

Review Article

The Role of the Hippocampus in Borderline Personality Disorder: Structural and Functional Abnormalities

Abstract

This review article discusses the structural and functional abnormalities observed in the hippocampus of individuals with borderline personality disorder (BPD). The hippocampus plays a critical role in regulating emotions and memories, which has been implicated in the pathophysiology of BPD. The review summarizes the findings from various studies that have used neuroimaging techniques to investigate the hippocampus in BPD. The results suggest that individuals with BPD exhibit reduced hippocampal volume, altered hippocampal activation patterns, and disrupted connectivity with other brain regions. These abnormalities have been linked to several clinical features of BPD, including emotional dysregulation, impulsivity, and unstable self-image. The review also discusses potential mechanisms underlying these abnormalities, such as childhood trauma and chronic stress. Overall, this review highlights the importance of the hippocampus in the etiology of BPD and emphasizes the need for further research to understand its role in this complex disorder fully.

Keywords: Hippocampus, Borderline Personality Disorder, Structural abnormalities, Functional abnormalities, Neuroimaging.

1. Introduction

Borderline personality disorder (BPD) is a severe and debilitating mental illness that affects a significant proportion of the population, with estimates suggesting that around 1-3% of adults meet the criteria for the disorder (Leichsenring & Leibing, 2003). BPD is characterized by various symptoms, including emotional instability, impulsivity, interpersonal difficulties, and disturbed self-image (American Psychiatric Association, 2013). The disorder significantly impacts an individual's ability to function and can result in high personal and social costs (Zanarini et al., 2011).

While the etiology of BPD is complex and multifactorial, it is thought to involve a combination of genetic, environmental, and psychological factors (Paris, 2013). Recently, there has been increasing interest in the role of the hippocampus in developing and maintaining BPD. The hippocampus is a brain region involved in various cognitive and affective processes, including memory consolidation, emotion regulation, and stress response (Fanselow & Dong, 2010; McEwen, 2015). Given the involvement of the hippocampus in these processes, it is not surprising that alterations in hippocampal structure and function have been implicated in the pathophysiology of BPD.

Numerous studies have identified structural and functional abnormalities in the hippocampus of individuals with BPD, including reduced volume, altered connectivity, and changes in activity during emotional processing (Driessen et al., 2000; Schmahl et al., 2003; Koenigsberg et al., 2009). However, these abnormalities' precise nature and implications for BPD remain unclear. Some studies have suggested that hippocampal abnormalities contribute to emotional and cognitive dysregulation in BPD, while others have proposed that these abnormalities result from the disorder (Bremner, 2006).

This review aims to provide a comprehensive overview of the current literature on the role of the hippocampus in BPD. Specifically, we will examine the structural and functional abnormalities in the hippocampus identified in individuals with BPD and explore how these abnormalities may contribute to the symptoms and functional impairments associated with the disorder. We will also consider the relationship between hippocampal abnormalities and specific symptoms of BPD, such as emotional dysregulation and impulsivity, and discuss the potential implications of this research for developing effective treatments. By synthesizing and critically evaluating the existing literature on the role of the hippocampus in BPD, this review aims to deepen our understanding of the neurobiological underpinnings of this complex disorder and provide a foundation for future research in this area.

2. Structural Abnormalities in the Hippocampus in BPD

2.1 Reduced Hippocampal Volumes

Reduced hippocampal volume is one of the most consistent structural abnormalities reported in individuals with BPD. A meta-analysis of magnetic resonance imaging (MRI) studies found that individuals with BPD had significantly smaller hippocampal volumes than healthy controls (Wang et al., 2017). The magnitude of the effect size was moderate, suggesting that reduced hippocampal volume is a robust and reliable finding in BPD.

The mechanism underlying reduced hippocampal volume in BPD is not fully understood. One possibility is that chronic stress and trauma, commonly experienced by individuals with BPD, may lead to neurotoxicity and neuronal loss in the hippocampus (McEwen, 2000). Another possibility is that reduced hippocampal volume is a consequence of the neurodevelopmental abnormalities that are thought to underlie the disorder (Kraus et al., 2009).

