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Study directions and development of cognitive theory of depression

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Summary

Aaron T. Beck's cognitive model of depression became the basis for the analysis of cognitive factors as significant correlates and predictors of depression. Psychological mechanisms of depression related to the depressive cognitive triad, the specificity of cognitive schemas and errors in cognitive processing determined the directions of further research on depression.

The presented analysis of selected studies confirms and extends Beck's assumptions about the role of cognitive factors in the development and maintenance of depressive symptoms. The analysis of psychological, neuroimaging and biochemical studies provides a broader perspective of understanding both the etiology and mechanisms that sustain the symptoms of depression. The development of neuroimaging research has broadened the knowledge about the brain mechanisms of depression – the presented research combine the theoretical constructs of the cognitive concept of depression with the characteristics of the activity of brain structures and their functional connections. The following conclusions seem to be particularly important for clinical practice: (1) impairment of the cognitive inhibition function in depression reduces the control of negative automatic thoughts – strengthening working memory is important; (2) different brain mechanisms for processing rewards and punishments in people suffering from depression and in healthy people explain the formation of self-esteem – this knowledge can be an important element of psychoeducation in therapeutic work; (3) neuroimaging studies indicate neuronal correlates of cognitive distortions observed in depression – for further research on depression, it would be particularly important to monitor changes in brain activity in the course of its treatment.

Key words: depression, cognitive theory, Aaron T. Beck

Introduction

Just over thirty years ago, *Psychiatria Polska* published two articles in which the authors presented three cognitive theories of depression. These were: Beck's theory, Brown and Harris's theory of susceptibility, and Seligman's theory of learned helplessness [1, 2]. At that time, for many researchers and clinicians it was an innovative approach, constituting a kind of turning point in the understanding of the etiology of depression. The novelty of the cognitive approach to the causes of the appearance of symptoms of depression was based on the assumption that specific distortion of cognitive processes are the main cause of these symptoms. The distortions concerned cognitive constructs, which were divided into two fundamental types: (1) cognitive biases, including distortions of information processing processes and/or focusing attention on negative stimuli, and (2) cognitive deficits including attention and short-term memory deficits, and weakening of executive functions.

The aim of this study is to analyze the progress of research on the depressogenic role of cognitive factors and to evaluate the development of the cognitive theory of depression. We were inspired by an article written for the same purpose by its author – Aaron T. Beck [3]. It is particularly interesting whether the fundamental assumptions of the cognitive theory of depression, including the relationship between the functioning of the cognitive system and experienced emotions, are still valid in the light of many years of research and therapeutic practice. Additionally, to what extent the research with the use of modern tools and methodologies has enriched the knowledge about the neuronal correlates of depression and whether there are sufficient premises to see the causes of depression in functional disconnectivity disorders of the brain.

Aaron T. Beck's cognitive theory of depression

Beck divided the cognitive model of depression into three detailed sub-theories explaining the psychological mechanisms of depression, referring to: the depressive cognitive triad, the specificity of cognitive schemas and errors in cognitive processing [3, 4].

1. Depressive cognitive triad

The first of the sub-theories relates directly to clinical observations and describes three main cognitive patterns which are used automatically and habitually by the patient. These are: (1) negative beliefs and judgments about oneself (e.g., the belief that you are worthless, unnecessary, incapable of achieving happiness); (2) tendency to interpret current experiences in a negative way (e.g., the surrounding world is perceived as making excessive demands, which are extremely difficult to meet); (3) negative judgments and predictions about the future (e.g., ideas about the future are filled with beliefs about difficulties and failures).

The question then arises, how is it possible that the negative contents of the depressive cognitive triad persist in the patient's consciousness even though they are inconsistent – sometimes blatantly – with the objective facts? Two further sub-theories

of the cognitive model of depression are attempted to answer this question, especially the concept of depressive cognitive schemas relating to the methods of collecting and organizing information.

2. The concept of depressive cognitive schemas.

The depressive cognitive triad is shaped by early aggravating experiences that activate negative beliefs about oneself, other people and the future. Shaped patterns, a kind of instructions for collecting and processing information, determine the specificity of perception, evaluation and behavior. Activating negative beliefs inhibits previously available positive beliefs and evokes distortions in cognitive processes (Figure 1).

