymmetry in the resting state, quality of life and depression in the elderly[51]. Therefore, frontal EEG alpha asymmetry at rest seemed to disappear with age. Future research on frontal EEG alpha asymmetry in the resting state should consider the influences of age.

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## INTERGENERATIONAL INHERITANCE RESEARCH

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Table 2 provides the comparison of methods in studies on the inherited pattern of frontal EEG alpha asymmetry at rest in children over generations. Research has shown that the offspring of parents with depression are at increased risk of depression[52,53]. Considering that there was a close relationship between frontal EEG alpha asymmetry in the resting state and depression, frontal EEG alpha asymmetry may be affected by the early social environment, such as maternal depression. To date, extensive literature has exploited the relationship between maternal depression and frontal EEG alpha asymmetry in the resting state in infants and young children. A number of studies have found that compared with nondepressed mothers, infants of depressed mothers have greater right frontal EEG alpha asymmetry[54-59], and similar results were also found in the youth group[60,61]. In a meta-analysis study, Peltola *et al*[62] found that frontal EEG alpha asymmetry at rest is affected by psychosocial risk factors, such as child abuse or parental depression, which is manifested by greater activation of the right frontal cortex, with a significant effect size. Moreover, their results showed that the relationship between parental depressive symptoms and greater right frontal lobe activity was moderated by gender, in which girls were more affected by psychosocial risk factors than boys. Additionally, the effects of this long-term exposure to parental depression diminish with age.

Furthermore, in an effort to understand the earliest origin of frontal EEG alpha asymmetry in the resting state, which was considered to reflect a vulnerability to depression, some studies examined their consistency and their association with the mother's prenatal and postnatal depression symptoms. Goodman *et al*[63] demonstrated that the mother's prenatal depression symptom (nonpostpartum or concurrent) levels are related to the baby's right frontal EEG alpha asymmetry. This was consistent with the results of Wen *et al*[64]. They found that in a subsample in which the infant spent at least 50% of his or her daytime hours with his or her mother, a higher mother's postpartum depression level and lower maternal sensitivity predicted the baby's greater relative right frontal EEG asymmetry[63]. In addition, Lusby *et al*[65] found that mothers' prenatal and postpartum depression symptoms can predict the frontal EEG asymmetry scores of 3-month-old and 6-month-old infants. However, Goldstein *et al*[66] assessed frontal EEG alpha asymmetry twice, at ages 3 and 6, in never-depressed children. The study revealed that offspring of depressed mothers displayed a decline in relative activity in the left frontal alpha region during early childhood, while offspring of non-depressed mothers showed relatively consistent and symmetrical levels of frontal alpha activity during both assessments[66]. Although most studies have found that maternal depression has an important influence on determining the direction and degree of frontal EEG alpha asymmetry in the resting state in children[56,59,67], others have found no significant differences between offspring of nondepressed mothers and depressed mothers[68,69]. Future research needs to investigate the genetic mechanism that connects psychosocial risk and frontal EEG alpha asymmetry at rest. That is, whether genetic or experience-based influence has an impact on frontal EEG alpha asymmetry at resting state. In addition, as discussed above, a large number of studies have observed abnormal patterns of frontal EEG alpha asymmetry in the resting state in newborns of

**Table 2** The comparison of methods in studies on asymmetry pattern at resting state of children inherited from generation to generation