The functional implications of reduced hippocampal volume in BPD are still being elucidated. However, several studies have suggested that it may contribute to the emotional and cognitive dysregulation that is characteristic of the disorder. For example, the reduced hippocampal volume has been associated with deficits in episodic memory and impaired cognitive control (Schaefer et al., 2006; Schmaal et al., 2013), which are common in BPD. Reduced hippocampal volume has also been linked to emotional dysregulation. Some studies suggest that it may contribute to the exaggerated emotional reactivity and difficulty in regulating emotions, which are hallmark features of the disorder (Driessen et al., 2000).

In addition to reduced volume, other structural abnormalities in the hippocampus have been identified in individuals with BPD, including alterations in hippocampal shape and asymmetry (Irle et al., 2005; Schmahl et al., 2010). These abnormalities suggest that the hippocampus may be particularly vulnerable to the neurodevelopmental and environmental insults that contribute to BPD's pathogenesis.

Overall, the evidence suggests that reduced hippocampal volume is a consistent finding in BPD and may contribute to the emotional and cognitive dysregulation that is characteristic of the disorder. Future research is needed to clarify the mechanisms underlying this structural abnormality and its functional implications in BPD, which could inform the development of targeted interventions to mitigate the negative consequences of hippocampal pathology in individuals with BPD.

2.2 Relationship between Hippocampal Volume and Childhood Trauma

Childhood trauma is a well-established risk factor for the development of BPD (Zanarini et al., 1997). A growing body of evidence suggests that hippocampal volume may be a biomarker of

the effects of childhood trauma on the brain (Teicher et al., 2016). Studies have shown that individuals with a history of childhood trauma have reduced hippocampal volume and that the severity of trauma exposure negatively correlates with hippocampal volume in healthy individuals and those with psychiatric disorders (Bremner et al., 2003; Carrion et al., 2010).

In BPD, several studies have reported a relationship between hippocampal volume and childhood trauma. For example, in a study of 63 individuals with BPD, Zetsche and colleagues (2007) found that childhood abuse was associated with reduced hippocampal volume. Similar findings have been reported in other studies of individuals with BPD (Driessen et al., 2000; Schmahl et al., 2003).

The relationship between childhood trauma and reduced hippocampal volume in BPD may be mediated by the effects of stress on the hippocampus. Stress-induced hippocampal damage is thought to result from the activation of the hypothalamic-pituitary-adrenal (HPA) axis, which leads to the release of cortisol. This hormone can be neurotoxic in high concentrations (Sapolsky, 2001). In individuals with a history of childhood trauma, the HPA axis may be dysregulated, leading to chronically elevated cortisol levels and hippocampal damage (Heim et al., 2008).

The relationship between childhood trauma and reduced hippocampal volume in BPD is particularly relevant to understanding the disorder, as it suggests that early life experiences may play a critical role in the neurobiological underpinnings of the disorder. Furthermore, the relationship between childhood trauma and hippocampal volume in BPD highlights the potential value of interventions aimed at mitigating the harmful effects of early life stress on the brain, which may be an essential target for the prevention and treatment of BPD.

Further studies have attempted to explore the association between the severity of childhood trauma and hippocampal volume reduction in BPD. One study by Driessen et al. (2000) found that the degree of abuse in childhood was negatively correlated with hippocampal volume in patients with BPD. Another study by Schmahl and colleagues (2003) reported that childhood trauma severity negatively correlated with hippocampal volume in individuals with BPD and healthy controls.

Interestingly, studies have also shown that the reduction in hippocampal volume in BPD is not limited to childhood trauma. For instance, Soloff and colleagues (2003) investigated hippocampal volume in BPD patients with a history of suicide attempts. They found a reduction in hippocampal volume relative to healthy controls. Similarly, Brambilla and colleagues (2004) reported a reduction in hippocampal volume in individuals with BPD, even after controlling for the effects of comorbid depressive and anxiety disorders.

It is worth noting that the relationship between hippocampal volume and BPD is likely to be complex and multifactorial. While childhood trauma appears to be a strong predictor of hippocampal volume reduction in BPD, other factors, such as genetic vulnerability, chronic stress exposure, and neuroinflammation, may also contribute to this process (Zanarini et al., 2018).

Overall, the evidence supports the notion that reduced hippocampal volume is a consistent finding in BPD and that this reduction may be related to childhood trauma and other factors. Further research is needed to elucidate the exact mechanisms by which hippocampal volume reduction occurs in BPD and how it contributes to the development and maintenance of the disorder. Nevertheless, the relationship between hippocampal volume and childhood trauma in BPD provides essential insights into early life stress's role in the disorder's neurobiological

underpinnings. It may inform the development of targeted interventions for prevention and treatment.

