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Identifying the relationship between depression and cognitive neuroscience

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Abstract. Major depression (MD) is increasingly prevalent worldwide and more practical treatments are in demand. Discovering its mechanisms and affective factors could help in finding better treatment and understanding how each individual's differences affect the mechanisms. This article focuses on discovering the relationship between depression and neuroscience, particularly on the cognitive aspect, emotion regulation, and brain structure. Three main executive functions in cognitive function, including updating, shifting, and inhibition are analyzed. Moreover, this paper indicates the relationship between dorsal anterior cingulate cortex (ACC) and posterior cingulate cortex (PCC) in the brain structure and depression by using functional magnetic resonance imaging (fMRI). With integrated information providing insights, several notable factors and mechanisms have been found. This proves that cognitive executive processes have a direct impact on emotion, where updating working memory can reduce the effects of negative emotions; however, shifting impairment tends to experience emotional dysregulation, and inhibitory deficits may impair adaptive emotion regulation processes. In addition, the prefrontal cortex (PFC) region of the brain structure manages the functions of emotion regulation and cognitive regulation. In a parallel understanding, cognitive dysfunction is associated with dynamic emotional states and MD psychopathology. Based on these findings, several possible treatments are subsequently put forward. Meanwhile, researchers discovered new directions to examine the relations between MD and neuroscience.

Keywords: depression, neuroscience, cognitive dysfunction, emotion regulation, mental disorder.

1. Introduction

Major depression (MD) is one of the world's most prevalent mental disorders with altered cognition and featured emotional dysfunctions. One of its key features is that it downgrades patients' quality of life to all extent, especially their social life. It represents a negative cognitive mode. Contemporary studies have shown its strong association with cognitive dysfunctions in different aspects including attention, working memory, executive processes, perception of information, motivation, and psychomotor skills [1, 2]. Emotion regulation is also a significant contributor to MD [2]. In the discussion to straighten out the connection between neuroscience and depression, the brain structure cannot be overlooked.

This article discusses three primary executive functions, which are updating, shifting, and inhibition, along with the association between the human anatomy structure and the cognitive function, as well as the relationship between cognition and emotion control. Besides, this article also analyzes how the

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problem of lacking motivation affects MD and viable mechanisms. Some new possible treatments and research directions are studied as well. This article gathers integrated information about MD and neuroscience, providing a new and comprehensive image to better understand the underlying relations.

2. Cognitive dysfunctions

Cognitive control refers to a series of mental processes that moderate an individual's cognition and behavior into consistency with his/her current goals flexibly and accurately [3]. To fulfill a goal-aimed task, it is required that people keep task-related information alive and activated in working memory and keep task-irrelevant information out of the way. In other words, cognitive regulation helps moderate unpleasant experiences and one of its major effects is neutralizing negative awareness and possibly diminishing physiological arousal. Attention control is a cognitive ability to enable people to pay more attention to goal-related information and ignore irrelevant materials [2]. It makes cognitive appraisal of emotions easier by evaluating affective stimuli. It also lessens rumination. Recent studies showed that depression is linked with malfunction of cognitive control, majorly emphasized functions to update, shift, and inhibit, which are executive processes [2]. These deficits have relations with emotion dysregulation as well [2].

2.1. Updating

Updating is the ability to keep working memory flexibly and efficiently updated. Moreover, it can also add or discard working memory contents, which can avoid perseverative thinking or excessive focus on negative stimuli, such as rumination and persistent negative mood [2]. The updating process keeps working memory goal-relevant all the time. Studies have shown that depressed individuals have to pay more effort to manipulate negative materials in working memory than control participants [2]. In healthy participants, updating working memory decreases the influence of negative emotions by moderating reappraisal and rumination [2]. Plenty of empirical evidence has shown that in the experiments of multiple task cases, the updating function of the depressed individuals is in deficit and this may happen because of rumination and reappraisal ability [2].