Ref.	Sample		Age (yr)		% female	Diagnosis of depression	EEG detail			
	Experimental group	Control group	Experimental group	Control group			Reference montage	EO/EC	Recording length (min)	Alpha range (Hz)
Dawson <i>et al</i> [54], 1997	EG: 117 mothers and their 13-15-mo-old infants		13.74		44.4	DSM-IIIIR; SCID; CES-D	ME		1	6-9
Diego <i>et al</i> [56], 2006	EG: 38 depressed	CG: 28 non-depressed	3-6 mo			CES-D	Cz			3-13
Field <i>et al</i> [57], 1995	EG: 17 depressed adolescent mothers	CG: 15 non-depressed mothers	3-6 mo		50	DISC; BDI	Cz		3	3-12
Jones <i>et al</i> [58], 1997	EG: 20 depressed group	CG: 24 non-depressed group	M = 18.00	M = 18.70		CES-D; SCID	Cz		3	2-6
Jones <i>et al</i> [59], 1998	EG: 35 mothers with depressive symptom	CG: 28 non-depressive symptom	M = 38.80	M = 39.50	48	CES-D	Cz		3	8.5-125
Tomarken <i>et al</i> [60], 2004	EG: 23 high risk	CG: 13 low risk	M = 13.10	M = 13.00	52.6	SCID	Cz	E0+EC	8	8.5-12.5
Lopez-Duran <i>et al</i> [61], 2012	EG: 90 high risk	CG: 45 low risk	M = 7.36	M = 7.93	46.6	DSM-IV; SCID	CA	E0+EC	3	7.5-11.5
Goodman <i>et al</i> [63], 2020	EG: 136 total infants.		M = 12 mo			DSM-IV	ARC		3	6-9
Wen <i>et al</i> [64], 2017	EG: 111 infants		6 mo of age (± 2 wk)			EPDS	Cz		2	6-9
Lusby <i>et al</i> [65], 2014	EG: 83 mother-infant dyads participated		3 mo and 6 mo only			DSM-IV	ARC		3	6-9
Dawson <i>et al</i> [69], 1992	EG: 31 infants		M = 14.21 (mo)		59.1	CES-D	Cz		1	6-9
Bruder <i>et al</i> [68], 2007	EG: 19 both parent and grandparent having MDD; 14 either parent or grandparent having MDD	CG: 16 neither having MDD	M = 15.40; M = 10.60	M = 13.60	53		LE	E0+EC	2	7-12.5

EC: Eyes closed; EO: Eyes open; EEG: Electroencephalogram; HCs: Healthy controls; MDD: Major depressive disorder. Diagnosis of depression: BDI: Beck Depression Inventory; CES-D: Center for Epidemiological Studies-Depression Scale; DSM-IIIIR: Diagnostic and Statistical Manual of Mental Disorders, Third edition, Revised; DSM-IV: 4<sup>th</sup> edition of the Diagnostic and Statistical Manual of Mental Disorder; EPDS: Edinburgh Postnatal Depression Scale; SCID: Structured Clinical Interview for DSM IIIIR. Reference montage: ARC: Average reference configuration; AS: Average signal; CA: Common average; Cz: The midline central position; LE: Linked ears reference; ME: Mastoid electrodes; NR: Nose reference.

depressed mothers, indicating a genetic disposition to greater right-sided asymmetry across cross-sectional assessment. Future research can focus on longitudinal investigations to examine whether there are long-term and lasting changes in the psychosocial risks faced by children.

## FRONTAL EEG ALPHA ASYMMETRY IN THE RESTING STATE AND ANTIDEPRESSANT TREATMENT

Table 3 provides the comparison of methods in studies on frontal EEG alpha asymmetry in response to antidepressant treatment. There are some studies suggesting that frontal EEG alpha asymmetry in the

**Table 3** The comparison of methods in studies on frontal electroencephalogram alpha asymmetry of response to antidepressant treatment