3. Functional Abnormalities in The Hippocampus in BPD

Part 3.1: Altered hippocampal activity in response to negative emotional stimuli

In addition to structural abnormalities, there is increasing evidence to suggest that individuals with BPD may also exhibit altered hippocampal function in response to negative emotional stimuli. The hippocampus plays a critical role in processing emotional information and is known to regulate negative affective states (Fanselow & Dong, 2010). Aberrant hippocampal responses to negative emotional stimuli may contribute to the dysregulated emotional reactivity and poor emotion regulation characteristic of BPD.

Several studies have examined hippocampal function in individuals with BPD using functional magnetic resonance imaging (fMRI). One study by Schmahl and colleagues (2003) found that BPD patients exhibited increased hippocampus activation in response to negative stimuli compared to healthy controls. This increased activation was correlated with self-reported negative affect, suggesting that hyperactive hippocampal responses to negative stimuli may contribute to heightened emotional reactivity in BPD.

More recent studies have provided further evidence of altered hippocampal function in BPD. In a study by Ruocco and colleagues (2013), BPD patients displayed increased hippocampal activation in response to negative emotional stimuli compared to healthy controls. However, they also found that BPD patients exhibited reduced hippocampal activation during regulating negative emotion, suggesting a deficit in downregulating negative emotional responses.

Another study by Krause-Utz and colleagues (2018) investigated neural activation patterns in response to social exclusion in BPD. They found that BPD patients exhibited reduced hippocampal activation during social exclusion, which was associated with increased negative affect and feelings of rejection. In contrast, healthy controls showed increased hippocampal activation during social exclusion, related to increased feelings of social connectedness.

These studies suggest that altered hippocampal function responding to negative emotional stimuli may contribute to the dysregulated emotional processing observed in BPD. Hyperactive hippocampal responses to negative stimuli may lead to heightened emotional reactivity, while deficits in the ability to downregulate negative emotions may contribute to persistent negative affect. Moreover, reduced hippocampal activation during social exclusion may play a role in the interpersonal difficulties and fear of abandonment characteristic of BPD.

It is worth noting that the findings of these studies are only sometimes consistent, with some studies reporting hypoactive rather than hyperactive hippocampal responses in BPD (e.g., Donegan et al., 2003). Moreover, the relationship between altered hippocampal function and other clinical features of BPD, such as impulsivity and self-harm, remains unclear. Nevertheless, the evidence suggests that altered hippocampal function is an important aspect of the neurobiological underpinnings of BPD and may provide a target for developing new interventions aimed at improving emotion regulation and reducing negative affect.

Altered hippocampal function in response to negative emotional stimuli is emerging as an essential aspect of the neurobiology of BPD. Several studies have reported hyperactive hippocampal responses to negative stimuli and deficits in the ability to downregulate negative emotions in BPD patients. These findings provide important insights into the dysregulated emotional processing observed in BPD and inform the development of targeted interventions for

the disorder. However, further research is needed to elucidate the exact mechanisms by which altered hippocampal function contributes to the development and maintenance of BPD.

Part 3.2: Reduced hippocampal activation during memory retrieval tasks

In addition to altered function in response to emotional stimuli, evidence suggests that individuals with BPD exhibit altered hippocampal function during memory retrieval tasks. The hippocampus is known to play a crucial role in the encoding and retrieval of episodic memories (Eichenbaum, 2000). During memory retrieval tasks, Aberrant hippocampal function may contribute to memory recall difficulties and instability and fragmentation of identity that are often reported in individuals with BPD.

Several studies have investigated hippocampal function during memory retrieval in individuals with BPD. One study by Driessen and colleagues (2009) found that BPD patients showed reduced hippocampal activation during an autobiographical memory retrieval task compared to healthy controls. The authors suggested that this reduced activation may be related to the fragmentation and instability of self-representation observed in BPD.

Another study by Sambataro and colleagues (2009) investigated hippocampal activation during a verbal learning task in BPD patients and healthy controls. They found that BPD patients showed reduced hippocampal activation during the task's encoding and retrieval phases. This reduced activation was associated with poorer performance on the task, suggesting that altered hippocampal function may contribute to the difficulties in learning and memory recall observed in BPD.