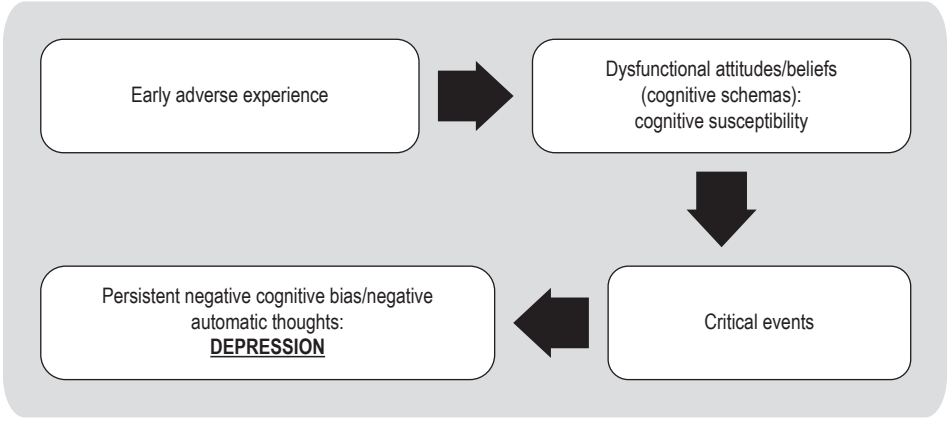

Figure 1. **Model of depression development based on cognitive susceptibility and stressful life events [3]**

The assumptions and beliefs are dysfunctional because they are not confirmed by reality, they evaluate new experiences habitually and automatically, and are extremely difficult to change. They can be in a "dormant state" and get activated, for example, by failure. Then, with varying degrees of intensity, they influence thought processes, intensifying the spiral of symptoms of the disease.

3. The concepts of dysfunctional information processing and thinking errors

The impact of the depressive cognitive triad and dysfunctional beliefs and assumptions are sanctioned by the presence of thinking errors appearing in the information processing. They make the patient feel that their system of cognition, thinking and judgments are correct and true. This conviction "releases" the patient – in their opinion – from the necessity to verify and confront the beliefs with the objective reality. Beck [4, 5] describes the most common errors of thinking: arbitrary conclusion, selective abstraction, overgeneralization, magnification and minimization, personalization, dichotomous and absolutist thinking. When describing oneself, the patient chooses

extremely negative categories, and shows a tendency to use specific attributions leading to self-blame [5].

In conclusion, Beck's cognitive model describes in detail the psychological properties of the cognitive system of people suffering from depression. In subsequent works, Beck [3] emphasizes the importance of disorders in the functioning of the nervous system, which may be important in the etiology of depression. Figure 2 shows a developed model of depression in which the hyperactivity of the limbic structures and the hypoactivity of the prefrontal cortex are of high significance.

The model based on genetic susceptibility explains the special sensitivity to experienced emotions, overestimating the threat and exaggerating stressful events, which is related to the hyperactivity of the amygdala. On the other hand, the impaired activity of the prefrontal cortex explains the difficulty in changing beliefs based on cognitive restructuring. The basis for working with the patient is the activation of cortical areas

