

# Frontal EEG alpha asymmetry and attachment styles of people suffering from depression

Dawid Subocz

## Abstract:

**Objective:** The aim of this paper is to review the literature on frontal alpha asymmetry and attachment styles of people suffering from depression. The paper describes their possible interactions with affective regulation in patients.

**Materials and method:** The literature review was conducted using the following databases: PsycINFO, PubMed, Google Scholar. Each of the databases was searched using combinations of the following key words: “depression”, “attachment style” and “frontal alpha asymmetry”.

**Results:** The analysis of the literature confirms that people suffering from depression more often demonstrate insecure attachment styles. Some studies also suggest relationships between frontal alpha asymmetry and attachment styles and depression.

**Discussion and conclusions:** Studies on frontal alpha asymmetry among people with depression are diverse in terms of the experimental procedures used and data analyses. However, there are relatively few studies on frontal asymmetry in attachment. Despite this, the literature review suggests that frontal alpha asymmetry may be a neurocorrelate of dysfunctions in affective regulation in patients. Further research on the role of attachment styles in the pathogenesis of these dysfunctions is necessary.

**attachment style; frontal alpha asymmetry; frontal EEG asymmetry**

## INTRODUCTION

Aristotle [1] already stated that man is a social being and needs other people to live. Today, no one is surprised that relationships with other people are so strongly associated with mental health. Insecure attachment styles are a risk factor for the development of depression [2, 3]. However, it is still an open research problem to distinguish the neurobehavioural mechanisms mediating between attachment and depressive

symptoms. One such mechanism may be frontal alpha asymmetry. The aim of this paper is to review research on attachment styles and frontal alpha asymmetry among people suffering from depression.

## MATERIAL AND METHOD

The literature review was conducted using the following databases: PsycINFO, PubMed, Google Scholar. Each of the databases was searched using combinations of the following key words: “depression”, “attachment style”, and “frontal alpha asymmetry”. During the search, 216 pub-

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**Dawid Subocz:** University of Szczecin, Institute of Psychology, Szczecin, Poland

**Correspondence address:** Dawid Subocz; dawid.subocz@usz.edu.pl

lications were identified. Duplicates and papers that were not related to the purpose of this thematic review were excluded. Based on this selection, 51 publications were included in the qualitative analysis. The search and selection process of studies is presented in the PRISMA scheme (Figure 1).

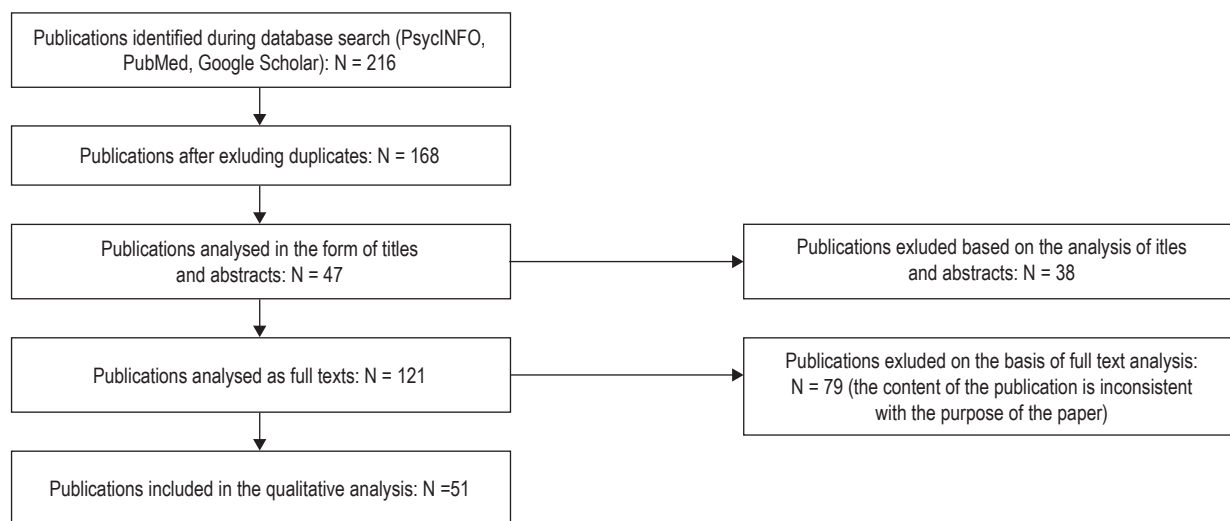


Figure 1. Attachment styles and depressive symptoms.