2.2. Shifting

Shifting refers to switching between different tasks or mental sets smoothly and efficiently [4]. Shifting shows a possible tendency to influence emotion regulation by promoting individuals to switch their emphasized focus away from negative emotional components and distribute it to positive emotional components and goal-related tasks. A related study has demonstrated that the sensitivity and speed of shifting are associated with the depressive state [2]. Notably, people having difficulties in switching tasks tend to have emotion dysregulation [2]. Patterns have been found that participants suffering from Major Depressive Disorder (MDD) and showing task-switching impairment usually have to endure rumination [2]. Rumination is an emotion regulation strategy. Increased rumination in daily life has great odds of aggravating task-switching impairment with a background of negative affective stimuli [2]. Some researchers advocate that the implication between the rumination process and shifting impairments come to be the strongest when the brooding component of rumination is exposed to subjects, especially with negative stimuli [5]. Researchers also discovered that when depressed individuals are directed to start rumination, they appear to have weakened switching ability compared with depressed individuals instructed to have distractions, as well as ruminating in distracted healthy controls [6]. Acting as a cognitive function role, shifting impairment shows strong links to the depression and activation level of different brain regions. There is believable evidence that rumination could drive or aggravate this implication. Since the evidence of global prevalence of shifting impairments in depression is lacking, the evidence showing its occurrence is particular in the context of emotional material stimuli.

2.3. Inhibition

Inhibition refers to the ability to dominate or override prepotent responses in awareness, which includes specific processes to inhibit the negative effect aiming at the task [4]. Inhibition deficits are likely to

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diminish adaptive emotion regulation processes, and they act in the role of modulating emotions to adapt current situation, for example, an individual overcomes and replaces an initial negative interpretation of an emotion-eliciting circumstance [2]. Evidence shows that inhibition deficits contribute to depression. Some trials have been carried out to prove the fact that depressed individuals have undermined their ability to inhibit negative materials toward a task, consequently influencing their ability to accomplish cognitive tasks [2, 3]. Inhibition also has links with rumination, and these links may become stronger with a specific emotional material [2]. Negative emotion distractors may aggravate conflicts for individuals with inhibition deficits, consequently showing high levels of depressive symptoms. Individuals with this case usually require additional efforts to cognitive control [2]. In summary, inhibition holds a significant component of cognitive function in depression, and an inhibition impairment exists in depressed patients, directly implicated with depressive symptoms and rumination.

Throughout the cognitive research and trials, it is obvious to find that a distinctive difference exists among individuals, namely heterogeneity. Therefore, there lies a potential value to investigate individual differences in cognitive control, which can bring profound insights to the understanding of the maintenance of negative affections and a vulnerable tendency to experience depressive episodes [2].

3. The association between brain structure and cognitive function

Functional magnetic resonance imaging (fMRI) shows evidence that depressive symptoms are implicated with a high level of activation of the dorsal anterior cingulate cortex (ACC) and posterior cingulate cortex (PCC) [2]. Thus these two regions of the brain structure are mainly focused on.

Prefrontal cortex (PFC) manages cognitive control and goal-directed action along with affective states and emotional processing [1, 2, 7]. The brain activation of the dorsomedial prefrontal cortex (dlPFC) increases executive dysfunction in patients and there come higher depression scores, implying that cognitive control dysfunction may act as a mediating in emotional dysregulation and psychopathology of MD [1]. Studies found that when using an external emotion cueing task to detect cognitive functions, participants in health control exhibited activation in the lateral PFC and parietal regions when attention is shifted away from irrelevant emotional cues. A possible reason to explain this is that irrelevant cues require more recruit of attention and additional efforts to control cognition. In other words, negative distractors display increased conflict for individuals with severe depressive symptoms, therefore demanding an exertion of additional cognition recruit. More depressive symptoms were claimed to implicate lessened activation in these regions on irrelevant emotion cues, implying a deficit in utilizing appropriate cognitive components to shift attention [2]. fMRI work presents that when depressive individuals remove negative words from working memory, their dorsal anterior cingulate and parietal and bilateral insular cortex are greatly activated [2]. Researchers also revealed a fact that an imbalance of function occurs between the right and left dlPFC in depressive cases, namely the "imbalance hypothesis of MD" [1]. This notion suggests hypoactivity on the left and hyperactivity on the right of the dlPFC. Researchers have combined this discovery with working memory impairment and attention issues [1], regarding this imbalance would reinforce depressive symptoms and disturb emotion processing. In summary, PFC has a directive effect on emotion, and when PFC region fails to play that role on emotion, dysregulation appears to disturb emotion [1].

From these research results, it can be observed that by decreasing the stimulation of PFC and improving cognitive control, patients would have great odds to diminish depressive symptoms.

ACC is another region related to cognitive control as well. Neuroimaging and electrophysiological studies provide evidence for reduced ACC activation in depressed individuals [3].

