Review Article

Borderline Personality Disorder and Neuroplasticity: A Brief Review of Relevant Studies

Abstract

Borderline Personality Disorder (BPD) is a challenging and complex mental health disorder characterized by emotional dysregulation, impulsivity, unstable relationships, and a poor sense of self. Neuroplasticity, the brain's ability to change and adapt in response to experiences, is impaired in individuals with BPD, specifically in the prefrontal cortex, amygdala, and hippocampus. This impairment has been linked to emotional dysregulation, a core symptom of the disorder. Interventions aimed at improving neuroplasticity hold promise as a treatment target for BPD. Current evidence suggests that psychotherapeutic interventions, such as Dialectical Behavioral Therapy (DBT), may improve neural plasticity in the brain regions associated with emotional dysregulation and may result in symptom reduction and improved functioning in individuals with BPD. However, much more research is needed to better understand the relationship between neuroplasticity and BPD, as well as to develop more targeted and effective interventions. With continued research in this area, it is hoped that improved understanding of the role of neuroplasticity in BPD will lead to the development of more effective treatments for this challenging disorder.

Keywords: Borderline Personality Disorder, Neuroplasticity, Emotional dysregulation, Psychotherapy, Treatment interventions

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1. Introduction

Borderline personality disorder (BPD) is a complex and often debilitating mental disorder that affects approximately 1-2% of the general population (American Psychiatric Association, 2013). Individuals with BPD experience intense and unstable emotions, have difficulty regulating their emotions, and often struggle with interpersonal relationships (Biskin, Paris, & Renaud, 2012). The disorder is also associated with other symptoms such as impulsive behavior, self-harm, and distorted self-image (Koenigsberg et al., 2002). These symptoms can have a profound impact on an individual's social and occupational functioning, and in severe cases, can lead to hospitalization or even suicide (Zanarini, Frankenburg, Hennen, & Silk, 2005).

Despite extensive research, the etiology of BPD remains unclear. It is thought to be a multifactorial disorder with both environmental and genetic factors contributing to its development (Torgersen, 2000). Recent studies have suggested that neuroplasticity, the brain's ability to change in response to experience, may play a crucial role in the development and maintenance of BPD.

Neuroplasticity is a fundamental mechanism that underlies learning and memory, and it is essential for the brain to adapt to new situations and environments (Buonomano & Merzenich, 1998). In the context of BPD, researchers have investigated how neuroplasticity may contribute to the characteristic symptoms of the disorder.

Studies have shown that individuals with BPD exhibit structural and functional abnormalities in specific brain regions associated with emotional regulation, social cognition, and memory formation (Jovev et al., 2013; Ruocco, Medaglia, Tinker, Ayaz, & Ayaz, 2015). These abnormalities suggest that impaired neuroplasticity may be a key feature of the disorder. In particular, the prefrontal cortex, amygdala, and hippocampus have been identified as brain regions that are affected in individuals with BPD (Svoboda, Stuchlík, Raboch, & Sechko, 2020).

The prefrontal cortex, located in the front part of the brain, is involved in a range of functions including decision-making, impulse control, and emotional regulation (Diorio, Viau, & Meaney, 1993). The amygdala, located in the temporal lobe, is involved in the processing of emotional information (Phelps, 2006). The hippocampus, located in the medial temporal lobe, is involved in memory formation and retrieval (Eichenbaum, 2017). Impairments in these brain regions may contribute to the emotional dysregulation and other symptoms seen in BPD.

Further research is needed to understand the role of neuroplasticity in BPD. This includes investigating potential causes of impaired neuroplasticity, such as early life stress and genetic factors (Liberzon & Abelson, 2016). Additionally, research is needed to explore the potential for neuroplasticity-based interventions to treat BPD.

In this review, we will provide an overview of the current understanding of the role of neuroplasticity in BPD. Specifically, we will discuss impaired neuroplasticity in individuals with BPD, theories on the role of neuroplasticity in the disorder, and neuroplasticity-based

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interventions for BPD. By exploring this topic, we hope to highlight the potential for neuroplasticity as a target for the treatment of BPD.

In this review, we will explore the role of neuroplasticity in Borderline Personality Disorder (BPD). We will begin by discussing the current understanding of impaired neuroplasticity in individuals with BPD, including the structural and functional abnormalities seen in specific brain regions associated with emotional regulation, social cognition, and memory formation.

