Review

Brain-Based Formulation of Cognitive Behavioral Therapy: A Compass of Therapists across the Brain Ocean

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Brain-Based Formulation of Cognitive Behavioral Therapy: A Compass of Therapists across the Brain Ocean

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Abstract: Although there is a vast body of neuroscientific research on cognitive behavioral therapy (CBT), many therapists are confused about how to incorporate the evidence into their clinical practice. This narrative review translates neuroscientific research on CBT for depression into a therapist-friendly formulation model. Four brain networks that are particularly relevant to depression, including the Central Executive Network, Default mode Network, Salience Network, and Affective Network, were identified based on their interactions and described in relation to depressive symptoms. Additionally, it clarified how each CBT technique works on a brain map. Assessment, intervention selection, and patient psychoeducation based on this model will facilitate the clinical application of neuroscience in CBT.

Keywords: depression; cognitive behavioral therapy; Brain Network Model; formulation

1. Introduction

Cognitive behavioral therapy (CBT) has long been the cornerstone of psychotherapy for a wide range of psychological disorders, such as depression [1,2], anxiety disorders [3,4], insomnia [5,6], and pain [7]. The CBT focuses on identifying and modifying the maladaptive thought patterns and behaviors believed to underlie and perpetuate these conditions. However, the objectivity and reproducibility of these action processes based on psychological hypotheses are still in doubt [8]. How to resolve this issue is an ongoing discussion among experts in the fields of neuropsychiatry and clinical psychology [9–11].

Recently, a vast body of evidence has shown that the CBT functions in the brain. Meta-analysis has been employed to examine the cross-sectional neural effects of CBT in patients with various psychiatric disorders, including major depressive, anxiety, and eating disorders [10]. No significant changes have been found in the emotional task; however, cognitive-task functional magnetic resonance imaging shows that the activation of the left anterior cingulate gyrus, left middle frontal gyrus, and left cuneus decreases from pre-CBT to post-CBT. Additionally, the reviewed literature has found that the CBT-affected regions overlap in the central executive network (CEN), default mode network (DMN), and salience network (SN). Other meta-analyses and systematic reviews have shown that these regions are involved in neural effects and are predictors of CBT. [12–17]. However, many CBT therapists fail to incorporate neuroscientific evidence into their practices. Although all aspects of the mind are generated by the brain, CBT therapists underestimate the relationship between the brain and the treatments they use. An overview of these neuroscientific studies is insufficient; we intend to translate this evidence into clinical practice.

1.1. What is available neuroscience integration for therapists?

An emerging trend in the integration of neuroscience into CBT practice is the incorporation of therapeutic techniques based on neuroscience principles [11]. Current studies indicate that the combination of the stimulation or modulation of specific brain regions, such as transcranial direct current stimulation (tDCS), transcranial magnetic stimulation (TMS), and neurofeedback, can

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enhance the efficacy of CBT [11]. The scope of this application varies, and a recent review has discussed the evidence for the effectiveness of CBT in combination with brain stimulation methods, such as tDCS and TMS, for addiction [18]. However, the practical application of such interventions has been hindered by cost considerations, which have limited widespread interest in clinical practice. Psychological therapists, particularly those delivering CBT outside medical settings and lacking training in advanced measurement techniques, may face substantial resistance to implementing these approaches.

Neurocounseling and neuroeducation can aid clinical activities with neuroevidence. Neurocounseling is defined as "the integration of neuroscience into the practice of counseling by teaching and illustrating the physiological underpinnings of many of our mental health concerns" [19]. This assistance extends beyond therapists to patients and others and is expected to reduce stigma and shame about their illnesses [20]. Additionally, neurological mechanisms can be associated with psychosocial issues, as shown in existing neuroscientific evidence, allowing the utilization of talk therapy to work with specific brain regions. Furthermore, neuroeducation is partially consistent with the definition of neurocounseling, which is strictly defined as "a didactic or experiential-based intervention that aims to reduce client distress and improve client outcome by helping clients understand the neurological processes underlying mental functioning" [21]. These two approaches have the greatest advantage of not requiring special equipment or training while incorporating neuroscience into the traditional counseling process. Therefore, even therapists without such resources can immediately apply neuroscience in their clinical practice. Educating individuals about the neurological basis of psychosocial issues can empower them to understand their conditions and treatment options. This may reduce hesitance to seek help and contribute to effective evidence-based interventions.