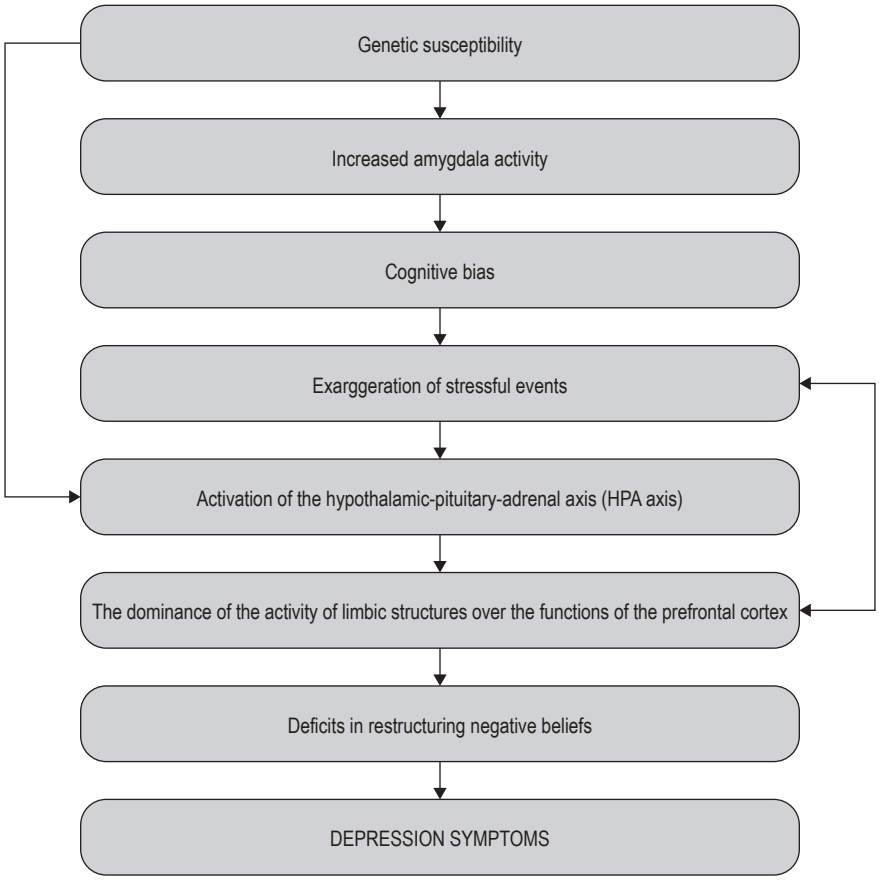

Figure 2. **Model of depression development based on genetic susceptibility [3]**

through the intensification of analysis, rational thinking based on facts and systematic cognitive restructuring, leading to the formulation of alternative thoughts and beliefs. Therapeutic work primarily focuses on cognitive aspects, but the behavioral sphere is also important, as it has a significant impact on the process of restructuring negative beliefs. In this context, behavioral activation is important, as it allows the inclusion of positive reinforcements in the formation of functional beliefs, e.g., in relation to self-efficacy and self-agency.

Development of the cognitive concept of depression

Contemporary research confirms the assumptions of the cognitive theory of depression proposed by Beck in the 1960s and 1970s. Significant extensions were also introduced, which contributed to a more complete understanding of the phenomena and processes occurring in the time preceding the appearance of the depression symptoms and in its course. The results of research on cognitive processes allow to explain the mechanisms of information processing in people suffering from depression.

Negative automatic thoughts and working memory

Negative automatic thoughts (NATs), according to Beck's concept, are mental reactions to situations that are associated with certain expectations, external or internal. The automatic nature of a negative thought lies in the fact that it is accepted without reflection, there is no attempt to analyze or verify the truth of this thought. Negativity stems from the belief that it is impossible to fulfill external or internal expectations. NATs appear in the thinking of the vast majority of people, but only in depressive people do they deteriorate mood more permanently. It has been shown that depressed people and non-depressed people do not differ significantly in their initial responses to negative life events, but differ in the degree of their ability to improve their mood after experiencing sadness or other negative emotion [6]. Joormann [7] explains the psychological mechanism of these differences by describing the cognitive inhibition mechanism. A negative mood, e.g., as a consequence of an unpleasant event, activates cognitive content in working memory in accordance with its emotional tone, i.e., negative, for example, NATs. In difficult situations without depression, this mechanism of cognitive inhibition is most often activated. It consists, on the one hand, in the selection of information, allowing access to the working memory (awareness) only of those that are significant, related to the situation, and, on the other hand, it eliminates the content that is useless in a specific situation. If this mechanism does not function properly, the risk of depression appearing or its symptoms worsening increases, because information that is consistent with a negative mood "gets" into working memory, but is not useful in solving a given situation [7]. In depression, therefore, the inhibition process is disturbed, which limits the access of irrelevant information. The consequence of the disturbed cognitive inhibition mechanism is the deterioration of cognitive functions, especially working memory – the ability to recall the necessary information from long-term memory, as well as maintaining important contents in the field of consciousness [8].