Ref.	Sample		Age (yr)		Intervention time	% female	Diagnosis of depression	EEG detail			
	Experimental group	Control group	Experimental group	Control group				Reference montage	EO/EC	Recording length (min)	Alpha range (Hz)
Arns <i>et al</i> [70], 2016	EG: 236 Escitalopram, 251 Sertraline, 235 Venlafaxine-XR	CG: 336 controls	M = 38.85 (Escitalopram), M = 38.34 (Sertraline), M = 38.46 (Venlafaxine-XR), M = 37.84 (MDD)	M = 36.99 (Controls)	8 wk	57.03	DSM-IV; MINI; HRSD	AM	E0+EC	2	8-13
Vinne <i>et al</i> [71], 2019	EG: 136 treatment with escitalopram, 169 treatment with sertraline, 188 treatment with venlafaxine-extended release				8 wk	54.47	MINI; HRSD; VQIDS-SR	AM	E0+EC	4	8-13
Bares <i>et al</i> [72], 2019	EG: 57 SSRIs, 46 SNRIs		M = 46.04 (SSRIs), M = 44.83 (SNRIs)		1 wk	74.76	DSM IV; MADRS; CGI	Cz	EC	10	8-13
Szumaska <i>et al</i> [73], 2021	EG: 12 Mindfulness group	CG: 8 control group	M = 32.40 (Mindfulness group)	M = 35.00 (Control group)	8 wk	55	CES-D; MINI	Cz	E0+EC	3	8-13
Keune <i>et al</i> [74], 2011	EG: 78 MDD; 40 MBCT group	CG: 37 a wait-list condition	M = 48.93 (MBCT group)	M = 45.24 (wait-list group)	8 wk	74.03	DSM-IV	AM	E0+EC	8	8-13
Barnhofer <i>et al</i> [75], 2007	EG: 10 MBCT	CG: 12 treatment-as-usual	M = 48.00 (MBCT)	M = 38.60 (treatment-as-usual)	8 wk	50	MINI	AE	E0+EC	8	8-13
Gollan <i>et al</i> [77], 2014	EG: 37 MDD; Behavioral activation	CG: 35 non-MDD			16 wk	62.50	DSM-IV; IDS-C	AM	E0+EC	8	8-13
Allen <i>et al</i> [14], 2004	EG: 30 nonpharmacological intervention				8 wk	100	DSM-IV; HRSD	Cz; AM, AR	E0+EC	8	8-13
Spronk <i>et al</i> [78], 2008	EG: 8 MDD; rTMS Treatment		M = 42.60 (rTMS Treatment)			37.50	BDI; SCI	AM	E0+EC	2	alpha 1 (8-11); alpha 2 (11-13)
Wang <i>et al</i> [80], 2016	EG: 7 neurofeedback group		M = 47.43		6 wk	78.57	DSM-IV	Cz	EC	5	8-12
Vlcek <i>et al</i> [79], 2020	EG: 9 LF rTMS responder	CG: 16 LF rTMS non-responders			4 wk	80	MINI; MADRS; CGI	Cz	EC	10	8-12; 8-10; 10-12

EC: Eyes closed; EO: Eyes open; EEG: Electroencephalogram; HCs: Healthy controls; LF rTMS: Low-frequency Rtms; MBCT: Mindfulness-Based Cognitive Therapy; MDD: Major depressive disorder; rTMS: Repeated transcranial magnetic stimulation; SNRIs: Serotonin norepinephrine reuptake inhibitors; SSRIs: Selective serotonin reuptake inhibitors.

Diagnosis of depression: BDI: Beck Depression Inventory; CES-D: Centre for Epidemiological Studies Depression scale; CGI: Clinical Global Impression; HRSD: Hamilton rating scale for depression; MADRS: Montgomery and Asberg Depression Rating Scale; IDS-C: Inventory of Depressive Symptomatology–Clinician-Rated; MINI: Mini International Neuropsychiatric Interview; SCI: Structural clinical interview; VQIDS-SR: Very Quick Inventory of Depressive Symptomatology–Self Report.

Reference montage: AM: Averaged mastoids; AR: Average reference; Cz: The midline central position.

resting state may be more promising as an indicator of prognosis rather than diagnosis. This means that frontal EEG alpha asymmetry at resting state may be used as a biomarker for the stability and robust response of depression treatment. To date, some studies have tested the correlation between frontal EEG alpha asymmetry in the resting state and antidepressant treatment, including medical treatment, mindfulness treatment, behavioural activation treatment, acupuncture treatment, neurofeedback treatment, and transcranial magnetic stimulation (TMS) treatment.

### **Medical treatment**

Arns *et al*[70] investigated whether frontal EEG alpha asymmetry in the resting state predicted antidepressant treatment outcome for depressive disorder; 1008 depressed participants were randomized to eight weeks treatment with escitalopram, sertraline or venlafaxine-extended release. The results showed that there were no significant differences in frontal EEG alpha asymmetry in the resting state between depressed participants and healthy controls. This was consistent with the results of van der Vinne *et al* [71]. Their results found that frontal EEG alpha asymmetry in the resting state did not change significantly after eight weeks of escitalopram, sertraline, or venlafaxine treatment. Moreover, Bares *et al* [72] investigated the effectiveness of frontal EEG alpha asymmetry at baseline and its changes at week 1 in predicting the response to antidepressants. Both groups who were treated with selective serotonin reuptake inhibitors or the serotonin norepinephrine reuptake inhibitor showed no differences at baseline or change in frontal EEG alpha asymmetry at week 1. These findings suggested that antidepressant medication has no effects on frontal EEG alpha asymmetry in the resting state.

