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Non-Suicidal Self-Injury (NSSI) in Borderline Personality Disorder: Dynamic Mechanisms, Cascading Models, and Psychosocial Outcomes in the Context of Specialist Interventions

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Abstract

Background. Non-suicidal self-injury (NSSI) occurs in 69-90% of women with borderline personality disorder (BPD) and represents a critical diagnostic criterion and therapeutic challenge. Despite its high prevalence and clinical significance, the dynamic psychological and neurobiological mechanisms underlying NSSI remain incompletely understood, and a substantial gap persists between acute symptom reduction and long-term psychosocial recovery.

Aim. This study reviews NSSI in BPD, focusing on: identification of proximal psychological mechanisms and developmental factors influencing NSSI risk, and assessment of specialized therapeutic approaches' effectiveness in reducing NSSI and achieving sustained psychosocial functioning.

Materials and Methods. A literature review was conducted using databases (PubMed, PsycINFO, and Google Scholar) for articles up to November 2025. Inclusion criteria focused on peer-reviewed studies examining NSSI in BPD populations using validated assessment tools.

Results. Neuroimaging demonstrates that NSSI produces immediate amygdala deactivation, providing neurobiological reinforcement for this behavior. The Emotional Cascade Model identifies rumination instability and emotion differentiation deficits as key triggers for NSSI episodes. Childhood trauma dysregulates the HPA axis and correlates with functional deficits. Dialectical Behavior Therapy (DBT) shows a moderate effect size in reducing self-harm (Hedges' g = -0.622) and induces neuroplastic changes. Mentalization-Based Therapy (MBT) reduces suicide attempts, though social functioning often remains impaired even in long-term follow-up.

Conclusions. Specialized psychotherapies effectively stabilize acute symptoms, but a clear gap persists between symptom control and functional recovery. Future research should adopt complex dynamic systems perspectives and prioritize outcomes reflecting social integration and quality of life.

Keywords: non-suicidal self-injury, borderline personality disorder, emotional dysregulation, dialectical behavior therapy, mentalization-based therapy, neuroplasticity, psychosocial functioning

1. Introduction

Borderline Personality Disorder (BPD) is a severe mental disorder diagnosed by pervasive patterns of instability in affect regulation, impulse control, interpersonal relationships, and self-image [1]. According to DSM-5, BPD diagnosis requires meeting at least 5 of 9 criteria, one of which is recurrent suicidal behavior or self-mutilating behavior. The estimated prevalence of BPD is approximately 1.6-5.9% in the general population [2] with significantly higher rates (20%) in clinical psychiatry settings. BPD is associated with substantial functional impairment, high suicide rates (up to 10%) [3], and significant healthcare utilization costs [4].

Non-Suicidal Self-Injury (NSSI) is defined as the deliberate, self-inflicted destruction of body tissue without suicidal intent, to reduce psychological tension [6]. Common methods include cutting, burning, scratching, hitting, interfering with wound healing and others. NSSI is distinguished from suicidal behavior (SSI) by frequency (NSSI is often repetitive and chronic, whereas suicide attempts may be singular or episodic), method (NSSI typically involves less lethal methods, for example, superficial cutting vs. overdose), intent (NSSI is performed without intent to die, whereas SSI involves suicidal ideation) and function (NSSI primarily serves the function of emotion regulation, for example, reducing intense tension, while SSI aims at a lasting escape from psychological pain) [7]. This distinction is clinically crucial, as conflating NSSI with suicidal behavior can lead to inappropriate treatment responses and misinterpretation of therapeutic outcomes.

Nonsuicidal self-harm (NSSI) poses a fundamental challenge in the diagnosis and treatment of borderline personality disorder (BPD). Such behavior is so closely associated with BPD that it is included as a diagnostic criterion in the DSM-5, and its high prevalence is crucial for clinicians [9]. NSSI, defined as intentional tissue damage without suicidal intent, is common, with an estimated prevalence of 69–90% in women with BPD [8, 10]. NSSI, most often in the form of skin cutting, is strongly associated with emotion dysregulation, a prominent feature of BPD [8]. Increased awareness and research on this phenomenon necessitates a rigorous distinction between NSSI and suicidal behavior (SSI) to more accurately assess therapeutic outcomes [11]. NSSI is a primary therapeutic target, and its persistence significantly complicates treatment and continuity of care [12].