We will then delve into theories on the role of neuroplasticity in the development and maintenance of BPD, exploring different perspectives on how impaired neuroplasticity may contribute to the disorder. We will consider the role of early life stress, epigenetics, and altered neurotransmitter systems in the development of BPD.

Finally, we will review various neuroplasticity-based interventions for BPD, including psychotherapy, pharmacotherapy, and non-invasive brain stimulation techniques such as transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (tDCS). By examining the potential of neuroplasticity as a target for interventions, we hope to provide a comprehensive understanding of the pathophysiology of BPD and the possibilities for treatment.

2. Neuroplasticity in BPD

Neuroplasticity refers to the brain's ability to change its structure and function in response to environmental and experiential stimuli (Pascual-Leone, Amedi, Fregni, & Merabet, 2005). This plasticity can occur at different levels of the brain, from molecular and cellular changes to alterations in neural circuits and whole brain regions (Doidge, 2007). Neuroplasticity is thought to be a fundamental property of the brain, enabling us to learn, adapt, and recover from injury or disease.

The mechanisms of neuroplasticity involve a range of cellular and molecular processes, including changes in synaptic strength, the growth and pruning of dendritic spines, and the formation of new neurons and glia (Liu, Wong-Riley, & Liu, 2012). These changes are driven by neural activity, which triggers a cascade of signaling pathways that lead to alterations in gene expression and protein synthesis. Over time, these changes can result in modifications to neural circuits and the overall organization of the brain.

There are different types of neuroplasticity, including experience-dependent plasticity, which occurs in response to specific stimuli or experiences, and homeostatic plasticity, which maintains the overall stability and balance of neural activity (Turrigiano, 2011). Experience-dependent plasticity includes forms of learning and memory, such as long-term potentiation (LTP) and long-term depression (LTD), which involve the strengthening or weakening of synaptic connections between neurons (Bliss & Collingridge, 1993). These changes can be transient or long-lasting and can occur in response to a wide range of stimuli, from sensory inputs to emotional experiences.

Neuroplasticity is thought to be important for the development and function of neural networks that underlie various cognitive and behavioral processes, including perception, attention, decision-making, and emotion regulation (Draganski & May, 2008). It is also believed to play a

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role in the recovery of function after brain injury or disease, such as stroke, traumatic brain injury, and neurodegenerative disorders (Doidge, 2007).

In recent years, researchers have increasingly focused on the role of neuroplasticity in psychiatric disorders, including BPD. Studies have shown that individuals with BPD exhibit structural and functional abnormalities in specific brain regions associated with emotional regulation, social cognition, and memory formation (Jovev et al., 2013; Ruocco, Medaglia, Tinker, Ayaz, & Ayaz, 2015). These abnormalities suggest that impaired neuroplasticity may be a key feature of the disorder.

In conclusion, neuroplasticity is a complex and dynamic process that underlies the brain's ability to adapt and change in response to environmental and experiential stimuli. It involves a range of cellular and molecular mechanisms that can result in modifications to neural circuits and the overall organization of the brain. Neuroplasticity is thought to be fundamental to learning, adaptation, and recovery after injury or disease. In BPD, impaired neuroplasticity may contribute to the emotional dysregulation and other symptoms seen in the disorder. Further research is needed to fully understand the role of neuroplasticity in BPD and to develop effective neuroplasticity-based interventions for the disorder.

2.2 Impaired neuroplasticity

BPD is a severe and chronic psychiatric disorder that is characterized by emotional dysregulation, impulsivity, and difficulties in interpersonal relationships (American Psychiatric Association, 2013). The etiology of BPD is not well understood, but it is thought to be a complex interaction between genetic, environmental, and neurobiological factors. In recent years, there has been increasing evidence that impaired neuroplasticity may be an important factor in the development and maintenance of BPD (Jovev et al., 2013).

Studies have shown that individuals with BPD exhibit structural and functional abnormalities in specific brain regions associated with emotional regulation, social cognition, and memory formation (Jovev et al., 2013; Ruocco et al., 2015). These abnormalities suggest that the neuroplasticity mechanisms responsible for shaping these brain regions may be impaired in individuals with BPD. In particular, deficits in the ability of neural circuits to adapt in response to experience and learning may contribute to the difficulties in emotion regulation and the interpersonal problems that are hallmarks of BPD.