4. Structural mechanisms and potential treatments

From neuroimages in findings, investigators suggest an existing cognitive-biological model compromises two processes [8]. The first process, featured by hyperactivity, is regarded to show relations with rapid, degraded processing of affective materials and may trigger information biases in cognition. It displays active functions in the amygdala, thalamus, nucleus accumbens, hippocampus, caudate, putamen, and the anterior cingulate cortex. The second process, a dysfunctional top-down

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process, occurs among depressed individuals with minus cognitive control. It maintains cognitive information biases and follow-up negative sensation states and shows a promoted activation in ventral, dorsal, and medial areas of the PFC [2].

Cognitive bias modification (CBM) and cognitive control training (CCT) reduce depressive symptoms [2]. Experiments have shown that cognitive control has a leading effect on depressive symptoms and increased cognitive control through training lessens depressive symptoms in depressed or high-risk individuals [3]. CCT has a promising future and will likely help patients relieve from MD.

Another factor that requires attention is the lack of motivation, a contributor to depressive symptoms, which can be addressed by psychological therapies, and with its increase, patients would feel easier to recruit attention and cognitive function. However,current theories and empirical research paid little attention to motivation's effect on cognitive control in depressed individuals [3]. The control of goal-directed behavior has magnificent importance, thus motivation process bonds tightly with cognitive control. A lack of motivation may cause deficits in cognition control, yet how much motivation loss devotes to depression is waiting to be discovered. Moreover, the motivation level and attention control show paralleled extent [2]. The specific relationship between these two factors remains to be discovered.

During this research, the discovery of the effect of associative processing has caught people's eyes. Associative processing refers to promoting thought processes to proceed from one topic to another. Broad associative processing promotes positive mood, suppresses rumination, and activates shifting function, bringing depressed individuals positive sensations [9]. This notion provides a promising, viable, and seemingly effective approach to tackling depression.

5. Conclusion

Evidence shows that cognitive processes can be disturbed by psychiatric disorders such as depression, and relations between cognitive deficits and emotion dysregulation have been revealed. Previous studies showing cognitive components implicated with MD psychopathology suggest that in MD cases, biased information processing in cognition is relevant to emotion dysregulation [1, 2]. There also exist different advocations suggesting the relation contains mixed results. Cognitive executive processes have a direct influence on emotion, causing persistently frustrated moods. Another example is that inhibition impairment, a component of cognitive dysfunction, may diminish the function of adaptive emotion regulation processes such as reappraisal [2]. Also, updating processes can minimize the downside of the emotional effect [2]. There is also a structural focus on the role of PFC for this. The PFC region of the brain structure manages the function of both emotion modulation and cognitive regulation [1]. In a parallel understanding, cognitive malfunction is related to a dynamic mood state and MD psychopathology. In summary, cognitive regulation can be seen as an emotion regulation strategy that makes a difference in to emotional responsivity.

Depression has major implications to undermine the cognitive ability to set limits to the entry of irrelevant negative material into working memory (inhibition impairment) and the ability to erase negative contents from working memory that is no longer goal-related (updating impairment) [3]. Researchers propose that depressive-related dysfunctions are linked to the time, duration, and approaches to engage in goal-oriented processing [2].

Structural concerns focus on the brain regions of PFC and ACC. The excessive or diminished expression as the hypoactivity and hyperactivity of the brain regions and the phenomenon of dlPFC imbalance can cause malfunction, while cognitive control dysfunction is a systematic failure to engage in goal-aimed processes [3].

Individual differences referring to heterogeneity, influence cognitive functions [2]. It is also worthwhile to look into high-risk people to detect their developmental aspects of cognition management. More attention is required to study the causal effect of cognitive control, emotion dysregulation, and depression onset and maintenance [2]. Contemporary studies demonstrated that early adverse emotional information processing early may have something to do with genetic factors [3]. The specific mechanisms still require digging. There also lies a relationship between the above and parental warmth in childhood [3]. Furthermore, the significant impact that motivation and willingness provide on

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recruiting more effort in control cognition has been neglected [3]. The fact that cognitive dysregulation has tight bonds with MD is for sure. Yet the amount of its contribution is an open question demanding more research [1]. Notably, current research has put more focus on descriptive study and conceptualization rather than mechanic study. The integration of cognition, motivation, and emotion is yet to be summarized.

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