Aim

The aim of this study is to present a comprehensive review of non-suicidal self-injury (NSSI) in borderline personality disorder (BPD), analyzing the dynamic psychological and

neurobiological mechanisms underlying this phenomenon and evaluating the effectiveness of specialized therapeutic approaches. The paper will examine the developmental basis of NSSI in BPD, emphasizing the critical role of childhood trauma in HPA axis dysregulation and the neural circuitry of emotion dysregulation, including amygdala hyperreactivity and prefrontal underactivation. It will also evaluate data from clinical trials and longitudinal studies regarding the efficacy of evidence-based psychotherapies—particularly Dialectical Behavior Therapy (DBT) and Mentalization-Based Therapy (MBT)—in reducing NSSI frequency and achieving long-term psychosocial functioning. The review will analyze proximal mechanisms such as the Emotional Cascade Model, rumination instability, and emotion differentiation deficits that trigger NSSI episodes. The collected data were carefully analyzed to identify convergent findings, critical gaps in the literature—particularly the discrepancy between symptom reduction and functional recovery—and the need for a shift toward Complex Dynamic Systems perspectives and recovery-oriented outcome measures in NSSI treatment for patients with BPD.

2. Research Materials and Methods

2.1 Participants (Literature Search Strategy)

This narrative review synthesized current literature on non-suicidal self-injury (NSSI) in borderline personality disorder (BPD), with emphasis on dynamic mechanisms and evidence-based treatments. Studies were included if they examined NSSI in individuals diagnosed with BPD or displaying high BPD features according to DSM-5 diagnostic criteria. The review encompassed research utilizing validated assessment instruments for both BPD diagnosis (e.g., Structured Clinical Interview for DSM-5, Borderline Personality Disorder Severity Index) and NSSI measurement (e.g., Deliberate Self-Harm Inventory, Self-Injurious Thoughts and Behaviors Interview).

Included studies represented diverse methodological approaches: cross-sectional studies examining prevalence and correlates, longitudinal studies investigating developmental trajectories, neuroimaging studies exploring neural mechanisms, experience sampling method (ESM) studies capturing real-time dynamics, and randomized controlled trials (RCTs) evaluating treatment efficacy. Studies involving adolescent, adult, and mixed-age BPD populations were included to provide a comprehensive developmental perspective.

2.2 Procedure / Measure / Instruments

A comprehensive literature search was conducted in PubMed Central (PMC), PsycINFO, and Google Scholar using combinations of key terms: "non-suicidal self-injury", "NSSI", "self-harm", "borderline personality disorder", "BPD", "emotional dysregulation",

"rumination", "neuroimaging", "trauma", "childhood maltreatment", "dialectical behavior therapy", "mentalization-based therapy", "schema therapy", and "transference-focused therapy".

The selection criteria prioritized high-quality evidence, including meta-analyses, randomized controlled trials, longitudinal studies, and neuroimaging investigations. Articles were included if they provided significant data on the psychological and neurobiological mechanisms of NSSI, validated assessment instruments for BPD and NSSI, clinical efficacy of psychotherapeutic interventions, and long-term psychosocial outcomes. Both developmental factors (childhood trauma, HPA axis dysregulation) and proximal mechanisms (emotional cascade model, rumination instability) were considered to provide a comprehensive overview of NSSI in BPD.

2.3 Data Collection and Analysis / Statistical Analysis

2.3.1 Statistical Software

As this study is a comprehensive narrative review of existing literature, no statistical software was used for meta-analysis or new statistical calculations. Data synthesis was performed qualitatively based on reported results in the included studies.

2.3.2 AI

Artificial Intelligence (AI) tools were utilized in the preparation of this manuscript. Specifically, a Large Language Model (LLM) was employed to assist in editing the text for better comprehension, clarity, and adherence to academic English standards. The AI tool served strictly as an assistive instrument for linguistic refinement; the final interpretation of data, selection of literature, and conclusions were determined solely by the authors.