The cerebral mechanisms of punishment and reward

The cognitive model of depression assumes that the traumatic experiences on the basis of which dysfunctional beliefs are formed appear already in the early stages of development. They are the risk factor for developing depression. One of the consequences of an excess of negative events and/or a lack of positive events may be the dysfunction of the brain's reward and punishment systems.

In people with depression, a different behavioral response to rewards and/or punishments was found, as well as impaired functioning of the monoaminergic, frontostriatal reward and punishment systems [9]. More specifically, in people diagnosed with depression, research on anticipating and receiving rewards and punishments showed activation of the dorsal anterior cingulate cortex (dACC) during reward anticipation, while in the control group such an increase was observed when anticipating punishment. Bearing in mind the function of this part of the cingulate cortex, i.e., participation in cognitive conflicts, the authors suggest that patients experience conflict when they anticipate the emergence of positive stimuli [10]. It can therefore be assumed that the resistance to the change of both dysfunctional beliefs and negative automatic thoughts results – probably in part – from the fact that the announcement of something positive, e.g., an opinion or event activating the dACC, triggers a sense of conflict, in fact a discrepancy between the well-established negative beliefs and imaginations, and the possibility of the emergence of positivity. On the psychological side, this creates a sense of cognitive dissonance that can be mitigated or eliminated by negating positivity. Other studies have also shown a significantly lower activation of the left-sided nucleus accumbens and bilateral caudate nuclei in response to receiving a reward compared to the control group [11].

The research results suggest a functional differentiation of the brain's reward and punishment systems in depressive people. However, the question arises as to whether the observed differences are the cause or perhaps the result of depression. The neuroimaging studies by Gotlib et al. [12], in which two groups of girls aged 10–14 participated: (1) 13 girls with mothers diagnosed with depression (group at high risk of developing depression); (2) 13 girls with mothers not diagnosed with depression (low risk group). During the study, none of the girls in both groups had symptoms of depression. It was found that during the anticipation of rewards, high-risk participants showed less activation – compared to low-risk participants – in the putamen and left-sided insular cortex, but more activation in the right-sided insular cortex. When the test subjects experienced punishment, the high-risk group showed greater activation in the dACC, while the low-risk subjects showed greater activation in the caudate nuclei and putamen. In conclusion, the authors emphasize that differences in the functioning of reward and punishment systems in the high-risk group were observed before the potential development of symptoms of depression.

The consequence of dysfunction of the brain mechanisms of punishment and reward may be disturbances in the sphere of predictive processing. Kube et al. [13] suggest that people with depression experience a distortion of the learning process of comparing predictions with actual events and experiences. In depression, there is a tendency

to negative reassessment and to disregard positive information that could undermine negative expectations. This results in persistent negative predictions and a bias in the learning process. Analyzing the neurophysiological correlates of cognitive deficits in depression, Kube et al. [13] suggest that there are brain mechanisms explaining the tendency to expect mostly negative events or experiences and a self-reinforcing negative feedback loop. As the main problem, researchers point to excessive precision in the processing of negative beliefs and errors related to ignoring or weakening positive predictions.

Neural correlates of the cognitive model of depression

In a review of the literature on the neural mechanisms of the cognitive model of depression, Disner et al. [14] present the results of neuroimaging studies that confirm the neural basis of Beck's cognitive concept. According to the researchers, attention bias may result from the difficulty of disengaging attention from aversive stimuli, which is associated with reduced activation in the upper parietal lobe, ventrolateral prefrontal cortex (VLPFC) and dorsolateral prefrontal cortex (DLPFC). The subsequent analyses, presented below, indicate the neural determinants of depression and allow for a more precise explanation of the mechanisms of cognitive distortions typical for depression.

The bias in emotional processing is associated with a particular amygdala reactivity, left DLPFC hypoactivity and right DLPFC hyperactivity. In turn, the inhibition of negative information may be disturbed due to the abnormal activity of rostral anterior cingulate cortex (rACC), which is observed in a group of people suffering from depression. Reduced ability to experience positive affect and lower sensitivity to rewards are associated with decreased activity of the nucleus accumbens and the prefrontal cortex [14].

