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The relation between cerebral morphological abnormalities in major depressive disorder and antidepressant treatment

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Abstract. The cerebral morphological abnormalities caused by Major Depressive Disorder (MDD) are markedly different from those of many other cerebral parts in non-patients. This brain disorder may be coordinated by antidepressant treatment. This paper investigates the links between antidepressant medication and morphological features in MDD using a literature review method. The paper finds that there is a positive response and feedback on these neurobiological alterations. The relationship between morphological features and treatment may make it easier to objectively observe the effects of antidepressants on MDD patients. The review emphasizes the importance of understanding the cerebral morphological changes associated with MDD, as well as how antidepressant therapy can potentially modulate these alterations. By exploring the relationship between pharmacological interventions and structural brain changes, the paper provides insights into the neurobiological mechanisms underlying the therapeutic effects of antidepressants. This knowledge can enhance the advancement of more focused and efficient therapy approaches for patients with Major Depressive Disorder.

Keywords: Cerebral morphological abnormalities, Major Depressive Disorder, antidepressant treatment.

1. Introduction

Major depressive disorder is a severe illness. Symptoms of depression include a low mood, reduced interests, difficulties with thinking, and physical symptoms such disrupted sleep or changes in eating. MDD is linked to changes in specific areas of the brain, including the hippocampus, as well as modifications in brain circuits such as the cognitive control network and the affective-salience network [1]. A major depression is connected to atrophy within the central nervous system [2]. Many studies attest that antidepressants can be targeted and effective in treating the cerebral morphological abnormalities in Major Depressive Disorder.

Researchers propose that Major Depressive Disorder is associated with cerebral morphological abnormalities. Antidepressant medication can potentially regulate disorder [1]. The neuronal activity in depression, as well as the observed changes in cerebral morphology resulting from neuroplasticity, may be improved by antidepressant therapy.

The part of the brain where MDD has an obvious influence on cerebral morphology and the part of the brain that is non-negligible to physical health, such as the amygdala, hippocampus, and grey matter, are selected as the principal parts of the discussion via a literature review. The connections between morphological features and treatment may make it easier to observe objectively the effects of

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antidepressants on MDD patients. This work may provide a perspective on understanding the mechanisms of antidepressant action and may offer clues for biomarker research in Major Depressive Disorder.

2. Case Study

Impaired functioning of the amygdala may contribute to the development of depressive symptoms [2]. The amygdala is responsible for the retrieval of affective memories, particularly those that are linked to anxiety or fear [3]. It is well-established that the amygdala is involved in the retrieval of emotional memories, particularly those that are acquired in apprehensive or anxious situations.

2.1. Amygdala abnormalities.

Researchers selected eighty patients with MDD who compared with eighty-three HC without MDD. The researchers discovered a noticeable and substantial rise in volume measurements on the right side compared to the left side in the group with Major Depressive Disorder (MDD), which was not observed in the healthy control (HC) group. In Major Depressive Disorder (MDD), there was a substantial increase in the size of the right entire amygdala and right laterobasal composite compared to the left side. The central and medial nuclei in MDD exclusively showed greater right-left asymmetry at the nuclei level, as shown in Figure 1 [2].

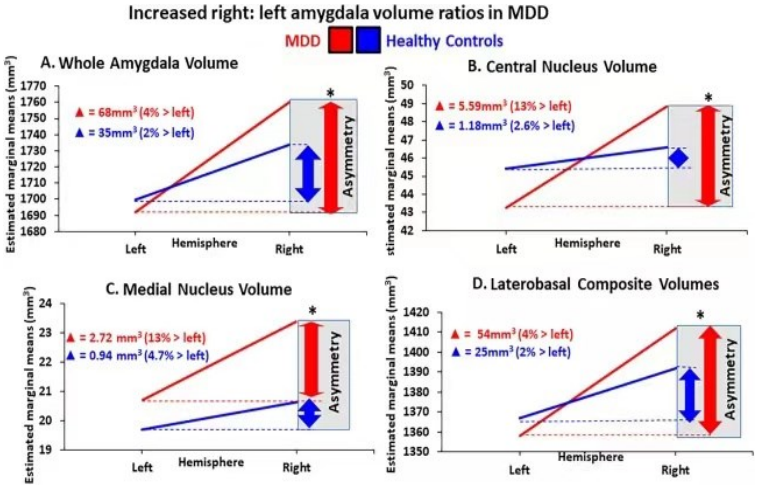

Figure 1. Amygdala substructure volumes in Major Depressive Disorder [2]

This study investigated the disparities in the size of the amygdala substructures between individuals diagnosed with depression and a group of healthy individuals. The results showed that MDD patients have a larger right intermediate nucleus volume, and the right-to-left ratio of the amygdala as a whole and its substructures increased. Additionally, the cortisol arousal response (CAR) of MDD patients was negatively correlated with the left cortico-amygdaloid transition zone (CATA). These results illustrate that the volume of amygdala substructures may be significant for the MDD pathophysiology. This actually demonstrates the influence of major depressive disorder on amygdala form and function.