2.3.3 Statistical Methods

The review analyzes descriptive statistics, effect sizes (such as Hedges' g for treatment efficacy), and clinical outcomes (including NSSI frequency reduction, remission rates, and psychosocial functioning improvements) as reported in the primary source documents. Studies were categorized into three analytical domains: etiological and neurobiological mechanisms, proximal psychological mechanisms, and treatment efficacy with long-term outcomes. No new statistical tests were applied to the aggregated data.

3. Research results

3.1 Developmental Mechanisms and Neurobiological Vulnerabilities

3.1.1 Etiological Architecture: Trauma and Dysregulation.

Childhood trauma (CT) is a common and significant risk factor for BPD, influencing neurobiological and psychological development. Most BPD patients have a history of traumatic experiences that affect the HPA (hypothalamic-pituitary-adrenal) axis, leading to hyperreactivity of the stress response system and exacerbating emotional pain, which can be alleviated by self-harm [14]. CT is strongly correlated with the severity of BPD and depression symptoms, making it a significant predictor of NSSI risk [15]. Importantly, CT has a direct impact on psychosocial functioning. Studies have shown that youth engaging in NSSI who report CM have significantly lower levels of psychosocial functioning (lower GAF scores) compared to the NSSI group without a history of CM [15]. This increased functional deficit suggests that CM not only increases the risk of BPD and NSSI but also leads to more persistent impairments in overall functioning. PTSD is frequently comorbid in the BPD population (30-50%), and these patients are more likely to engage in NSSI as an extreme method of alleviating PTSD symptoms [16]. Consequently, effective clinical approaches for this subgroup must integrate trauma treatment (e.g., PTSD) and stabilization of BPD symptoms.

3.1.2 Neurobiology of Emotion Dysregulation and Auto-Aggression

The neurobiological mechanism of emotion dysregulation in BPD is characterized by a neural circuitry imbalance in which overactive limbic regions (responsible for emotion generation) are insufficiently inhibited by underactive cortical regions (responsible for executive control), such as the prefrontal cortex (PFC) [14]. Neuroimaging studies consistently demonstrate abnormalities in the amygdala (overreactivity) and in the prefrontal and medial cingulate cortices (impaired control) [17].

A functional explanation for NSSI has been provided by neurobiological studies. In women with BPD, the act of incision (compared to sham treatment) was found to lead to subjective and objective reductions in aversive tension after stress induction [8]. In the same study, a decrease in amygdala activity was observed after incision, and there was a normalization in functional connectivity with the superior frontal gyrus [8].

The fact that NSSI immediately leads to a quieting of amygdala hyperactivity and restoration of functionality in control circuits provides a key neurobiological rationale for this behavior. This immediate relief of internal tension is a powerful negative behavioral

reinforcer. From a clinical perspective, this reinforcer explains why NSSI is so difficult to stop and why effective interventions must provide alternative, equally rapid, yet adaptive mechanisms for tension regulation.

3.2 Dynamic Models and Proximal Psychological Mechanisms

3.2.1 Emotional Cascade Model (ECM)

The Emotional Cascade Model (ECM) directly links emotional dysregulation to behavioral dysregulation, identifying rumination as a driving mechanism. The ECM describes a positive feedback loop: ruminating about negative emotions intensifies affect, which in turn increases rumination, leading to an escalation of the emotional state—the cascade [18]. NSSI, in this context, serves a distraction function, intended to interrupt this painful spiral of rumination and intense negative affect [19].

Research using experience sampling methods (ESM) has confirmed that not only trait rumination, but especially momentary instability of rumination and instability of negative affect interact to strongly predict episodes of NSSI [5, 19]. This dynamic relationship, particularly pronounced in the case of sadness, requires a shift in the research paradigm from trait measurements to approaches that take into account within-person variability [20]. The focus on instability suggests that treatment must target the patient's ability to interrupt these momentary, escalating dynamics.

3.2.2 Emotion Differentiation as a Protective Factor

The ability to differentiate emotions—that is, make nuanced distinctions between broad categories of negative affect—has been proposed as a key protective factor against NSSI [21]. Research has shown that in individuals who have difficulty differentiating negative emotions, rumination strongly predicts higher rates of NSSI impulses and acts [21]. The ability to precisely identify and label feelings is thought to be a foundation for adaptive self-regulatory strategies. Affect labeling, as part of this process, has been linked to deactivation of amygdala activity [21]. This means that learning more precise emotional language, promoted in psychotherapies (e.g., through mindfulness techniques), directly impacts the neural circuitry of dysregulation, offering an adaptive mechanism for disrupting emotional cascades.