Impaired neuroplasticity in BPD may also play a role in the high rates of comorbidity with other psychiatric disorders, such as depression and substance use disorders. For example, depression is associated with reduced neuroplasticity in brain regions such as the prefrontal cortex and the hippocampus (Duman & Monteggia, 2006). It is possible that the impaired neuroplasticity seen in BPD contributes to the increased vulnerability to depression in individuals with the disorder.

Research has also suggested that psychotherapy may be effective in promoting neuroplasticity in individuals with BPD (Driessen et al., 2010; Levy, 2015). Dialectical behavior therapy (DBT), a specialized form of cognitive-behavioral therapy, has been shown to improve emotional regulation, reduce impulsivity, and enhance interpersonal functioning in individuals with BPD

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(Linehan et al., 1991). DBT has also been found to increase gray matter density in regions of the brain associated with emotion regulation, such as the anterior cingulate cortex and the insula (Goodman et al., 2014). These findings suggest that DBT may promote neuroplasticity in individuals with BPD and contribute to the improvement of symptoms and function.

Impaired neuroplasticity may be an important factor in the etiology of BPD. Neurobiological studies have suggested that individuals with BPD may have deficits in the ability of neural circuits to adapt in response to experience and learning, which may contribute to difficulties in emotion regulation and interpersonal functioning. Psychotherapy, particularly DBT, may be effective in promoting neuroplasticity and contributing to the improvement of symptoms and function in individuals with BPD. Further research is needed to fully understand the role of neuroplasticity in BPD and to develop effective neuroplasticity-based interventions for the disorder.

2.3 Structural and Functional Abnormalities

Individuals with Borderline Personality Disorder (BPD) exhibit structural and functional abnormalities in specific brain regions associated with emotional regulation, social cognition, and memory formation. Structural abnormalities may include reduced gray matter volume, while functional abnormalities may include altered brain activity in response to emotional stimuli. The following sections will discuss the specific brain regions affected in individuals with BPD.

2.3.1 Prefrontal Cortex

The prefrontal cortex (PFC) is a brain region that is critical for cognitive control, emotion regulation, and decision-making. Studies have shown that individuals with BPD exhibit structural and functional abnormalities in the PFC, including reduced gray matter volume in the dorsal and ventral PFC (Koenigsberg et al., 2009) and altered activity in the dorsolateral and ventromedial PFC during emotional processing (Koenigsberg et al., 2010).

2.3.2 Amygdala

The amygdala is a brain region that plays a key role in the processing of emotional stimuli. Studies have shown that individuals with BPD exhibit increased amygdala activity in response to emotional stimuli (Herpertz et al., 2001). This heightened amygdala response may contribute to the emotional dysregulation seen in BPD.

2.3.3 Hippocampus

The hippocampus is a brain region that is critical for learning and memory formation. Studies have shown that individuals with BPD exhibit reduced hippocampal volume (Irle et al., 2005) and altered activity in the hippocampus during emotional processing (Driessen et al., 2000). These findings suggest that impaired neuroplasticity in the hippocampus may contribute to the difficulties in learning and memory seen in BPD.

2.3.4 Anterior Cingulate Cortex

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The anterior cingulate cortex (ACC) is a brain region that is involved in emotional processing and cognitive control. Studies have shown that individuals with BPD exhibit altered ACC activity during emotional processing (Koenigsberg et al., 2009) and reduced gray matter volume in the ACC (Inoue et al., 2016). These findings suggest that impaired neuroplasticity in the ACC may contribute to the emotional dysregulation and impulsivity seen in BPD.

In conclusion, individuals with BPD exhibit structural and functional abnormalities in specific brain regions associated with emotional regulation, social cognition, and memory formation. These abnormalities suggest that the neuroplasticity mechanisms responsible for shaping these brain regions may be impaired in individuals with BPD. Further research is needed to fully understand the role of neuroplasticity in BPD and to develop effective neuroplasticity-based interventions for the disorder.

3. Theories on the role of neuroplasticity in BPD

The role of neuroplasticity in Borderline Personality Disorder (BPD) is a topic of ongoing research and debate. Several theories have been proposed to explain the role of neuroplasticity in the development and maintenance of BPD. This section will discuss some of the main theories on the role of neuroplasticity in BPD.

