Examining the Effect of Cognitive Behavioral Therapy on Anxiety Disorders from a Neurological Perspective

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Abstract. Cognitive Behavioral Therapy (CBT) stands out as an effective approach for addressing anxiety disorders. The advancement of neuroimaging technologies, including Magnetic Resonance Imaging (MRI), Positron Emission Tomography (PET), and Functional Magnetic Resonance Imaging (fMRI), has provided scientists with invaluable tools to delve into the study of mental disorders. This essay aims to explore recent evidence supporting the neurological basis of CBT’s effectiveness in treating anxiety disorders. Beginning with an introduction to the concept of emotion regulation and cognitive fear structure, this essay proceeds to analyze experimental results, elucidating the fundamental mechanisms underlying anxiety disorders and presenting compelling scientific evidence attesting to the efficacy of CBT.

Keywords: Anxiety Disorder, Cognitive Behavioral Therapy, Emotion Regulation, Emotion Processing Theory, Neuroimaging, Neuroscience.

1. Introduction

Anxiety is a normal emotional reaction to stressors. It is commonly triggered by the fear of future events, characterized by “recurring intrusive thoughts or concerns” accompanied by physical symptoms such as “sweating, trembling, dizziness, or a rapid heartbeat” (American Psychiatric Association, 2022). However, if anxiety, which is supposed to be a transient emotion, becomes overwhelming and uncontrollable, it can lead to a mental illness known as anxiety disorder. Anxiety disorder, as the DSM-V defines it, involves “excessive worry and anxiety” that leads to significant impairment in daily functioning (American Psychiatric Association, 2013). Fortunately, cognitive behavioral therapy (CBT) has proven efficacious in treating anxiety disorders (Carpenter, 2018). The principle behind CBT is based on thoughts, behavior, and feelings interfering with each other, and overall, they influence people’s mental health. CBT aims to find and change behavioral or thinking patterns that lead to mental illnesses or intensify symptoms, and, in doing so, it improves subjects’ ability to control their thoughts and enhance their well-being (Informed Health, 2016). Along with integrating medical models, scientists in clinical psychology became increasingly interested in understanding the neural mechanism of psychopathologies in the 1970s. Exploring the neuroplastic changes in the brain caused by psychotherapy can help identify neural markers that predict the outcome of therapies and the biological bases of mental disorders (Barsaglini et al., 2014). However, because of the lack of neuroscientific technologies researchers could not study the neural mechanisms underlying mental illnesses until the late twentieth century onwards with the invention of neuroimaging technologies, including magnetic resonance imaging (MRI), positron emission tomography (PET), and functional magnetic resonance imaging (fMRI). These technologies allow researchers to investigate psychopathologies with a non-invasive, ethical approach. This essay intends to review CBT’s effects on anxiety disorder by examining articles that are focused on CBT-induced neurological changes in the brain.

To sustain proper cognitive functioning, the ability to de-escalate negative emotions is vital. Emotion regulation (ER) is the attempt to influence one’s emotions, which are defined as “time-limited, situationally bound, and valenced [sic] states” (McRae, 2020). The presently accepted model of emotion generation introduces the following stages: experiencing situations, attending to situations, appraising the situations based on “active goals”, and experiencing “experiential, physiological, and/or behavioral responses” to the situation (McRae, 2020). Research in this field has been focused
on how personal emotions are regulated, and one subtype of this intrinsic regulation is cognitive reappraisal, which is the most well-studied emotional mediating strategy. As Uusberg (2019) mentioned in the article, interests in cognitive reappraisal—the intention to reinterpret a situation to change the “trajectory of emotion” with purposes—can be derived from ego-defense mechanisms in psychoanalysis. Cognitive reappraisal involves “changing situational construal and goal set[s]” (Uusberg, 2019), and thus, one will experience different emotional responses to the same situation and regulate emotions. However, people who are exposed to anxiety disorders experience difficulties in applying emotional regulation, as described above (Uusberg, 2019).