1.2. Our goals

Currently, the neurological evidence on cognitive-behavioral therapy is understood in terms of individual techniques. To significantly utilize the evidence in real-world practice, a comprehensive model is required to aid in case formulation, including assessment and treatment selection. Knowing how individual techniques integrate and relate to each other on the platform of the brain allows for flexibility in selecting and combining techniques for therapists. The primary aim of this narrative review is to provide an extensive overview of the neurological evidence, with a particular emphasis on brain imaging, for the application of CBT in the treatment of depression. Furthermore, this review proposes a comprehensive model for integrating neurological findings into the CBT practice. By elucidating the neurological mechanisms and their relationship with CBT techniques, this model aims to enhance the psychotherapeutic process, ultimately contributing to improved outcomes in individuals with depression.

2. Three-Core-Network Model for Depression

Specific brain lesions that engage in depression or depression-related behaviors have been found in various regions [22–24]. The current mainstream recognizes depression as a result of dysfunctional brain networks based on the interactions between anatomically distant brain regions [25,26]. Many reviews and studies have focused on three core networks, called the triple network model [27–32]. These consist of the CEN, DMN, and SN. It is well known in the field of depression, as well as in general cognitive science, that the interaction of these three networks contributes to various cognitive aspects of a human being [33,34]. The involvement of these networks in depression has already been described in the literature, as described above; therefore, this paper summarizes them to the extent that CBT therapists can easily share them with their patients.

The CEN, also called the frontoparietal network or cognitive control network, is responsible for cognitive/executive functions, such as goal-oriented action planning, active control of attention, cognitive flexibility, logical judgment, and decision-making. These are closely related to problem-solving and self-control skills in the context of CBT, i.e., skills related to adaptive cognitive-emotional control and active cognitive-behavioral control. As is known from the classical brain model of

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depression [35], reduced CEN activity has been consistently reported in patients with depression. The lateral prefrontal and lateral posterior parietal cortices are the central regions, and abnormal functional connectivity between these regions is strongly associated with severity, executive function, and diagnostic markers [36–38].

The DMN is a medial midline network that is activated in the resting state, with the medial prefrontal cortex and posterior cingulate cortex/precuneus as the central regions. These brain regions are involved in the processing of the self and contribute to introspection and autobiographical memory. DMN activity is stronger at rest in individuals with depression than in healthy individuals [39], suggesting that the DMN induces depression-specific states of introspection, such as rumination [40]. As self-recognition and rumination are frequent targets of CBT for depression, it is important to understand the involvement of the DMN in these symptoms. DMN activity often contrasts with that of the CEN. It is necessary to switch the CEN/DMN appropriately during on/off tasks [41]. Patients with difficulty performing tasks with the CEN are thought to have strong DMN activity and self-referential processes that interfere with task performance [42,43].

The SN detects salient stimuli and events to guide behavior. Within this central region, the insular cortex is responsible for switching between the CEN and DMN, whereas the dorsal anterior cingulate cortex is associated with monitoring and selecting behaviors [44]. In depression, the SN works with the affective network (AN) to enhance negative information processing and enforces adherence to self-referential processes through strong functional coupling with the DMN [45]. In particular, the transmission system from the DMN to the SN may underlie the negative bias observed in depression [46]. Therefore, the SN is the center of interactions between other networks, facilitating self-control [47]. Therapists can suspect SN lesions in patients with negative biases in emotion, attention, and memory as well as attention rigidity [48].

The CEN, DMN, and SN reflect self-control, self-related thoughts/memory, and passive attention/processing bias, respectively, in depression. It is important to understand these symptomatic concepts comprehensively and not independently. If your patients report that they are always caught up in negative thoughts and always focus on them but are not aware of other sources of information in the environment, you may be aware of the possibility that these three networks are interdependent and dysfunctional. This is because the brain of the patient keeps the SN overly attentive to DMN activity, and the persistent DMN activity interferes with the utilization of the CEN. The papers cited above and related reviews [27,49,50] provide readers with further insight into the relationship between the three networks and depression.

3. Extensions to the Four-Network Model

Depressed mood is a core symptom, and depression is referred to as a mood/affective disorder. Therefore, it is essential to incorporate the AN into the three-network model of depression. The first depression model that focuses on the interactions between brain networks comprises an AN [25]. A recent study comparing the neural effects of CBT and pharmacotherapy has evaluated the AN, as well as the CEN, DMN, and SN [51]. These four networks are recognized as fundamental components in modeling neuropathology and CBT mechanisms related to depression [52]. The AN is a complex network of brain regions responsible for processing and regulating emotions [53]. The key components include the amygdala, ventromedial prefrontal cortex, and orbitofrontal cortex. The AN is vital in shaping emotional responses, affective learning, and emotional regulation. Many researchers have reported that the AN in patients with depression often exhibits abnormalities in activity and connectivity, leading to a heightened experience of negative emotions and impaired emotional regulation [54,55]. The importance of the AN in relation to CBT for depression has been reported primarily in discussions on treatment responsiveness [56]. For example, both selective serotonin reuptake inhibitors and CBT reduce hyperarousal in the AN and SN in response to emotional stimuli, and the level of activity in these brain regions before treatment predicts the degree of improvement in symptoms [57]. Interestingly, the degree of enhancement in these brain regions did not necessarily match the extent of symptom improvement. Additionally, extreme AN overactivity is not successfully improved by CBT. Among patients with depression, non-responders