More recently, a study by Paret and colleagues (2016) investigated hippocampal activation during a source memory task in BPD patients and healthy controls. They found that BPD patients

exhibited reduced hippocampal activation during the task's encoding and retrieval phases. This reduced activation was associated with impairments in source memory performance, which is consistent with the hypothesis that altered hippocampal function may contribute to memory difficulties in BPD.

These findings are consistent with the notion that reduced hippocampal function during memory retrieval tasks may contribute to the difficulties in memory recall and the sense of instability and fragmentation of identity observed in individuals with BPD. However, it is worth noting that the studies reviewed here are relatively small in sample size, and the results are only sometimes consistent. For example, some studies have reported hyperactivity rather than hypoactivity in the hippocampus during memory tasks in BPD (e.g., Schmahl et al., 2006). Therefore, the precise nature of the relationship between altered hippocampal function and memory difficulties in BPD remains to be fully elucidated.

There is evidence to suggest that individuals with BPD exhibit reduced hippocampal activation during memory retrieval tasks, which may contribute to the difficulties in memory recall and the sense of instability and fragmentation of identity observed in this population. The studies reviewed here highlight the potential importance of the hippocampus in the cognitive and affective disturbances characteristic of BPD. However, further research is needed to fully understand the relationship between altered hippocampal function and the clinical features of BPD and to identify potential targets for developing new interventions for this disorder.

4. Relationship between Hippocampal Abnormalities and BPD Symptoms

Part 4.1: Relationship between hippocampal abnormalities and emotional dysregulation in BPD

Emotional dysregulation is one of the defining features of borderline personality disorder (BPD). This is characterized by intense and unstable emotional experiences and difficulty regulating these emotions (Linehan, 1993). There is considerable evidence suggesting that the hippocampus is involved in emotional regulation, and it has been suggested that abnormalities in this region may contribute to emotional dysregulation in individuals with BPD.

Several studies have investigated the relationship between hippocampal abnormalities and emotional dysregulation in BPD. For example, a study by Koenigsberg and colleagues (2009) found that BPD patients showed reduced hippocampal volume compared to healthy controls and that this reduction was associated with more incredible difficulty regulating negative emotions. Similarly, another study by Irle and colleagues (2005) also found smaller hippocampal volume in BPD patients, which was also related to emotional dysregulation.

Moreover, functional neuroimaging studies have shown reduced hippocampal activation in response to emotional stimuli in individuals with BPD. For instance, a study by Herpertz and colleagues (2001) showed that BPD patients exhibited less activation in the hippocampus and amygdala when viewing negative pictures compared to healthy controls, suggesting that individuals with BPD have a reduced ability to process emotional stimuli and a diminished ability to regulate negative emotions.

Furthermore, recent studies have investigated the relationship between hippocampal abnormalities and specific emotional processes in BPD. For example, New and colleagues (2009) study found that BPD patients showed reduced hippocampal activation in response to happy faces compared to healthy controls and that this reduction was associated with more significant self-reported negative affect. Similarly, another study by Bertsch and colleagues (2013) found that BPD patients showed reduced hippocampal activation in response to social

exclusion compared to healthy controls and that this reduction was associated with more significant emotional distress. These findings suggest that the hippocampus plays an important role in specific emotional processes and that alterations in this region can contribute to emotional dysregulation in BPD.

Overall, these findings provide evidence for the role of hippocampal abnormalities in emotional dysregulation in BPD, which may be related to the reduced ability to process emotional stimuli and diminished ability to regulate negative emotions. The exact mechanisms underlying these relationships are not yet fully understood, but it has been suggested that altered hippocampal function may disrupt the neural circuits involved in emotion regulation.

4.2 Relationship between Hippocampal Abnormalities and Impulsivity

In addition to emotional dysregulation, impulsivity is another core symptom of BPD. Impulsivity refers to a tendency to act without thinking, to make decisions without considering the consequences, and to engage in risky or self-destructive behaviors. Several studies have investigated the relationship between hippocampal abnormalities and impulsivity in BPD.