The role of experiencing loss in the development of depressive symptoms was noticed already in the mid-20th century [4]. Loss of health, loss of job, as well as loss of a loved one usually signal the appearance of low mood [5]. One of the most traumatic experiences for a child is the loss of a mother. Rene Spitz [4] drew attention to this. Based on observations of children separated from their mothers, he described the symptoms of anaclitic depression. These included: loss of appetite, sleep-wake rhythm disturbances, low mobility, lack of crying reaction typical for this period, symptoms of dyspepsia, loss of sucking and episodes of hyperthermia. The above symptoms disappeared within 3 months after the mother was reunited with the child [4]. The attachment relationship with the mother – even with her apparent physical presence – may manifest itself in periods of neglect or emotional rejection. In order to diagnose attachment styles, Mary Ainsworth [6] created the strange situation procedure. As part of this procedure, the caregiver plays with the child in a room. After some time, at the researcher's

request, the caregiver leaves the room, leaving the child alone. Finally, the caregiver returns to the room. The child's attachment style is diagnosed based on their reactions within the above procedure. A secure attachment is characterised by negative reactions when the caregiver leaves and positive reactions when he or she returns

to the room. Children with an anxious-ambivalent attachment are most often depressed when the caregiver leaves, while negative or ambivalent reactions appear when the caregiver returns. Children with an avoidant attachment, on the other hand, are characterised by avoiding or ignoring the caregiver, both when the caregiver leaves and when he or she returns [7]. According to attachment theory [8, 9, 10], memories of interactions with the parent are the basis for the development of internal working models. Internal working models are cognitive structures based on generalised memories of previous interactions with the attachment figure. They allow for the formulation of expectations regarding the future reactions of the attachment figure. These are cognitive structures defining "self", "others" and the relations between them [11]. In the first period of life, internal working models are non-declarative memory structures. They do not require language or consciousness. During development, some of the internal operating models may enter the conscious level [11]. Later attempts to create attachments may modify

them, but longitudinal studies confirm that they are stable over time [12]. Therefore, attachment styles are defined as individual characteristics. Questionnaires such as Experience in Close Relationship (ECR) [13], Adult Attachment Scale (AAS) [14], Relationship Questionnaire (RQ) [15] and Attachment Style Questionnaire (ASQ) [16] are used to diagnose them.

Questionnaire surveys indicate that both avoidant and anxious attachment styles are associated with depressive symptoms [17, 3]. Both of these attachment styles increase the risk of developing depressive symptoms during both an eight-week and a two-year observation period [18]. Studies indicate an association between avoidant attachment style and antenatal depression in mothers [19]. Moreover, people with clinical depression have a higher level of anxiety and avoidance in relational situations compared to healthy individuals. Suicide attempts were also more common among depressed patients with anxious attachment styles [20]. Studies confirm the association between avoidant and anxious attachment styles and depressive symptoms in both children [21] and adults [2]. Adults with insecure attachment styles are convinced that they cannot count on help from others in difficult situations and try to cope with emotions on their own. They avoid seeking social support, which makes them more vulnerable to developing posttraumatic and depressive disorders [11].

The key to explaining the relationship between depression and attachment styles is affective regulation. The basic task facing the caregiver in the first year of an infant's life is to create a secure bond with the infant [22]. It depends on the psychobiological synchronisation of the mother with the child's internal arousal states. Through visual-facial, tactile-gestural and auditory-prosodic communication, the caregiver and the infant modify their behaviours in order to attune to each other [23]. In this way, a trusting attachment is created. During affective communication based on bodily experiences, the caregiver and the infant gaze at each other. A mother attuned to the infant synchronises her behaviours with the infant's internal arousal state. Through this mutual responsiveness, the mother assesses the child's non-verbal expressions of affective states, then regulates them and communicates

them to the child [22, 23]. A child with trusting attachment observes the mother's facial expressions and learns to identify and regulate its own emotional states [23]. The exact opposite is true in the case of an insecure attachment relationship. The unavailability of the caregiver results in the emergence of secondary affective regulation strategies.