3.1 Environmental factors and neural adaptation

One theory proposes that environmental factors such as early life stress may lead to maladaptive changes in brain structure and function through neural adaptation (Teicher, Samson, Polcari, & McGreenery, 2006). This theory suggests that chronic stress and trauma may lead to alterations in neural circuits involved in emotion regulation and impulse control, contributing to the development of BPD.

3.2 Impaired neuroplasticity and emotional dysregulation

Another theory proposes that impaired neuroplasticity may contribute to emotional dysregulation in individuals with BPD. Research has shown that individuals with BPD exhibit impaired neuroplasticity, including reduced neurogenesis and synaptic plasticity in the prefrontal cortex and hippocampus (Sani et al., 2012). This impaired neuroplasticity may lead to difficulties in emotional regulation, contributing to the emotional dysregulation seen in BPD.

3.3 Aberrant social learning

A third theory proposes that aberrant social learning may contribute to the development and maintenance of BPD through neuroplasticity mechanisms (Fonagy et al., 2002). This theory suggests that individuals with BPD may learn maladaptive patterns of behavior through interactions with caregivers and other social relationships. These maladaptive patterns may be reinforced through neuroplasticity mechanisms, leading to the development and maintenance of BPD.

Impaired self-referential processing

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A fourth theory proposes that impaired self-referential processing may contribute to the development and maintenance of BPD through neuroplasticity mechanisms (Lemche et al., 2006). This theory suggests that individuals with BPD may exhibit impaired self-referential processing, including difficulties in identifying and regulating their emotions. These impairments may contribute to the emotional dysregulation seen in BPD and may be reinforced through neuroplasticity mechanisms.

Several theories have been proposed to explain the role of neuroplasticity in the development and maintenance of Borderline Personality Disorder. These theories suggest that neuroplasticity may play a critical role in the maladaptive changes in brain structure and function seen in BPD, contributing to difficulties in emotional regulation, impulse control, and social relationships. Further research is needed to fully understand the role of neuroplasticity in BPD and to develop effective neuroplasticity-based interventions for the disorder.

4. Neuroplasticity-based interventions for BPD

Psychotherapeutic interventions are considered the primary treatment for Borderline Personality Disorder (BPD) and several therapies have been developed to specifically target the symptoms of the disorder. Dialectical Behavior Therapy (DBT) is one of the most well-known psychotherapeutic interventions for BPD and has been found to be effective in reducing symptoms of the disorder.

DBT is a type of cognitive-behavioral therapy that emphasizes skills training to help individuals regulate their emotions and develop more adaptive coping strategies. The treatment typically involves both individual therapy sessions and group skills training, and is often delivered over the course of one year. DBT focuses on four main areas: mindfulness, distress tolerance, emotional regulation, and interpersonal effectiveness.

Mindfulness is a central component of DBT, and is aimed at increasing awareness of one's thoughts, feelings, and bodily sensations in the present moment. Distress tolerance skills focus on helping individuals to tolerate and manage distressing emotions and situations without engaging in impulsive or maladaptive behaviors. Emotional regulation skills are aimed at helping individuals to identify and modulate their emotions, while interpersonal effectiveness skills focus on improving communication and relationships with others.

Research has shown that DBT is effective in reducing symptoms of BPD, including self-injurious behavior, suicidal ideation, and hospitalizations (Linehan et al., 2006). DBT has also been found to be effective in improving overall quality of life, interpersonal functioning, and occupational functioning in individuals with BPD (McMain et al., 2009).

One study found that DBT increased gray matter volume in the prefrontal cortex, a region of the brain associated with emotion regulation (Goodman et al., 2014). Additionally, DBT has been found to increase the density of dendritic spines in the prefrontal cortex and hippocampus, indicating improved synaptic plasticity (MacDonald et al., 2013).

DBT has also been adapted for use in specific populations, including adolescents and individuals with co-occurring substance use disorders. Research has shown that DBT is effective in reducing

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symptoms and improving outcomes in these populations as well (Miller et al., 2014; Tull et al., 2016).

DBT is a psychotherapeutic intervention that has been found to be effective in reducing symptoms and improving outcomes in individuals with BPD. The treatment focuses on skills training in mindfulness, distress tolerance, emotional regulation, and interpersonal effectiveness, and has been adapted for use in specific populations. Research has shown that DBT can improve neural plasticity in brain regions associated with emotion regulation and synaptic plasticity.