Thinking bias and a tendency to ruminate are associated with hyperactivity of the functional network including the amygdala, hippocampus, subgenual cingulate cortex, and medial prefrontal cortex (MPFC), as well as altered rACC activity. Neuroimaging and experimental studies have shown that deep brain stimulation, including stimulation of the subgenual anterior cingulate cortex (sgACC) may be helpful in reducing depression symptoms and treating drug-resistant depression [15].

According to Disner et al. [14], impaired memory is related to amygdala hyperactivity, which is positively correlated with the activity of the hippocampus, caudate nucleus and putamen. The different levels of activation in the ventral MPFC during happy and sad memories support the notion that the depressed brain requires less cognitive effort to recall negative events.

Dysfunctional attitudes and negative schemas are associated with decreased connectivity between the dorsal ACC and the limbic system, suggesting a decline in cognitive control. The degree of functional connectivity negatively correlates with the activity of the amygdala, MPFC, and anterior and ventral ACC. This may be suggested by lower regulatory capacity and stronger experience of negative stimuli [14].

Numerous reports from neuroimaging studies confirm the dysfunction of the cortico-limbic areas in people suffering from depression. Siegle et al. [16] found

a decrease in the activity of the prefrontal cortex and an increase in the activity of the amygdala in the majority of depressive patients. Other studies have shown that the reduced connections of the amygdala with the orbitofrontal cortex and the dorsomedial prefrontal cortex can predict the degree of attenuation of negative feelings as a result of the reappraisal technique [17]. Disner et al. [14] concluding a review of neuroimaging studies on depression, conclude that maladaptive thoughts appear to be triggered and sustained by increased bottom-up responsiveness (especially in areas such as the amygdala, hippocampus, subgenual, ventral and rostral ACC) and impaired cognitive control by reducing the effect of top-down brain mechanisms (especially in areas such as the DLPFC, VLPFC, and the dorsal ACC region) on the lower regions of the brain.

Electroencephalographic studies are also an important point of reference in the description of neural correlates of depression. Several studies have shown, inter alia, that symptoms of depression are associated with a reduced positive potential appearing approx. 200–400 ms after an incorrect reaction [18–21]. This potential, referred to as *Pe*, is a neural response to an erroneous response and is related to the process of monitoring actions and consciously recognizing the error [22]. According to the researchers, *Pe* is an indicator of awareness of a mistake, it is also associated with the focus and allocation of attention, which determines the possibility of making corrections and indicates the efficiency of cognitive processes [21]. The reduced *Pe* amplitude in patients with depression may therefore be directly related to attention deficits and indicate weaknesses in executive functions in the context of mistakes.

Neural correlates of depression are also associated with the level of neurotransmitters, which is explained by the serotonin and dopamine hypotheses. Cowen and Browning [23], while still pointing to some doubts in determining the mechanism of changes and cause-effect relationships, refer to the nearly 50-year history of the "serotonin hypothesis," which is one of the dominant theories in the biochemical description of depression. It suggests that the underlying pathophysiology of depression is the decreased activity of the serotonin pathways, which explains the positive therapeutic response to selective serotonin reuptake inhibitors. Similarly, Moncrieff et al. [24] in a recent systematic review of studies indicate that there is no clear evidence of a direct relationship between serotonin levels and depression. Researchers emphasize that linking depression with a biochemical imbalance should therefore still be treated as hypothetical.

According to the "dopamine hypothesis," decreased dopamine transmission has been reported in depressed patients. According to Dunlop and Nemeroff [25], although the results of neuroimaging studies are inconclusive and the biochemical basis of the pathophysiology of depression is still unknown, there is evidence that dopamine transmission dysfunction is the primary cause of some depression subtypes.

The analysis of biochemical correlates of depression also indicates a relationship with the level of cortisol. Bhagwagar et al. [26], examining the level of cortisol at various intervals after waking up, observed significantly higher levels of cortisol in patients with depression. However, they indicated that it is important to capture the right moment of the study – the differences were observed 30 minutes after waking up (in people with depression, the level of cortisol was higher by 25% compared to the

control group), but after 60 minutes since awakening, the differences were no longer significant. Burke et al. [27] showed that in response to psychological stressors, patients with depression exhibit higher cortisol levels after 25 minutes from the exposure of stressors, however, there were no significant differences in the direct response to stressors or in the period up to 25 minutes. Researchers also showed the effect of the time of day – generally, higher baseline cortisol levels in people suffering from depression were observed in the afternoon. The results of these studies point to an important trail in determining the relationship between depression and cortisol, however, they show the complexity of these mechanisms and methodological difficulties in the analysis of the biochemical basis of depression.