According to the model of Mikulincer, Shaver and Pereg [24], two secondary affective regulation strategies can be distinguished: deactivation and hyperactivation. In a situation when relational experiences do not provide gratification or are punishing in nature, the child develops a deactivation strategy. In order to avoid distress, the child distances itself from the parent. It suppresses its emotions, thoughts and memories related to the attachment figure. Deactivation is used when the relationship with the attachment figure is not perceived by the child as having the potential to regulate the state of arousal. When the child perceives the rewarding potential of the relationship, the deficit of which is experienced, it will most likely use the hyperactivation strategy. This strategy is characterised by increased vigilance. It is focused on attracting the attention of the caregiver and maintaining closeness [24]. The hyperactivation strategy is responsible for the creation of a self-developing cycle of distress with chronic activation of the attachment system [25]. Secondary affective regulation strategies may be associated with the risk of developing mood disorders.

### Frontal alpha asymmetry and attachment styles

Attachment theory [8, 9, 10] can be called a regulatory theory in which attachment is a process of biological synchronisation between mother and child. The next step in the development of Bowlby's theory [8, 9, 10] is the search for neural circuits on the basis of which patterns of behaviour, thinking and feeling develop within attachment relationships. Allan N. Schore [26] pointed out that in infants under 2 years of age, the volume of the right hemisphere of the brain is significantly larger than the left one. What the child experienced in the attachment relationship during the first 2 years of life therefore has a direct impact on the experience-dependent maturation of

the right hemisphere of the brain. Schore [22, 26] emphasises the role of strong connections between the right hemisphere and the limbic system, which allows for the processing of socio-emotional information. Studies suggest that the bioelectric activity of the frontal areas of the brain is related to affective regulation, which develops within a secure attachment relationship [27]. This may constitute a starting point for neurophysiological studies of frontal alpha asymmetry in attachment. Alpha waves have a frequency of 8 to 12 Hz. They are characteristic of the state of relaxation. Frontal alpha asymmetry is calculated as the difference in the activity of the right and left hemispheres in the frontal areas. It is determined based on raw EEG data obtained from electrodes F3/F4 and F7/F8. The most commonly used way to calculate the asymmetry index [28] is to subtract the natural logarithm of the left hemisphere alpha power from the natural logarithm of the right hemisphere alpha power ( $\text{LnRight} - \text{LnLeft}$ ). The Fox and Davidson model [29] interprets the right frontal activation as being associated with withdrawal and the expression of negative affect, while the left frontal activation with approach and the expression of positive affect. Davidson and Fox [30] assessed frontal asymmetry in 13-month-old children during their separation from their mothers. The researchers showed that children with right-sided frontal asymmetry cried significantly more often during separation than children with left-sided alpha wave asymmetry. After three years, the study was repeated and the results showed the same relationships [31]. Right frontal activation is often found in infants with an ambivalent attachment style. This may suggest a lower threshold for releasing negative affect in this group. In infants with an avoidant attachment style, left-sided frontal activation is more common. This may suggest a higher crying threshold in this group and a tendency to express positive emotions more often. The strange situation procedure was also used to study children whose mothers suffered from depression. The researchers [32] showed that children with a secure attachment style and mothers who did not suffer from depression showed higher right-sided frontal activity. Children with a secure attachment style, whose mothers suffered from depression, showed higher left-sided frontal ac-

tivity during separation. The increased left-sided activity in these children suggests that they inhibit their negative emotions more intensely.

The researchers [33] designed an experiment in which infants watched a separation situation on a monitor screen ending in either ignorance or comfort. The patterns of EEG activity were unrelated to how the separation situation ended. However, children with organised attachment styles were characterised by right-sided activity in the frontal alpha bands in the parts of the animation depicting separation. This indicates motivation to withdraw from the stressful relational situation. The withdrawal pattern was not present in children with a disorganised attachment style. These children did not tend to adaptively withdraw from the situation in which separation from the attachment figure occurred.