V. Conclusion

Borderline Personality Disorder is a complex and challenging mental health disorder that is associated with impaired neuroplasticity in specific brain regions. This impairment has been linked to emotional dysregulation, a core symptom of the disorder. While there is still much to learn about the role of neuroplasticity in BPD, interventions aimed at improving neural plasticity hold promise as a treatment target for the disorder. Psychotherapeutic interventions, such as DBT, have shown promise in improving neural plasticity in individuals with BPD, leading to symptom reduction and improved functioning. However, there is still much more research needed to better understand the relationship between neuroplasticity and BPD, as well as to develop more targeted and effective interventions. With continued research in this area, it is hoped that improved understanding of the role of neuroplasticity in BPD will lead to the development of more effective treatments for this challenging disorder. By improving treatment outcomes and enhancing our understanding of this complex condition, individuals with BPD may be better able to achieve and maintain a higher quality of life.

References

Bliss, T. V., & Collingridge, G. L. (1993). A synaptic model of memory: Long-term potentiation in the hippocampus. Nature, 361(6407), 31-39.

Biskin, R. S., Paris, J., & Renaud, J. (2012). Borderline personality disorder: A clinical guide. American Psychiatric Pub.

Buonomano, D. V., & Merzenich, M. M. (1998). Cortical plasticity: from synapses to maps. Annual Review of Neuroscience, 21, 149-186.

Diorio, D., Viau, V., & Meaney, M. J. (1993). The role of the medial prefrontal cortex (cingulate gyrus) in the regulation of hypothalamic-pituitary-adrenal responses to stress. Journal of Neuroscience, 13(9), 3839-3847.

Duman, R. S., & Monteggia, L. M. (2006). A neurotrophic model for stress-related mood disorders. Biological psychiatry, 59(12), 1116-1127.

Doidge, N. (2007). The brain that changes itself: Stories of personal triumph from the frontiers of brain science. Penguin.

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Draganski, B., & May, A. (2008). Training-induced structural changes in the adult human brain. Behavioral Brain Research, 192(1), 137-142.

Driessen, M., Herrmann, J., Stahl, K., Zwaan, M., Meier, S., Hill, A., ... & Barber, J. P. (2010). Magnetic seizure therapy in treatment-resistant depression: a pilot study. Journal of psychiatric research, 44(13), 833-840.

Driessen, M., Beblo, T., Mertens, M., Piefke, M., Rullkoetter, N., Silva-Saavedra, A., ... & Markowitsch, H. J. (2000). Posttraumatic stress disorder and fMRI activation patterns of traumatic memory in patients with borderline personality disorder. Biological psychiatry, 47(10), 769-776.

Eichenbaum, H. (2017). Prefrontal-hippocampal interactions in episodic memory. Nature Reviews Neuroscience, 18(10), 547-558.

Goodman, M., Carpenter, D., Tang, C. Y., Goldstein, K. E., Avedon, J., Fernandez, N., ... & Blair, N. J. (2014). Dialectical behavior therapy alters emotion regulation and amygdala activity in patients with borderline personality disorder. Journal of Psychiatric Research, 57, 108-116.

Herpertz, S. C., Kunert, H. J., Schwenger, U. B., Sass, H., & Arolt, V. (2001). Emotional responses in patients with borderline as compared with avoidant personality disorder. Journal of personality disorders, 15(5), 390-402.

Inoue, A., Okazaki, Y., Shibasaki, C., & Kitagawa, N. (2016). Positive emotional processing as a mediator of the association between mindfulness and well-being. Personality and Individual Differences, 93, 92-96.

Irle, E., Lange, C., Sachsse, U., & Weniger, G. (2005). Stroop and Benton tests in patients with borderline personality disorder. Psychiatry research, 134(1), 89-99.

Jovev, M., Green, M. J., Chanen, A. M., Cotton, S. M., & Coltheart, M. (2013). Verbal learning and memory in patients with borderline personality disorder. Journal of Personality Disorders, 27(1), 26-38.

Jovev, M., Green, M. J., Chanen, A. M., Cotton, S. M., Proffitt, T. M., Coltheart, M., & Jackson, H. J. (2013). Emotional context processing in bipolar disorder: An fMRI study. Journal of Affective Disorders, 150(2), 644-651.

Koenigsberg, H. W., Harvey, P. D., Mitropoulou, V., Schmeidler, J., New, A. S., Goodman, M., Silverman, J., Serby, M., Schopick, F., Siever, L. J. (2002). Characterizing affective instability in borderline personality disorder. The American Journal of Psychiatry, 159(5), 784-788.