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to treatment exhibit high hyperactivity in the right amygdala before treatment [58]. Thus, many depression treatment guidelines recommend CBT for mild to moderate depression. Although the AN is a critical component in the success or failure of a treatment, direct improvement with CBT may be difficult to achieve. Therefore, CBT therapists should develop strategies based on an integrated model that includes the other three networks to effectively calm the AN or improve it indirectly through interactions with networks other than the AN.

Here, before incorporating the specific techniques of CBT into the model, I first consider how AN can be integrated into the three networks. The cross-inhibitory relationship between AN and CEN is well known; for emotional responses via bottom-up processing in AN, top-down processing occurs in the CEN (particularly on the left side) for cognitive reappraisal via processing works, and emotion is appropriately regulated [59,60]. Patients with depression exhibit both CEN hypoactivity and AN hyperactivity, making emotion regulation difficult [35,61,62]. The cooperative activation of the AN and SN during exposure to negative situations enhances the detection of negative stimuli and emotions and evokes a strong negative effect [63]. It has even been reported that functional connectivity between the AN and SN predicts a relapse of depression that has been in remission [64]. The relationship between the AN and DMN has been modeled as having low associations with the initial depression network models [25]. In the area of social cognitive science, there is an "extended social-affective default mode network" that adds AN regions, such as the amygdala and hippocampus, to the DMN [65]. It is primarily implicated in empathy based on self-reference in the DMN and emotional responses in the AN [66]. However, the association between DMN–AN interaction and depression remains unclear.

Based on these findings, a four-network model is proposed (Figure 1A). This chart can be used by therapists to obtain visual information regarding the interactions between networks. When neighboring networks oppose each other (or are independent), the health of an individual is good. Even if the AN responds temporarily to a negative situation, the CEN inhibits it, and the SN discourages it from looking for similar negative stimuli. As mentioned in Section 2, switching between the CEN and DMN according to the on/off task assists in appropriate cognitive activity. The SN is responsible for the switching; therefore, the excessive binding of the SN and DMN is not recommended [45,67]. Contrasting results have been observed in patients with depression (Figure 1B). The AN responsible for the core symptoms is overly aroused and invades other network areas to create an emotionally dominant state. This is also known as "amygdala hijacking" as an alert response to anxiety and fear. The collaboration between the AN and SN produces a cascade of negative moods and attention [63]. The CEN, which is supposed to stop that flare-up, is underpowered, and proper emotional regulation is lost [61]. Thereafter, the SN works with the DMN to increase attention to negative introspective thoughts and further interfere with CEN activity in task performance situations [42,43,68]. Therapists can use this model as a basis for assessing the brain state of a patient in correspondence with the characteristic symptoms of the patient.