A separate area of research on frontal EEG asymmetry is research conducted on adults. This group of subjects is usually exposed to stimuli related to attachment. One such study [34] used 48 short video clips. People characterised as avoidant showed right-sided frontal asymmetry in response to clips showing positive attachment-related situations. People with a pre-occupied attachment style showed greater left-sided activity in response to the same positive situations. Exactly the opposite patterns were observed in the case of clips showing fear-related relational situations. The study did not show any differences in frontal asymmetry between secure attachment and all insecure attachment styles combined. This is important because other researchers [35] showed such differences at the level of absolute power of alpha2 bands in frontal areas. They compared adults with secure attachment styles with those with insecure attachment styles. It turned out that when listening to a baby's cry, people with a secure attachment style showed lower absolute power of alpha2 bands in frontal areas. The above comparison of results suggests that while the frontal asymmetry index does not allow for a categorical distinction between people with secure attachment styles and those with insecure attachment styles [34], the absolute power index of alpha2 waves does [35].

To conclude, studies on frontal alpha asymmetry in attachment are diverse in terms of the experimental procedures used. Interpretation of

the results of studies is also difficult due to their relatively small number. However, the data to date suggest associations between frontal alpha asymmetry and reactions in relational situations. It has been shown that these reactions are dependent on the attachment styles of the subjects.

#### Frontal alpha asymmetry and depression

Frontal alpha asymmetry is one of the most popular measures of depressive disorders in electrophysiological studies. Some studies [36, 37, 38] have shown significantly greater activity of the right frontal area and/or lower activity of the left frontal area in adults suffering from depression, compared to healthy individuals. The above data lead some practitioners to interpret alpha power asymmetry as a biological marker indicating the risk of developing depressive disorders. Unfortunately, not all studies confirm the pattern of asymmetry among patients with depression [39, 40]. Due to the above doubts, a group of Polish researchers [41] decided on a multi-path approach. They used five independent data sets ( $N = 388$ ) and conducted 270 analyses on them. Only 13 out of 270 analyses showed significant results.

A possible reason for the discrepancy in the results of the studies is the methodology used in them. In most studies, measurements of frontal EEG asymmetry are performed during rest. Smith et al. [42] suggest that the relationship between depressive disorders and frontal EEG asymmetry is stronger when the participant is assigned an emotional task as part of the experimental procedure. Another area that requires clarification is whether frontal EEG asymmetry in depression should be understood as a state that manifests itself during a depressive episode or as a feature that occurs both before the onset of depression and during remission of its symptoms. Studies support the hypothesis based on the concept of trait. They have shown that reduced activity of the left frontal hemisphere persists in adults with depression during remission of symptoms. The activity of the left hemisphere was reduced in them compared to the control group [36, 37]. Other studies [43] supporting the trait hypothesis indicate no relationship between frontal alpha asymmetry and the severity of acute depressive symptoms in patients with a history of depression. Other researchers have shown that while in healthy individuals the

frontal EEG asymmetry is stable over time, in people suffering from depression it is not [44]. This may in turn be an argument for the supporters of the hypothesis that EEG asymmetry is only a state.

Another reason for the discrepancies in the results regarding frontal asymmetry in depression may be the differences in terms of gender control in studies. Some studies suggest that the effect of frontal asymmetry is dependent on it [45]. Moreover, study reviews suggest a more frequent occurrence of depression among women [46]. Some researchers [47] look for the reasons for these differences in the neural systems regulating the response to stress and higher rates of psychological resilience among men. The more frequent occurrence of depression in men may also be an artefact, the causes of which lie in the less frequent seeking of help by men [48]. In addition, male depression is more often manifested by acting-out behaviours, impulsivity and substance abuse [49]. In women, anxiety symptoms are more frequent [50]. These discrepancies in symptomatology may make it difficult to diagnose depression in men. Considering the above issues, gender control should be implemented in studies on depression and frontal asymmetry in the alpha band.

The reason for the discrepancy in the results of studies on frontal asymmetry may also be diseases co-occurring with depression, especially anxiety disorders. This thesis is supported by a study [51], in which greater activity of the right frontal area was demonstrated in people suffering from depression and anxiety disorders at the same time, compared to healthy people. This study did not show that people suffering from depression, without co-occurring anxiety disorders, showed greater frontal asymmetry compared to the control group.

Additional doubts in treating frontal EEG asymmetry as a marker of depression are raised by issues related to data analysis. Topographic maps are rarely presented in studies [41]. Without them, it is difficult to determine whether alpha asymmetry in frontal channels does not result from asymmetric projections from extra-frontal sources. Unfortunately, studies usually analyse only one or two pairs of channels. Analyses on many pairs of channels with correction for multiple comparisons and localisa-

tion of the basic neuronal source of suggested activities would be beneficial. We still know little about the connections between cortical activity and subcortical structures. Increasing knowledge in this area requires the use of several measurement tools, for example, simultaneous use of EEG and fMRI.