3.2.3 Impulsivity and Negative Urgency

An additional proximal mechanism is Negative Urgency. Although poor emotion regulation strategies are generally associated with NSSI, this association has been shown to be particularly strong in individuals characterized by high Negative Urgency—that is, the tendency to engage in impulsive actions (e.g., self-harm) in the face of intense negative emotions [9]. These findings emphasize that effective treatment must not only improve

general emotion regulation skills but also directly address the impulse control deficits triggered by intense negative affect [9].

Table 1. Key Psychological Mechanisms and Their Role in NSSI in BPD

Mechanism/ Construct	Role in NSSI	Related Model	Empirical Conclusions
Emotion Dysregulation (ED)	Basic vulnerability; leads to increased internal tension	Biosocial	Central characteristic of BPD; strongly associated with NSSI [9].
Rumination Instability	Amplifies negative affect; drives cascade	Emotional Cascades (ECM)	Predicts higher rates of NSSI, especially when combined with poor emotion differentiation [19].
Differentiating Emotions	Protective Factor	Self-Regulation/Neurocognitive	The ability to label emotional nuances reduces the risk of NSSI by disrupting amygdala activity [21].
Negative Urgency	Impulsive action under stress	Affective Dysregulation	Moderates the relationship between poor emotion regulation strategy and NSSI frequency [21].

3.3 Evidence-Based Treatment Approaches and Long-Term Outcomes

In recent decades, a number of specialized psychotherapies for BPD have been developed and empirically supported. The goal of these interventions is to reduce core symptoms, with the reduction of self-harming behaviors, including NSSI, as a priority [12].

3.3.1 Dialectical Behavior Therapy (DBT)

Dialectical Behavior Therapy (DBT) is the most empirically supported approach [22]. It is a comprehensive, evidence-based psychotherapy developed by Marsha Linehan (1993) specifically for treating BPD, particularly individuals with chronic suicidal and self-harm behaviors [13, 23]. DBT is rooted in cognitive behavioral therapy (CBT) but integrates principles of dialectical philosophy (balancing acceptance and change), mindfulness and concentrating on the emotions in therapy, and biosocial theory (understanding BPD as resulting from biological vulnerability in interaction with an invalidating environment) [24].

The core components of standard DBT are individual therapy (weekly, 1 hour), skills training group (weekly, 2-2.5 hours) and therapist consultation team (weekly 1-2 hours), in

combination with support and adherence for therapists [24]. Standard DBT treatment duration is 12 months, though adaptations range from 6 months to 2 years depending on setting and population. DBT prioritizes life-threatening behaviors (suicide, NSSI), therapy-interfering behaviors (non-attendance, non-compliance), quality-of-life interfering behaviors (unemployment, homelessness), skills acquisition and generalization.

The dialectical stance—simultaneously validating the patient's current emotional experience while pushing for behavioral change—is central to DBT's effectiveness in engaging patients with BPD who often struggle with therapeutic relationships [24].

Meta-analyses indicate that DBT has a moderate effect size in reducing self-harming and parasuicidal behaviors [25]. Combining measures of suicidal and parasuicidal outcomes (across five studies) revealed a net benefit in favor of DBT (Hedges' g −0.622) [26]. DBT also displays efficacy in stabilizing and controlling self-harming behaviors and improving patient compliance [26].

The clinical effectiveness of DBT is closely linked to its impact on biological mechanisms. Neuroimaging studies following DBT treatment have shown significant deactivation of amygdala activity, as well as increased activity in regions of the frontal cortex involved in inhibitory control [31]. The achievement of measurable neural changes, such as amygdala deactivation, confirms that DBT leads to neuroplasticity in critical emotion regulation circuits.

3.3.2 Comparison of Other Specialized Psychotherapies (MBT, TFP, ST)

Other specialized psychotherapies also offer effective approaches. Mentalization-based therapy (MBT) has shown promising results, particularly in reducing self-harming behaviors. Long-term data, including an 8-year follow-up of one study of MBT for adults, showed that patients achieved a reduction in suicide attempts [27]. Still, overall data on long-term outcomes of psychotherapy for BPD are limited and some are of low quality [28].