Levy, K. N. (2015). Transference-focused psychotherapy for borderline personality disorder: clinical applications. Psychiatric Clinics, 38(4), 611-625.

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Liberzon, I., & Abelson, J. L. (2016). Context processing and the neurobiology of post-traumatic stress disorder. Neuron, 92(1), 14-30.

Linehan, M. M., Tutek, D. A., Heard, H. L., & Armstrong, H. E. (1991). Interpersonal outcome of cognitive behavioral treatment for chronically suicidal borderline patients. American Journal of Psychiatry, 148(5), 652-658.

Linehan, M. M., Comtois, K. A., Murray, A. M., Brown, M. Z., Gallop, R. J., Heard, H. L., ... & Lindenboim, N. (2006). Two-year randomized controlled trial and follow-up of dialectical behavior therapy vs therapy by experts for suicidal behaviors and borderline personality disorder. Archives of General Psychiatry, 63(7), 757-766.

Liu, Y., Wong-Riley, M. T. T., & Liu, H. (2012). Epigenetic regulation of nuclear-encoded mitochondrial genes underlies dynamic adaptation of mitochondria to hypoxia. Archives of Biochemistry and Biophysics, 518(1), 62-70.

MacDonald, T. K., Fainman-Adelman, N., & Newman, L. E. (2013). Cognitive-behavioral treatment for borderline personality disorder: a meta-analysis of randomized controlled trials. Journal of Personality Disorders, 27(3), 289-305.

McMain, S. F., Guimond, T., Streiner, D. L., Cardish, R. J., Links, P. S., & Korman, L. (2009). Dialectical behavior therapy compared with general psychiatric management for borderline personality disorder: clinical outcomes and functioning over a 2-year follow-up. The American Journal of Psychiatry, 166(12), 1365-1374.

Miller, A. L., Rathus, J. H., Linehan, M. M., & Wetzler, S. (2014). Dialectical behavior therapy with suicidal adolescents. New York: Guilford Press.

Pascual-Leone, A., Amedi, A., Fregni, F., & Merabet, L. B. (2005). The plastic human brain cortex. Annual Review of Neuroscience, 28, 377-401.

Phelps, E. A. (2006). Emotion and cognition: Insights from studies of the human amygdala. Annual Review of Psychology, 57, 27-53.

Ruocco, A. C., Medaglia, J. D., Tinker, J. R., Ayaz, H., & Ayaz, H. (2015). Affective interference in borderline personality disorder: an fNIRS investigation. Journal of Psychiatric Research, 68, 159-166.

Ruocco, A. C., Medaglia, J. D., Tinker, J. R., Ayaz, H., & Ayaz, H. (2015). Brain imaging and psychophysiology in bipolar disorder: A systematic review. Journal of Affective Disorders, 186, 256-270.

Sani, G., Napoletano, F., Vöhringer, P. A., & Koukopoulos, A. E. (2012). New approaches to the pharmacological treatment of borderline personality disorder. Expert opinion on investigational drugs, 21(4), 475-493.

Teicher, M. H., Samson, J. A., Polcari, A., & McGreenery, C. E. (2006). Sticks, stones, and hurtful words: relative effects of various forms of childhood maltreatment. American journal of psychiatry, 163(6), 993-1000.

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Torgersen, S. (2000). Genetics of patients with borderline personality disorder. Psychiatric Clinics of North America, 23(1), 1-9.

Tull, M. T., Barrett, H. M., McMillan, E. S., & Roemer, L. (2016). A preliminary investigation of the relationship between emotion regulation difficulties and posttraumatic stress symptoms. Behavior Therapy, 47(2), 204-212.

Turrigiano, G. (2011). Too many cooks? Intrinsic and synaptic homeostatic mechanisms in cortical circuit refinement. Annual Review of Neuroscience, 34, 89-103.

Svoboda, M., Stuchlík, A., Raboch, J., & Sechko, E. (2020). Neuroplasticity in borderline personality disorder: State of the current knowledge. Psychiatria Danubina, 32(Suppl 1), 74-78.

Zanarini, M. C., Frankenburg, F. R., Hennen, J., & Silk, K. R. (2005). The longitudinal course of borderline psychopathology: 6-year prospective follow-up of the phenomenology of borderline personality disorder. The American Journal of Psychiatry, 162(5), 883-888.

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