At this point, it is worth mentioning the results of studies with animals, which showed that under the conditions of repeated exposure to a stressful stimulus, atrophy of the prefrontal cortex and hippocampus as well as enlargement of the amygdala are observed [28]. More conclusive conclusions are presented by Herbert [29], who directly associates an increased level of cortisol in response to stress with greater susceptibility to depression, emphasizing the important role of genetic determinants, psychological characteristics and individual experiences. According to this author, the existing data indicate the importance of cortisol levels in the development of some types of depression, and an increased level of cortisol is a predictor of the appearance of subsequent episodes of depression. Treatment of depression should take into account general cortisol levels and a disturbance in the natural circadian rhythm of cortisol secretion. Stabilizing or reducing the level of cortisol may significantly affect both the probability of the appearance of subsequent episodes of depression and reduce the long-term consequences of cortisol disorders, such as cognitive decline. Taking into account the relationship of cortisol with the level of stress in an individual [30], it can be assumed that cognitive bias leading to focus on negative content and interpretations may result in chronic, increased levels of stress in people suffering from depression.

Recapitulation

In conclusion, owing to research inspired by the assumptions of the cognitive theory of depression, our knowledge of the mechanisms that cause and sustain symptoms of depression has been significantly enriched. This also applies to the theory itself. The following findings should be emphasized in particular:

1. Research on the role of working memory and the mechanism of cognitive inhibition in maintaining negative mood by preventing or hindering the elimination of negative automatic thoughts (NATs). If the functional efficiency of working memory is impaired, inter alia, due to deficiencies in cognitive inhibition, then the selection of information is weakened – NATs, which are not "screened out" by cognitive inhibition, enter the consciousness. Improving the functions of working memory may be an important element of the correction and elimination of NATs, which can significantly support psychotherapeutic techniques.
2. The cognitive theory of depression has been enriched with knowledge on the functioning of the brain reward and punishment systems. People suffering from

depression show a loss or reduced ability to experience rewards and an increased tendency to experience punishment. The functioning of reward and punishment systems is one of the determinants of the influencing the level of self-esteem – experiencing rewards and the lack of punishments contribute to its positivity and high level, while the lack of rewards and experiencing punishments act the opposite. Cerebral reward and punishment systems function differently in depressed people compared to healthy people. In people with depression, anticipation of reward is associated with activation of the dorsal anterior cingulate cortex (dACC). In contrast, in healthy individuals, the same effect was observed when anticipating punishment. Since the cingulate cortex is involved in the processing of cognitive conflicts, it can be assumed that in depressive people the anticipation of reward causes a conflict (e.g., "The reward cannot happen to me"), while in non-depressed people a conflict occurs when anticipating punishment ("I don't see the reasons to expect punishment"). Knowledge about different brain mechanisms of reward and punishment processing may be an important element of psychoeducation in the therapeutic work of people suffering from depression.

3. The results of research with the use of non-invasive methods of brain research indicate that the majority of cognitive anomalies observed in depression can be correlated with dysfunctions of specific brain structures and their functional connectivity. The attention bias associated with persistent focus on negativity correlates with decreased activity in the upper parietal lobe and the ventrolateral and dorsolateral prefrontal cortex. Cognitive bias and a tendency to ruminate are related to the hyperactivity of the functional network of the amygdala, hippocampus, part of the cingulate cortex, and the medial prefrontal cortex. It has been shown that the "depressed brain" requires less cognitive effort to recall negative content. Probably the key role in the development and maintenance of cognitive deformations in depression is played by the prefrontal cortex and its interactions with subcortical structures. Monitoring changes in brain activity during treatment would be particularly important for further research on depression.

In the perspective of future studies on the cognitive aspects of depression, research using neuroimaging and experimental procedures are particularly promising. The use of cognitive tasks varied both in terms of content and complexity will allow for a deeper understanding of the mechanisms and specificity of information processing in depression.

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