This paper explores the relationship between antidepressant treatment and the repair of amygdala structure, taking into account the impact of depression on the amygdala. The hypothesis of the study was that the administration of antidepressant therapy would be associated with changes in the functional connectivity of the amygdala. The correlations between task-related brain activity in the right or left amygdalae and the perception of sad faces, as well as all other brain regions documented in functional magnetic resonance imaging (fMRI), are analyzed in a factorial study design.

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2.2. Amygdala coupling in MDD with antidepressant drugs

Figure 2 illustrates a positive association between the right and left amygdalae and the medial temporal lobes and ventral occipital cortex on both sides. The amygdala had a negative connection with the anterior cingulate cortex. The study found that both healthy volunteers and depressed patients showed similar patterns of amygdala coupling, on average, at both time points (week 0 and week 8) [4].

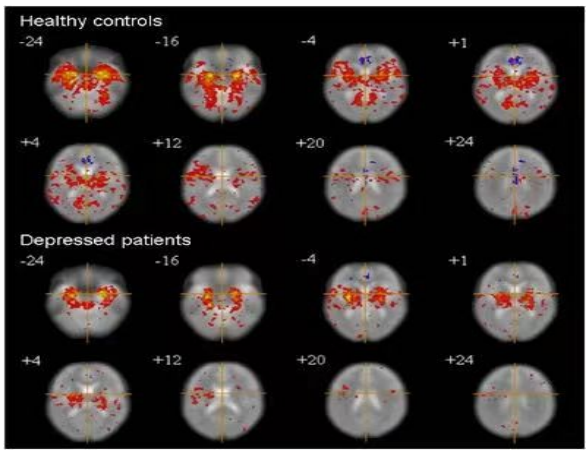

Figure 2. Functional connectivity of the amygdala in patients with depression undergoing treatment with antidepressant medication. [4]

Figure 3 provides evidence that the alterations in the timing-related connection between the amygdala and the nucleus accumbens have a significant influence. Patients with depression exhibited a notable increase in the connection between the amygdala and the right prefrontal cortex, specifically in Brodmann areas 46, 45, and 48. This increase was also observed in the anterior cingulate cortex (BA 32), insula, thalamus, caudate nucleus, and putamen. These findings provide anatomical details regarding the observed increase in coupling over time. Given that the patients were administered antidepressant medication during the course of 8 weeks between the initial assessment and the final imaging evaluation, these findings indicate that the antidepressant medication was successful.

2.3. Functional coupling of the amygdala in MDD with antidepressant drugs

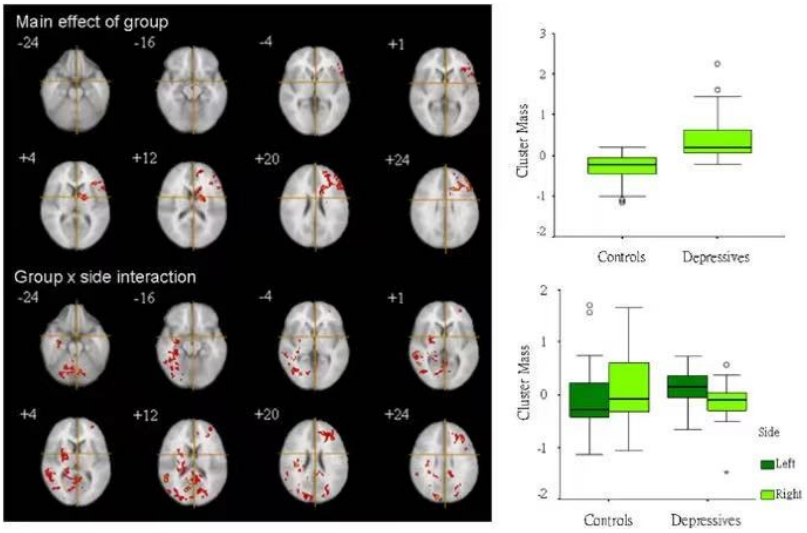

Figure 3. Functional connectivity of the amygdala in patients with depression undergoing treatment with antidepressant medication [4]

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Temporal correlations have a factorial effect on the coupling of the amygdala function. As shown in the left figure of Figure 3, the first two rows show the anatomical locations that significantly influence the main effects of the group on the temporal correlations in the coupling of the amygdala function. Over the course of the 8-week therapy with antidepressant drugs, there was a notable rise in the temporal correlations between the bilateral amygdala and certain regions, for example, the right prefrontal cortex and the anterior cingulate cortex. The final two rows illustrate the anatomical positions where an important group interaction occurs. Those regions exhibit temporal correlations.