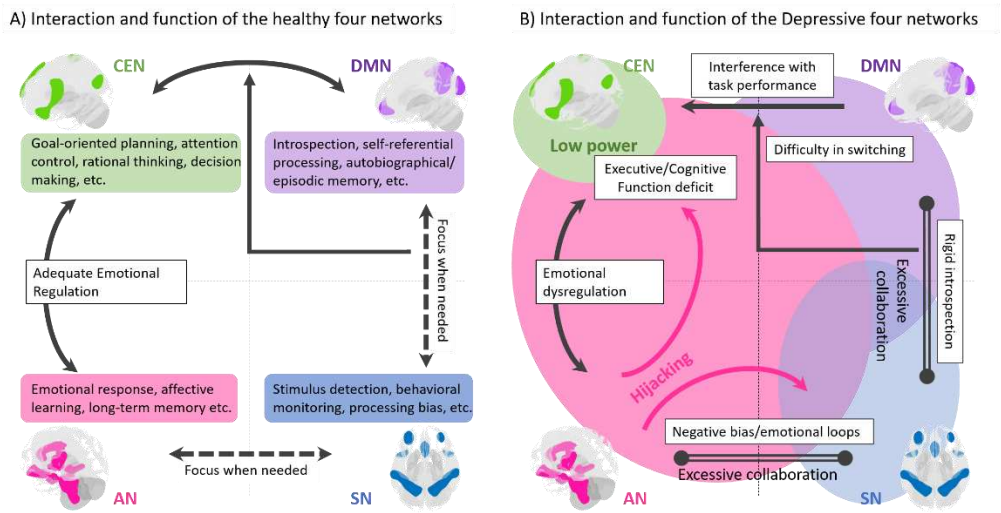

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Figure 1. Four-network model and formulation of depression based on it. Solid double arrows represent bidirectional controlling/opposing relationships. Solid single arrows represent instructive effects. Dashed arrows connect independently or only when necessary. Double lines with circles represent excessive linkages/collaborative relationships. (A): All brain networks are well controlled. The boxed descriptions are functions for each network and each other. (B): Typical brain network for depression. CEN: Central executive network; DMN: Default mode network; AN: Affective Network; SN: Salience Network.

4. Integration of Cognitive Behavioral Therapy Techniques into the Four-Network Model

A comprehensive model that elucidates how individual CBT techniques integrate and relate to each other within the brain framework can provide therapists with high flexibility in choosing and combining techniques for individual patients.

Many studies have summarized that CBT for depression improves the emotional regulation system of the CEN–AN. In particular, it improves the activity level of the CEN and its ability to regulate top-down processes [69,70]. These studies have implemented Beck's cognitive therapy packages consisting of cognitive-behavioral techniques centered on cognitive reconstruction. In this technique, the anterior cingulate cortex (ACC) region leading from the CEN to the AN is considerably altered [12]. Surprisingly, the recovery of the ACC with respect to emotional regulation is a common neural mechanism in other psychotherapies, such as dynamics psychotherapy [13]. Given the functions of the CEN, it is expected that improved problem-solving skills will further bolster this network. Although few studies have been done, there is a recent report that problem-solving training improves the function of the DMN and the attention span of the CEN [71]. Furthermore, rumination-focused CBT, which targets repetitive negative thoughts, such as rumination, works specifically on the dorsolateral prefrontal cortex of the CEN [72,73]. Additionally, it appears to normalize connectivity between the CEN and DMN [73]. Behavioral activation, which supports increased reward-taking behavior, promotes improved regulation skills in the CEN when anticipating unpleasant outcomes [74] and metacognitive skills in the DMN region [75]. Additionally, this approach reduces the functional connectivity between the DMN and SN [45] and within the DMN [76,77]. Mindfulness-based CBT controls attention and promotes the appropriate disruption of self-referential processing. This reduces functional connectivity within the DMN more than behavioral activation [77], and the DMN is associated with long-term effects [78]. Rumination leads to a decrease in SN connectivity and improves sustained attention function [79]. In internetwork interactions, functional connectivity of the SN–DMN and SN–CEN has been shown to increase [80]. In other words, mindfulness interventions may enhance the switching between these networks through the SN function.

To address these findings, each CBT technique was placed in a four-network model (Figure 2). This model can serve as a roadmap for therapists, helping them make informed decisions regarding the most effective approaches for specific cases. When developing a treatment plan with a patient to gain control of rumination, we could discuss whether to (a) inhibit the DMN to decrease the occurrence of rumination, (b) engage the SN to facilitate a break from rumination, or (c) activate the CEN to successfully perform cognitive tasks as an alternative to rumination.

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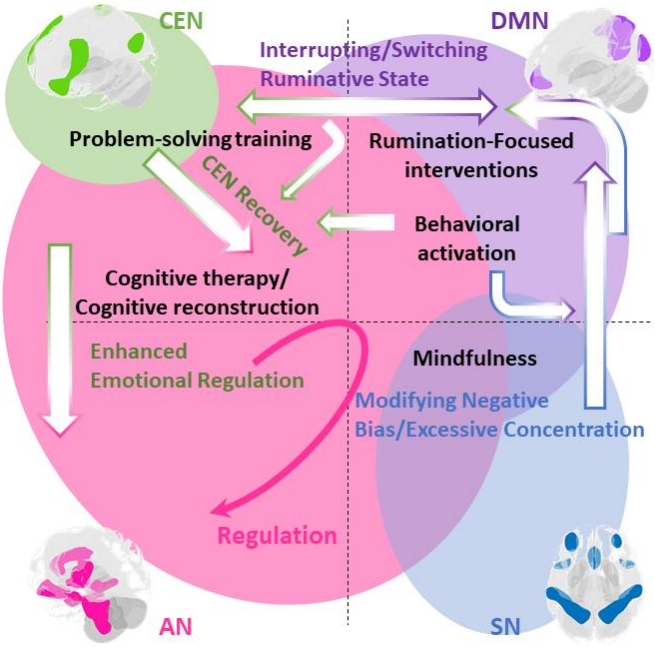

Figure 2. Therapeutic action formulation of cognitive behavioral therapy (CBT) on a four-network model of depression. Each CBT technique, shown in bold, is arranged with respect to the brain network on which it acts. The arrows beside the techniques indicate the pathway of action of each treatment. CEN: Central executive network; DMN: Default mode network; AN: Affective Network; SN: Salience Network.