![Figure 1 Image from Uusberg, 2019. Comparison of the resulting emotional responses before and after emotion regulation via reappraisal.](image)

Emotional processing theory explains the deficient emotion regulation skills in anxiety disorders. Based on emotional processing theory, fear information is stored in the associative networks, also known as cognitive fear structures. Together with fear responses, such as escape, avoidance, psychophysiological responses, and subjective interpretation of the stimuli (e.g., tiger = danger), fear information, fear responses, and interpretation of the stimuli assemble the fear structure, which is activated when encountering a stimulus that resembles the feared cue. The fear structure is maladaptive when it is falsely activated, meaning a non-fear stimulus triggers the fear structure (Antonia, 2015). Inhibitory functioning in the brain plays a crucial role in living a normal life in being able to regulate unwanted thoughts, impulsive actions, and emotions. When encountering interfering stimuli in real life, the ability to suppress or avoid irrelevant and unnecessary stimuli is fundamental to the normal thinking process (Garavan, 1999). Failure to inhibit “recurring intrusive thoughts or concerns” (Garavan, 1999) once the fear structure is mistakenly activated is one of the potential causes of anxiety disorder. Studies have also shown that processing fearful emotional stimuli activates the amygdala bilaterally, whereas labeling the emotions leads to increased activities in the right prefrontal cortex and decreased responses in the amygdala (De Raedt, 2006).

As Dr. Amit Etkin (2012) states, within the category of anxiety disorder, there is a distinction between “fear” disorders and “anxious/misery” disorders. “Fear” disorders are marked by “exaggerated reactivity” to fear cues, such as social anxiety disorder (SAD) and agoraphobia, whereas
“anxious/misery” disorders are characterized by non-contingent, wide-ranging anticipatory anxiety, such as generalized anxiety disorder (GAD) (Etkin, 2012). Despite the subtle differences between “fear” disorders and “anxious/misery” disorders, anxiety disorders including PTSD, SAD, and specific phobias are characterized by the same neural abnormalities—hyper-activation of the limbic regions: amygdala and insula (Etkin, 2012). Meanwhile, recent meta-analyses show that CBT may be effective in treating anxiety-related disorders by increasing prefrontal control of subcortical structures (Brooks et al., 2022). The interconnectivity between the prefrontal cortex and the amygdala is the basic principle of CBT, which explains the importance of using neuroimaging technologies in investigating CBT’s effects. Neuroimaging technologies can provide structural, precise, and quantitative data simultaneously while monitoring brain activities. For example, in the study Bomyea et al. conducted in 2020, researchers recruited 30 participants with panic disorder or generalized anxiety disorder, asking them to complete a reappraisal-based emotion regulation task while having their prefrontal and limbic regions fMRI scans taken before and after a CBT session. Aligning with Brooks’ (2022) findings on CBT’s effects, Bomyea et al. (2020) registered an increase in blood-oxygen-level dependent signal based on fMRI data from the prefrontal cortex, as well as an activity decrease in limbic regions associated with reduced anxiety symptoms. Based on the empirical evidence that shows neuronal changes triggered by CBT, scientists have illustrated the counterbalancing relationship between the prefrontal cortex and the amygdala mentioned in the previous paragraph, as well as the effectiveness of CBT.

In summary, this essay reviews the recent evidence which provides an informative picture of the effectiveness of therapy on mental illness; specifically, cognitive behavioral therapy’s effect on anxiety disorders. It approaches the main argument—CBT is effective in treating anxiety related disorders—in a progressive way: from explaining the processing model of emotion regulation to the dysfunctional fear structures and eventually to the concrete evidence that CBT alters neural activities. The counterbalancing relationships between prefrontal cortex and amygdala imply that, when targeting anxiety related disorders, CBT can focus on enhancing prefrontal cortex activities. Because of the high comorbidity and heterogeneity of psychopathologies, individual different responses to CBT entail finding neural markers that can predict the result of the therapy. Based on empirical evidence, cognitive reappraisal is analogous to extinction learning (Wang, 2023). Future research should focus more on how CBT fosters extinction learning via cognitive reappraisal and the neural representation of stabilized extinction learning neural circuits.

References