This study examined how antidepressant treatment affects the way the amygdala connects with other parts of the brain in depression patients. Using fMRI, the researchers discovered that the amygdala had positive connections with the medial temporal lobe and ventral occipital cortex, but negative connections with the anterior cingulate cortex. After taking antidepressants, there was a significant increase in the connections between the amygdala and the right prefrontal cortex, cingulate cortex, striatum, and thalamus. This increase in connections was more pronounced in the left amygdala than in the right. The findings suggest that antidepressant treatment can modify the way the cortex and limbic system communicate, thereby influencing brain function, particularly by strengthening the connections of the left amygdala.

2.4. the abnormalities of hippocampus

The hippocampus is a region of the brain, which has undergone thorough examination in individuals with mood problems. This fascination is based on an extensive collection of cognitive and neuroimaging research. The hippocampus has a role in the acquisition and storage of episodic, declarative, contextual, and spatial information [5,6]. A Danish positron emission tomography (PET) study, involving 42 individuals with acute depression and 47 healthy volunteers with similar characteristics, discovered a notable elevation in blood circulation to the right hippocampus. [7-8]. Consequently, numerous other positron emission tomography (PET) studies have discovered irregularities in this particular anatomical region in individuals with depression, using different scanning techniques [9]. Researchers have utilized magnetic resonance imaging (MRI) since 1993 to examine the hippocampus of individuals with unipolar depression. These studies have aimed to uncover alterations in volume, density, and water contents. Several volumetric studies have identified substantial bilateral reductions in brain volume in individuals with severe depressive illness [10]. Previous studies have reported reduced volumes either in the right hemisphere or in the left hemisphere [11]. A meta-analysis was conducted on 12 trials that included 351 individuals with bipolar depression and 279 healthy controls. The Derimonian-Laird effect size was determined using a random effects model to analyze the influence of depression on hippocampus volume. Individuals with depression exhibit a reduction in hippocampus volume, although research findings have been inconsistent. An analysis combining data from 12 monophasic MRI studies on depression, which matched particular criteria, revealed that patients experienced an average decrease of 8% in the volume of the left hippocampus and a 10% drop in the volume of the right hippocampus [12].

Researchers analyze the impact of MDD on the structure of the hippocampus and explore the potential of antidepressant treatment to promote hippocampus repair. The study specifically examined tree shrews, a primate resembling apes, which have been commonly utilized by researchers to investigate depression resulting from psychosocial conflict and social attachments [13]. The participants experienced five weeks of this type of stress and were administered either the placebo or the antidepressant tianeptine for the final four weeks. Thus, the temporal progression of stress and the administration of antidepressant treatment closely resembles the typical experience of individuals with depression who are on medication. The basal cortisol levels experienced a 50% increase. The proton magnetic resonance spectroscopy of the cerebrum showed a decrease of 13-15% in markers of neuronal viability and function (namely, the neuroaxonal marker N-acetylaspartate), cerebral metabolism (creatine and phosphocreatine), and membrane turnover (choline-containing compounds). In contrast, no change was detected in a glial marker associated with vitality (myo-inositol). Furthermore, psychosocial stress led to a decrease of around 30% in the production of new cells in the hippocampus. Ultimately, this stress was found to have a minimal effect on the overall volume of the hippocampus

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[14]. This demonstrates that antidepressant medications exert a specific impact on the structural alterations of the hippocampus.

3. Discussion

By comparing the volume of amygdala between healthy controls and MDD, the researchers come to the conclusion that the volume of amygdala in MDD patients is larger than that under healthy controls. After that, the imaging of healthy controls and MDD receiving antidepressant treatment was compared in the coupling mode. It is concluded that antidepressant drugs can alleviate the cerebral morphological abnormalities caused by major depression to some extent. In an MRI study, researchers found that the hippocampus of MDD patients is usually smaller than that of healthy controls. Researchers subjected tree shrews to long-term and severe social psychological stress in an experiment and successfully induced depression in them. After that, researchers used tianeptine, a representative of antidepressant drugs, to treat the tree shrews. The treatment results showed that antidepressant drugs have an effect on neuronal activity, neurotransmitter secretion, and hippocampus volume.

4. Conclusion

The paper primarily connects the brain morphological abnormalities caused by major depressive disorder with antidepressant treatment and discusses the possibility of making these brain distortions reversible through antidepressant treatment. The experiments reveal that antidepressant drugs can partially alleviate the cerebral morphological abnormalities caused by MDD, including the degree of functional coupling between the amygdala and neurotransmitter secretion. The paper selects brain regions that are more important and have a more significant impact, which also provides clues for understanding the pathogenesis of major depressive disorder. .It may provide a special perspective for antidepressant drugs and treatment and may provide clues for the study of the morphological and pathological characteristics of depression. However, there are still limitations in the study, and it is not clear how long-term the antidepressant drugs can relieve brain distortion. Selecting only a subset of brain structures associated with antidepressant treatment may result in sbias and reduced validity. Therefore, in subsequent studies, a more comprehensive understanding of the brain morphological distortions associated with depression and the corresponding effectiveness of antidepressant treatment in alleviating these distortions will be sought.

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