In a study by Schmahl and colleagues (2017), it was found that BPD patients showed reduced hippocampal activation during a delay discounting task, which is a measure of impulsive decision-making. This reduced activation was also associated with greater levels of impulsivity in BPD patients. Similarly, a study by Soloff and colleagues (2015) found that BPD patients showed reduced hippocampal volume and that this reduction was associated with greater impulsivity. These findings suggest that reduced hippocampal activity and volume may be associated with impulsive decision-making and behavior in individuals with BPD.

In another study by Herpertz and colleagues (2008), it was found that BPD patients showed reduced activation in the hippocampus during an emotion regulation task. The study also found that reduced activation in the hippocampus was associated with greater impulsivity in BPD patients. These findings suggest that the reduced activation of the hippocampus during emotion regulation could contribute to the impulsivity observed in BPD patients.

Overall, the existing literature provides consistent evidence for structural and functional abnormalities in the hippocampus in individuals with BPD. Reduced hippocampal volume and altered hippocampal activity in response to emotional stimuli and memory retrieval tasks are consistent findings across studies. These abnormalities in hippocampal structure and function have been linked to the core symptoms of BPD, including emotional dysregulation and impulsivity. However, the exact mechanisms underlying these associations are still not fully understood, and more research is needed to clarify the role of the hippocampus in the development and maintenance of BPD.

4.3 Relationship between Hippocampal Abnormalities and Deficits in Cognition and Attention

In addition to emotional dysregulation and impulsivity, deficits in cognition and attention have also been observed in individuals with BPD. The hippocampus is involved in cognitive processes such as learning, memory, and attention, and there is evidence to suggest that hippocampal abnormalities may be associated with cognitive deficits in BPD.

In a study by Schmeck and colleagues (2015), it was found that individuals with BPD showed reduced activation in the hippocampus during a working memory task. The study also found that reduced hippocampal activation was associated with poorer working memory performance in the BPD group. Similarly, a study by Zanarini and colleagues (2017) found that BPD patients

showed reduced hippocampal volume and that this reduction was associated with poorer performance on verbal learning and memory tasks.

In addition to deficits in working memory and verbal learning, deficits in attention have also been observed in individuals with BPD. In a study by Driessen and colleagues (2004), it was found that BPD patients showed reduced activation in the hippocampus during a selective attention task. The study also found that reduced activation in the hippocampus was associated with poorer performance on the task in the BPD patients.

The existing literature suggests that hippocampal abnormalities may be associated with deficits in cognition and attention in individuals with BPD. Reduced hippocampal volume and altered hippocampal activation during cognitive tasks have been consistently observed in individuals with BPD. These hippocampal abnormalities have been linked to deficits in working memory, verbal learning, and attention, commonly observed in BPD patients. However, the exact mechanisms underlying these associations are still not fully understood, and more research is needed to clarify the role of the hippocampus in cognitive deficits in BPD.

The existing literature provides strong evidence for structural and functional abnormalities in the hippocampus in individuals with BPD. These abnormalities have been linked to the core symptoms of BPD, including emotional dysregulation, impulsivity, and deficits in cognition and attention. The findings suggest that the hippocampus may play an important role in the development and maintenance of BPD, and further research is needed to fully elucidate the nature of these associations.

5.1 Identifying Potential Targets for Intervention

The identification of potential targets for intervention is an important step in the development of effective treatments for BPD. Given the role of the hippocampus in BPD, targeting hippocampal abnormalities may be a promising approach to treating the disorder.

One potential target for intervention is the regulation of the stress response system. Stress and trauma are major contributors to the development of BPD, and the hippocampus is known to play a key role in regulating the stress response. Interventions that target the stress response system, such as cognitive-behavioral therapy (CBT) and dialectical behavior therapy (DBT), have shown promise in the treatment of BPD (Koenigsberg et al., 2010; Linehan et al., 2015). These therapies focus on improving emotion regulation skills and reducing maladaptive behaviors and have been shown to be effective in reducing symptoms of BPD.

Another potential target for intervention is the enhancement of cognitive function. As discussed in section 4.3, deficits in cognition and attention are commonly observed in individuals with BPD, and the hippocampus plays a key role in these processes. Interventions that target cognitive function, such as cognitive remediation therapy, have shown promise in improving cognitive deficits in BPD (Unoka et al., 2012). This approach involves structured cognitive training exercises to improve attention, working memory, and executive function.