Despite the above doubts, some studies confirm relatively higher alpha band power in the left frontal canal compared to the right one in patients with depression [52, 53]. Considering the studies that did not confirm the above results, it is worth postponing premature optimism about finding a biomarker of depression. Patients also show lower activity of such neuronal structures as the hippocampus, locus coeruleus and raphe nuclei, in which neurons producing serotonin are located [54]. Reduced serotonin levels may make it difficult to suppress the activity of the amygdala, as well as reduce the activity of the frontal cortex, which is responsible for controlling affective processes. Complex structural and functional changes in the brain, as well as dysfunctions of the serotonergic and noradrenergic systems, make it difficult to control variables in studies on frontal asymmetry in depression.

In this study, the focus was on relational experiences, which are somehow reflected in affective regulation strategies and frontal EEG asymmetry. However, the discussed model cannot be separated from the broader perspective of the pathogenesis of disease symptoms, which are also related to genetic factors [55]. It is worth emphasising that studies suggest lower activity of the prefrontal cortex in people with depression [56], which may be related to both frontal EEG asymmetry and dysfunctions in the area of affective regulation [56]. Study reviews [57, 58] show that the regulation of emotions is mediated by connections between the prefrontal areas and the amygdala, the activity of which is higher in patients with depression [59]. This may suggest that the prefrontal areas of patients do not sufficiently control the activity of the amygdala, which in turn is the basis for the development of anxiety and depressive symptoms [59]. Some researchers suggest that the above dysfunctions may be related to the frontal asymmetry of patients with depression and to cognitive-emotional control and processing [60]. Affective regula-

tion disorders in patients are somehow entangled in neuronal dysfunctions, the projections of which may be related to the aforementioned frontal alpha asymmetry. This asymmetry cannot be treated as abstracted from genetic factors [55]. However, this does not mean that relational factors play a secondary role in the pathogenesis of disease symptoms. The perspective of Antoni Kepiński [61] still seems to be relevant, according to which mental illnesses result from the interaction of many factors, the understanding of which requires an individual approach to each patient. The role of attachment relationships may therefore be both crucial in the development of problems with affective regulation and also be a secondary problem in relation to the symptoms of depression. It is therefore necessary to learn about the patient's unique experiences, life context and to balance the role of genetic susceptibility and epigenetic factors in the pathogenesis of disease symptoms [62].

## CONCLUSIONS

Even the studies on the frontal alpha asymmetry among people suffering from depression are not unambiguous [39, 40, 41]. The most important reasons for the different results obtained by researchers: taking measurements in different experimental situations, non-unified data analysis standards, and varying control of confounding variables. While there is a relatively large number of studies on frontal asymmetry in depression, studies on frontal asymmetry in attachment relationships have attracted much less interest. This is intriguing, because it is precisely entering into interpersonal interactions that constitutes a significant strategy for regulating affective states. The lack of a secure attachment relationship may disturb affective regulation. Insecure attachment styles are more common among people with mood disorders [2, 18]. It can therefore be assumed that frontal asymmetry should mediate these relationships. However, the problem of the cause and effect of the mechanisms discussed remains unresolved. The relationship between depression and attachment styles may have three directions:

- a) Insecure attachment styles may lead to problems with affective regulation in diffi-

cult situations, which is why such people tend to develop mood disorders;

- a) People with mood disorders avoid interpersonal contacts, as a result of which they lack positive relational experiences and develop insecure attachment styles;
- a) Mood disorders and insecure attachment styles develop in parallel, interacting with each other and perpetuating maladaptive mechanisms of affective regulation.

At the same time, the question about the role of frontal alpha asymmetry in the above potential paths of development of psychopathology remains relevant. The above review of literature shows that frontal alpha asymmetry is not so much a biomarker of depression as a neurocorrelate of dysfunction in affective regulation, perpetuating only symptoms of depression. In order to confirm this hypothesis, however, further research is necessary on the relationships between individual symptoms of depression and affective regulation strategies and the mediating role of activity of cortical and subcortical areas of the brain.

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