5. Utilizing the Four-Network Model beyond Cognitive Behavioral Therapy

CBT therapists are typically unable to strategize how other specialty treatments, including pharmacotherapy and neurological modulation, can be linked to their treatment strategies. The four-network model can help facilitate their understanding and collaboration (Figure 3). Pharmacotherapy is considered for both acute and extreme AN activity. While this is a conventional guideline-based treatment, the CBT therapist can provide rich information to the patient about why the strategy is necessary using the brain model [81]. Antidepressants affect AN bottom-up responses and relatively broad networks, whereas CBT enhances top-down control, starting with the CEN [70,82]. Additionally, neurofeedback may be used to work on brain regions or networks where CBT has difficulty reaching to augment the neural effects of CBT. Many neurofeedback studies on depression have aimed to promote AN self-regulation [83]. This may be used in combination with the bottom-up therapeutic effects of the AN. Amygdala neurofeedback before CBT increases the concentration on positive thoughts and behaviors and results in high rates of long-term remission [56]. Additionally, patients and their therapists can consider incorporating exercises to stimulate the CEN before starting CBT. Brief cognitive tasks that use working memory [84] and verbal productive functions [85] can specifically stimulate these areas. A rich counseling technique for targeting specific brain regions has been consolidated [20]. Many brain stimulation therapies (neuromodulation), such as TMS, have the potential for antidepressant effects via the CEN [86]. For patients receiving multiple treatments, the integration of each treatment action into a four-network model is educational.

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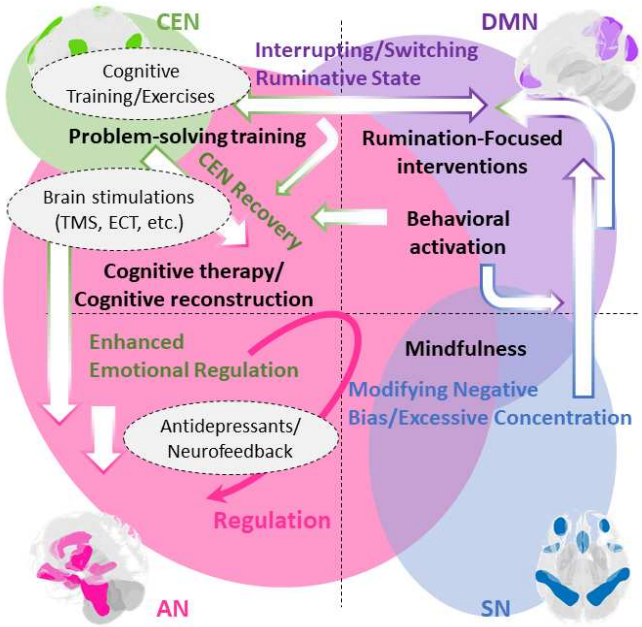

Figure 3. Planning in conjunction with other treatments. Cognitive training and exercise promote the activation of the CEN. Brain stimulation therapies produce antidepressant effects on other networks via the CEN. Antidepressants and neurofeedback can selectively work on brain networks that CBT cannot reach. These can be deployed flexibly, according to the type of training and the brain region targeted. CEN: Central executive network; DMN: Default mode network; AN: Affective Network; SN: Salience Network.

6. Conclusions

This narrative review attempts to bridge the gap between the psychological foundations of CBT and emerging neuroscientific evidence. By providing a deep understanding of the neurological underpinnings of CBT, we aspire to pave the way for an effective and evidence-based approach to the treatment of depression and other related conditions within the domains of neuropsychiatry and clinical psychology. Thus, this paper highlights the connections between neurocounseling and neuroeducation, which are more practical than the neuromodulation treatments that have already been discussed. Many CBT therapists are aware of the growing body of neuroscientific evidence; however, they are unsure of how to utilize it in real clinical situations. This study presents a visual compass to guide therapists as they navigate an uncharted brain ocean.

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Abbreviations

CBT: Cognitive Behavioral Therapy
CEN: Central Executive Network
DMN: Default Mode Network
SN: Salience Network
tDCS: transcranial Direct Current Stimulation
TMS: Transcranial Magnetic Stimulation

AN: Affective Network
ACC: Anterior Cingulate Cortex

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