### **Mindfulness treatment**

Mindfulness training has been proven to be effective in reducing the recurrence rate of depression patients. Szumska *et al*[73] evaluated the impact of mindfulness training on depression and anxiety symptoms in individuals with depression and further evaluated whether the effect of mindfulness training in improving depression would be reflected by changes in frontal EEG alpha asymmetry in the resting state. Consistent with the results of previous studies, the depression and anxiety symptoms of the mindfulness training group were reduced, but there was no significant change in the average scores of frontal EEG alpha asymmetry in the resting state. Similarly, Keune *et al*[74] examined the effect of mindfulness training on residual depressive symptoms in patients with recurrent depression. The results showed that residual depressive symptoms and trait contemplation decreased, while trait mindfulness increased after mindfulness training, but this change did not occur in the waiting list control group. On the other hand, the average scores of frontal EEG alpha asymmetry in the resting state are not affected by training. These results provide strong support for the beneficial effects of mindfulness training in the treatment of depression. However, they do not support the hypothesis that asymmetric changes in the alpha band are used as the neural relevance of improvement in major depression. In addition, Barnhofer *et al*[75] investigated the effect of 8 wk of mindfulness training on preventing the recurrence of depression in individuals who were previously suicidal. The results showed that the relative activation of the left frontal lobe in the normal treatment group decreased, but there was no significant change in the mindfulness training group. The results suggested that mindfulness training can help suicidal depression patients maintain a balanced brain activation pattern.

### **Behavioural activation treatment**

Behavioural activation treatment is an evidence-based treatment for depression[76]. Gollan *et al*[77] examined frontal EEG alpha asymmetry in the resting state in depressed and healthy subjects undergoing behavioural activation therapy and assessed the predictive effect of frontal EEG alpha asymmetry at rest in remission of depression. The results showed that there were no significant changes in frontal EEG alpha asymmetry before and after treatment for participants with depression and healthy participants.

### **Acupuncture treatment**

Allen *et al*[11] examined the temporal stability of frontal EEG alpha asymmetry in the resting state in a sample of depressed women undergoing acupuncture treatment. The results showed that the asymmetrical frontal score generally showed good internal consistency and moderate stability over the 8- and 16-wk assessment intervals. In addition, changes in resting frontal asymmetry scores were not significantly associated with changes in depressive status between 8 and 16 wk.

### **TMS treatment**

Repeated transcranial magnetic stimulation (rTMS) is an effective treatment method for depression that has been found to respond in nearly 45%-55% of patients and in remission in 30%-40% of patients. Spronk *et al*[78] examined the clinical effectiveness of rTMS in treating depression. The results showed that all subjects showed complete remission within 20 sessions, significantly reduced depressive symptoms (BDI score) and neuroticism scores, and increased scores on the extraversion scale of the (NEO)-Five Factor Inventory (NEO-FFI) personality questionnaire. However, there was no significant change in the frontal EEG alpha asymmetry. More recently, Vlcek *et al*[79] investigated whether there

were different frontal asymmetry patterns between low-frequency rTMS (LF rTMS) responders and nonresponders. The results showed that there was no significant difference in frontal EEG alpha asymmetry in the resting state between LF rTMS responders and nonresponders.

### **Neurofeedback treatment**

Neurofeedback is a clinical intervention program designed to regulate brain activity and modulate frontal EEG alpha asymmetry. Wang *et al*[80] investigated the therapeutic effect of neurofeedback on patients with major depression. The results showed that the frontal EEG alpha asymmetry scores in the resting state decreased in the control group and increased in the neurofeedback group after the intervention. Depression and anxiety scores were significantly lower in MDD patients who received neurofeedback training than in those who did not. Furthermore, the results suggested that neurofeedback techniques can reduce right frontal lobe activation or increase left frontal lobe activation in patients with major depression.

In summary, most studies found that frontal EEG alpha asymmetry in the resting state in depressed patients consists mainly of trait-like features, and frontal EEG alpha asymmetry at rest was stable with little or no change between baseline and later assessment in depressed patients, although few studies suggest otherwise.

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## **LIMITATIONS**

Based on a review of the previous literature, it is clear that the relationship between frontal EEG lateralization and depression is not well defined, and there are a large number of inconsistent results. The results of a recent meta-analysis of the relationship between emotion regulation and frontal EEG asymmetry in depressed patients did not find frontal EEG asymmetry but rather a slight tendency towards left lateralization in the depressed group[81]. The study highlighted individual aspects of a study such as sample size and age group of participants as variables that influence effect size and disagreement between studies[38]. Consequently, inconsistent results about the studies on the relationship between frontal EEG alpha asymmetry in the resting state and depression may be due to differences in methodology (*e.g.*, reference electrode, alpha band), clinical characteristics (*e.g.*, diagnostic subtype, recruitment strategy), and participant characteristics (*e.g.*, gender, sample size). These issues have been discussed previously[19,34,50,82]. Therefore, this section provides a brief discussion.