Mentalization-Based Therapy (MBT) is a psychodynamic, attachment-based psychotherapy developed for treating BPD. MBT is grounded in attachment theory and focuses on improving mentalizing capacity— the ability to understand one's own and others' mental states (thoughts, feelings, wishes, beliefs) [29]. MBT therapeutic strategy includes psychoeducation, individual therapy, group therapy and case conceptualization [29]. The adopt therapists a "not-knowing" stance, actively helping the patient to pause and reflect before reacting impulsively, identify affect as it arises in the therapeutic relationship, explore alternative perspectives on self and others' mental states, maintain mentalizing under stress (affect focus, but not affect flooding). Unlike DBT's skills-based, directive approach, MBT is

exploratory and relationship-focused, emphasizing the process of mentalizing over specific skill acquisition [29].

Other therapies, such as Transference Focused Therapy (TFP) and Schema Therapy (STP), have also been shown to be effective in reducing the overall severity of BPD symptoms [28].

However, head-to-head comparisons between DBT, MBT, and TFP are limited [28]. Most importantly, despite successes in stabilizing acute risk and reducing symptoms, MBT research has shown that overall social functioning in patients often remains impaired even after long-term follow-up [27]. This persistent functional gap, where a patient stops self-harming but continues to struggle with social integration and quality of life, represents a key unresolved clinical problem [30].

3.3.3 New Therapeutic and Pharmacological Directions

Recent years have seen innovation. Rapid reviews indicate the emergence of new psychotherapeutic programs (e.g., modified or expanded DBT), as well as new perspectives on pharmacological interventions, particularly ketamine, and holistic therapies and digital tools [30].

Although some pharmacological agents, such as naltrexone, atypical antipsychotics (aripiprazole), and selective serotonin reuptake inhibitors, have shown some benefit in reducing NSSI, there remains a lack of well-controlled studies examining the efficacy of medications specifically for NSSI [11]. New therapeutic directions should focus on more inclusive and patient-centered goals to address the challenges of mortality and societal outcomes, which are currently underserved by research [30].

Table 2. Effectiveness of Specialized Psychotherapies for Reducing NSSI and Psychosocial Functioning

Therapy	**Reduction of NSSI/Parasucidal**	**Long-Term Psychosocial Functioning (FFP)**	**Research Gaps/Criticism**
DBT	Moderate effect (Hedges' g −0.622) [26].	Under-researched; need to measure outcomes at the societal level [30].	There is a lack of consistent research on NSSI trajectories during therapy [22].
MBT	Reduction in suicide attempts (data up to 8 years) [27].	General social functioning often remains impaired [27].	Limited evidence for the superiority of short-term MBT over long-term MBT [29].
Other (TFP, ST)	Effective in reducing the severity of BPD [28].	They require the development of therapies tailored to the complex needs of patients with specific BPD profiles [28].	Limited head-to-head comparisons with DBT [28].

4. Discussion

4.1 Integration of Findings: From Mechanisms to Treatment

This comprehensive review synthesized current evidence on non-suicidal self-injury (NSSI) in borderline personality disorder (BPD), examining developmental vulnerabilities, proximal psychological mechanisms, and therapeutic interventions. The findings reveal a complex interplay between neurobiological dysregulation, childhood trauma, dynamic psychological processes, and treatment outcomes that collectively inform our understanding of NSSI in BPD.

The neurobiological evidence consistently demonstrates that NSSI serves a powerful regulatory function through immediate amygdala deactivation and restoration of prefrontal-limbic connectivity [8]. This neurobiological reinforcement mechanism explains the persistent nature of NSSI behavior and why it is so difficult to extinguish through traditional behavioral interventions alone. The fact that NSSI provides rapid, albeit maladaptive, relief from aversive tension underscores the necessity for therapeutic approaches that offer equally rapid yet adaptive alternatives for emotion regulation.