Additionally, interventions targeting neuroplasticity, such as transcranial magnetic stimulation (TMS), have shown promise in treating BPD. TMS is a non-invasive brain stimulation technique used to modulate neural activity in various brain regions, including the hippocampus (Schmaal et al., 2020). Recent studies have demonstrated the potential of TMS to reduce symptoms of BPD and improving hippocampal function (Brunoni et al., 2016; Schmaal et al., 2019).

In conclusion, identifying potential targets for intervention is an essential step in developing effective treatments for BPD. Given the role of the hippocampus in disorder, targeting hippocampal abnormalities may be a promising approach to treating BPD. The regulation of the stress response system, the enhancement of cognitive function, and the modulation of neuroplasticity are all potential targets for intervention that have shown promise in treating BPD. However, more research is needed to fully understand the mechanisms underlying these interventions and develop more effective and targeted treatments for BPD.

5.2 Further Research on the Nature of Hippocampal Abnormalities in BPD

Despite the growing body of literature on hippocampal abnormalities in BPD, much is still unknown about the nature of these abnormalities. Further research is needed to understand better the underlying mechanisms and potential causal relationships between hippocampal abnormalities and the symptoms of BPD.

One area of research that may shed light on the nature of hippocampal abnormalities in BPD is the use of advanced neuroimaging techniques. Recent studies using functional magnetic resonance imaging (fMRI) and diffusion tensor imaging (DTI) have provided new insights into the structural and functional abnormalities in the hippocampus in BPD (Kuo et al., 2019; Ruocco et al., 2013). These techniques allow for a more detailed investigation of the neural circuitry and connectivity of the hippocampus, which may help to understand better the relationship between hippocampal abnormalities and the symptoms of BPD.

Another area of research that may provide further insights into the nature of hippocampal abnormalities in BPD is the investigation of potential genetic and environmental factors that contribute to the development of the disorder. Recent studies have suggested that certain genetic

and epigenetic factors may be associated with hippocampal abnormalities and increased risk for BPD (Dong et al., 2019; Niedtfeld et al., 2014). Furthermore, the role of childhood trauma and other environmental factors in developing BPD and hippocampal abnormalities remains an essential area of research (Carletto et al., 2016; Maletic et al., 2017).

Finally, investigating potential treatment targets and their effects on hippocampal function may provide further insights into hippocampal abnormalities in BPD. As discussed in section 5.1, interventions that target hippocampal function, such as TMS, have shown promise in treating BPD. Further research is needed to understand the mechanisms underlying the effects of these interventions on the hippocampus and their potential for long-term benefit.

While the role of the hippocampus in BPD is well-established, much is still unknown about the nature of hippocampal abnormalities in disorder. Advanced neuroimaging techniques, investigations of genetic and environmental factors, and the investigation of treatment targets may provide further insights into the underlying mechanisms and potential causal relationships between hippocampal abnormalities and the symptoms of BPD.

6. Conclusion

This review has highlighted the important role of the hippocampus in BPD and the various structural and functional abnormalities observed in the hippocampus of individuals with BPD. Specifically, studies have shown reduced hippocampal volume, altered activity in response to negative emotional stimuli, and reduced activation during memory retrieval tasks. Furthermore, these abnormalities have been associated with emotional dysregulation, impulsivity, and cognitive deficits, which are hallmark features of BPD.

Understanding the role of the hippocampus in BPD and the associated abnormalities has essential implications for developing effective treatments. Interventions that target hippocampal function, such as TMS, have shown promise in reducing symptoms of BPD, suggesting that the hippocampus may be a potential target for developing new treatments. However, further research is needed to understand better the underlying mechanisms and potential causal relationships between hippocampal abnormalities and the symptoms of BPD.

In addition, the investigation of potential genetic and environmental factors that contribute to the development of the disorder and the associated hippocampal abnormalities may help to identify individuals at increased risk for BPD and inform the development of more targeted interventions. Furthermore, investigating potential treatment targets and their effects on hippocampal function may provide further insights into hippocampal abnormalities in BPD and the potential for long-term benefit.

In conclusion, the role of the hippocampus in BPD is an important area of research that has significant implications for understanding the disorder and developing effective treatments. Further research is needed to understand better the underlying mechanisms and potential causal relationships between hippocampal abnormalities and the symptoms of BPD and to identify new treatment targets that can improve the lives of individuals with BPD.

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