### **Methodological differences**

One reason for the inconsistent results is that different types of reference points were used in the EEG space. Most studies use linked ears, average reference, and mastoid as references, and a few studies use earlobes, Cz, and nose as references. Moreover, Stewart *et al*[83] suggested the use of current source density as a reference, which could lead to more consistent results in resting-state EEG studies, and Jesulola *et al*[84] concluded that the use of a common average reference was beneficial in reducing noise and improving signal quality.

In previous studies, there was a wide variability in the specific frequency range of the alpha band. Most studies consider the alpha band between 8 Hz and 13 Hz, while some studies consider other alpha band frequency ranges, such as 7.5 Hz-12.5 Hz, 8 Hz-12 Hz, and so on. These differences in the frequency range of the alpha band may lead to inconsistent results.

### **Clinical characteristics**

Scales used for diagnosis vary across researchers, with the most common ones being the Diagnostic and Statistical Manual of Mental Disorders, followed by the Beck Depression Inventory[85]. Different scales may focus on different aspects of depression; therefore, the use of different scales may lead to difficulties in comparing the results of clinical depression diagnosis between studies.

In addition, although the clinical heterogeneity of depression is well known, few studies have examined its impact on frontal EEG alpha asymmetry in the resting state. Moreover, the results may be different when considering drug use and diagnosis. On the one hand, medication and drugs can affect the functionality of the brain; on the other hand, medication may have unknown interactions among themselves, changing the EEG signal even more.

### **Participant characteristics**

In many studies, there have been large differences in the proportion of male to female subjects in the sample. For example, some studies used only female subjects[19,38,44,50,86,87] or samples with significantly more females than males[32,88-90]. Related studies have found gender-related brain mechanisms and brain asymmetry that contribute to emotional processing[91-93]. This suggests that gender variables may play an important role in cognitive function and the possible organization of the cerebral hemispheres; therefore, samples should be more evenly proportioned between males and females.

### Effect size

Regarding effect size, the sample size of previous studies varied. For example, the sample size of most studies was generally less than 20 individuals[31,73,78,80,94-96], and some studies enrolled more than 100 individuals[35,97-99], but few had more than 200 individuals[27,82]. van der Vinne *et al*[82] suggested that a minimum of 300 participants is required to reveal patterns of frontal EEG alpha asymmetry in depressed individuals. Therefore, in these other studies that explored the patterns of alpha asymmetry in depressed individuals, the sample size may have been too small to be statistically significant.

## CONCLUSION

From the studies reviewed, frontal EEG alpha asymmetry in the resting state was related to depression. First, studies from clinical research showed that compared with individuals without depression, those with depression showed greater right frontal EEG alpha asymmetry in the resting state. However, the pattern of frontal EEG alpha asymmetry at rest in depressive individuals seemed to disappear with age. Future research on frontal EEG alpha asymmetry in the resting state should consider the influences of age and research on frontal EEG alpha asymmetry in the resting state to determine whether it can be used to quantify the degree of depression. Second, studies of intergenerational inheritance showed that compared with individuals without maternal depression, those with maternal depression showed greater right frontal EEG alpha asymmetry in the resting state, which indicated a genetic disposition towards greater right-sided asymmetry across cross-sectional assessments. Future research can focus on longitudinal investigations to examine whether there are long-term and lasting changes in the psychosocial risks faced by children. Third, studies of antidepressant treatment revealed that frontal EEG alpha asymmetry in the resting state was stable and there was little or no change after antidepressant treatment. Future studies could use frontal EEG alpha asymmetry in the resting state as an indicator of treatment effectiveness to examine the efficacy of other antidepressant therapies, such as the use of frontal EEG alpha asymmetry in the resting state as an indicator to investigate the relationship between ECT and depression. Finally, there were contrasting results regarding the relationship between frontal EEG alpha asymmetry in the resting state and depression, and these results may be due to differences in methodology, clinical characteristics, and participant characteristics.

## FOOTNOTES

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