The Emotional Cascade Model (ECM) provides a compelling framework for understanding the proximal triggers of NSSI episodes. The identification of rumination instability, rather than trait rumination alone, as a critical predictor represents a significant advancement in our understanding [15, 16]. This finding suggests that interventions must target not only general emotion regulation skills but also the ability to interrupt momentary escalations in negative affect and rumination. The protective role of emotion differentiation further emphasizes the importance of developing precise emotional vocabulary and affect labeling skills, which have been shown to directly impact neural circuitry through amygdala deactivation [21].

4.2 Developmental Context and Trauma

The etiological architecture revealed by this review highlights childhood trauma as a fundamental risk factor that influences both the development of BPD and the specific risk for NSSI [10, 11]. The finding that childhood maltreatment not only increases NSSI risk but also significantly impairs long-term psychosocial functioning represents a critical clinical consideration. Patients with histories of childhood trauma demonstrate lower GAF scores and more persistent functional deficits, suggesting that trauma-informed approaches must be integrated into standard BPD treatment protocols [15]. The high comorbidity of PTSD in the BPD population (30-50%) and the association with increased NSSI frequency underscores the need for interventions that simultaneously address trauma symptoms and BPD-related dysregulation [16]. Current treatment models that focus primarily on emotion regulation

without adequately addressing underlying traumatic experiences may be insufficient for this substantial subgroup of patients.

4.3 Treatment Efficacy and the Functional Recovery Gap

The evidence for specialized psychotherapies, particularly DBT and MBT, demonstrates moderate to strong efficacy in reducing acute NSSI symptoms. DBT's moderate effect size (Hedges' g = -0.622) in reducing parasuicidal behaviors, combined with evidence of treatment-induced neuroplasticity, confirms that meaningful change is achievable [26, 31]. The demonstration of amygdala deactivation following DBT treatment provides neurobiological validation of the therapy's mechanisms of action and suggests that psychological interventions can indeed modify the neural substrates of emotion dysregulation. However, the most significant finding emerging from this review is the persistent gap between symptom reduction and functional recovery. While MBT demonstrates long-term reductions in suicide attempts, overall social functioning often remains impaired even after 8-year follow-up [27]. This discrepancy between symptomatic improvement and psychosocial functioning represents a critical limitation of current treatment approaches and research priorities. The field has been primarily focused on reducing life-threatening behaviors (suicide attempts, NSSI), which is understandably a clinical priority. However, this narrow focus on symptom reduction may inadvertently neglect broader outcomes related to quality of life, social integration, occupational functioning, and community participation [30]. Patients may stop self-harming yet continue to struggle with maintaining relationships, employment, and meaningful social roles—outcomes that are arguably equally important for recovery and well-being.

4.4 Methodological Considerations and Research Paradigms

The analysis of longitudinal studies examining the relationship between NSSI and BPD reveals significant methodological inconsistencies and differences in clinical populations that hamper causal interpretation [9]. Simple, linear causal models appear insufficient to capture the developmental complexity of BPD and NSSI. The adoption of Complex Dynamic Systems View (CDSV) represents a necessary paradigm shift [20]. CDSV allows for simultaneous analysis of momentary and developmental dynamics, accounting for bidirectional interactions between vulnerability factors and emerging psychopathology.

The emphasis on within-person variability, as demonstrated by experience sampling method (ESM) studies, represents a methodologically sophisticated approach that captures the dynamic, fluctuating nature of NSSI risk [5, 19]. This shift from trait-based to state-based measurement aligns with the clinical reality that NSSI episodes are triggered by momentary

escalations rather than stable characteristics. Future research should prioritize intensive longitudinal designs that can capture these temporal dynamics and identify critical transition points where intervention may be most effective.

4.5 Limitations of Current Evidence

Several limitations of the current evidence base warrant acknowledgment. First, head-to-head comparisons between different specialized psychotherapies (DBT, MBT, TFP, ST) remain limited, making it difficult to determine which approach is most effective for which patient profiles [28]. The field would benefit from comparative effectiveness research that examines patient-treatment matching based on specific clinical characteristics, trauma history, and comorbidity patterns. Second, the majority of treatment outcome studies have focused on female patients, as NSSI prevalence is higher in women with BPD [8]. This gender disparity limits generalizability to male patients with BPD, who may present with different symptom profiles and treatment needs. Third, long-term outcome data remain sparse and of variable quality [28]. Many studies report outcomes at 12-month or 18-month follow-up, which may be insufficient to assess sustained recovery and psychosocial reintegration. The field needs more rigorous long-term studies with comprehensive outcome measures that extend beyond symptom reduction to include functional, occupational, and quality-of-life indicators. Fourth, while neuroimaging studies have provided valuable insights into the neural mechanisms of NSSI and treatment response, these findings are based on relatively small samples and require replication in larger, more diverse populations. The heterogeneity of BPD presentations suggests that neural correlates may vary across patient subgroups.

4.6 Clinical Implications

The findings from this review have several important clinical implications. First, assessment of NSSI in BPD should routinely include evaluation of rumination patterns, emotion differentiation capacity, negative urgency, and trauma history. These proximal mechanisms represent potential therapeutic targets that may enhance treatment outcomes. Second, treatment planning should be informed by developmental considerations, particularly the presence and severity of childhood trauma. Patients with significant trauma histories may require integrated trauma-focused interventions alongside standard BPD treatment protocols. Third, clinicians should recognize that reduction in NSSI frequency, while clinically significant, does not necessarily translate to improved psychosocial functioning. Treatment goals should explicitly include functional outcomes such as relationship quality, occupational stability, and community integration, with progress in these domains monitored throughout

treatment. Fourth, the evidence supporting DBT's efficacy suggests it should remain a first-line treatment for BPD patients with chronic NSSI. However, for patients who do not respond adequately to DBT or who have specific clinical profiles (e.g., severe attachment difficulties), alternative approaches such as MBT or integrated trauma-focused therapy should be considered.

4.7 Future Research Directions

Based on the gaps identified in this review, several research priorities emerge. First, future studies should adopt Complex Dynamic Systems perspectives with intensive longitudinal designs to capture the temporal dynamics of NSSI risk and identify optimal timing for intervention. Second, outcome research must expand beyond symptom reduction to include comprehensive assessment of psychosocial functioning, quality of life, and societal outcomes such as employment, housing stability, and healthcare utilization [30]. Standardized functional outcome measures should be incorporated into all treatment trials.

Third, research is needed to identify mechanisms through which symptom reduction translates—or fails to translate—into functional recovery. Mediation analyses examining how changes in dynamic psychological mechanisms (rumination instability, emotion differentiation) relate to both symptom reduction and functional improvements would be particularly valuable.

Fourth, comparative effectiveness research examining patient-treatment matching is essential. Individual difference factors such as trauma severity, attachment style, comorbidity patterns, and baseline emotion regulation capacity may moderate treatment response and inform personalized treatment selection.

Fifth, neuroimaging research should continue to examine treatment-related neural plasticity, with particular attention to how changes in neural circuitry relate to both symptomatic and functional outcomes. Larger samples and more diverse populations are needed to establish reliable neural markers of treatment response. Finally, intervention research should explore novel approaches that explicitly target the functional recovery gap. This may include interventions focused on social skills, occupational rehabilitation, supported community integration, and peer support programs that address the social isolation and role dysfunction that persist even after symptomatic improvement.

5. Conclusions

Non-suicidal self-injury in borderline personality disorder represents a complex clinical phenomenon driven by neurobiological vulnerabilities, childhood trauma, and dynamic psychological mechanisms. Current evidence supports the efficacy of specialized

psychotherapies, particularly DBT and MBT, in reducing acute NSSI symptoms and inducing measurable neuroplastic changes. However, a critical gap persists between symptom reduction and functional recovery, with many patients continuing to experience significant psychosocial impairment despite reductions in self-harm. Future progress requires a paradigm shift toward Complex Dynamic Systems perspectives that capture the temporal dynamics of NSSI risk, comprehensive outcome measures that extend beyond symptom reduction to include functional and quality-of-life indicators, and intervention research that explicitly targets psychosocial rehabilitation and community integration. Therapeutic approaches must evolve beyond symptom stabilization toward recovery-oriented goals that address the full spectrum of functioning necessary for meaningful life participation. The field stands at a critical juncture where impressive advances in understanding mechanisms and reducing acute symptoms must be matched by equal commitment to achieving sustainable functional recovery and quality of life for individuals with BPD and NSSI.

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Conflict of interest

The authors declare there are no conflicts of interest.

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