

Over-regulation of emotion and poor health outcomes in borderline personality disorder: an integrative review with a neurophysiological perspective

Abstract

Borderline Personality Disorder (BPD) is a serious mental illness characterized by a chronic pattern of instability in interpersonal relationships and self-image, along with a higher baseline of emotional sensitivity and marked impulsivity that begins by early adulthood and persists regardless of context. Emotion dysregulation, a cardinal feature of the disorder, is of high relevance to clinical and medical contexts since it has been correlated with greater risk of parasuicidal behaviors, co-occurring psychiatric illnesses and multiple hospitalizations. Less understood is the role of emotion dysregulation on somatization and medical comorbidity. Physiological correlates of emotion dysregulation, such as decreased heart rate variability (HRV) and cascades of pro-inflammatory cytokines associated to dysphoric mood can increase the risk of medical comorbidity in the BPD population. This mini review assesses and discusses how maladaptive emotion regulation strategies, such as emotional suppression, and BPD-endogenous traits like attachment-seeking behavior can potentiate detrimental health outcomes through physiological correlates and medical self-harm. Future research directions should assess the role of emotional suppression in somatization and medical comorbidity in both sociodemographic and medically diverse BPD populations. In the clinical setting, encouraging reappraisal instead of emotional suppression can help improve treatment outcomes in both psychiatric and medical domains.

Keywords: borderline personality disorder, emotion dysregulation, emotion suppression, somatization, medical comorbidity, metabolic syndrome

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Introduction

Emotion dysregulation is a core feature of borderline personality disorder (BPD).¹ According to Biosocial Theory,² individuals with BPD are physiologically predisposed to experiencing frequent and severe episodes of emotion dysregulation, which are either punished or reinforced by harsh, invalidating social environments from early developmental years and into adulthood. Individuals with BPD frequently present complex traumatic histories of neglect, as well as physical and sexual abuse. As a result, most persons with BPD develop insecure styles of attachment that further predispose them to emotional dysregulation. Many individuals with BPD attempt to regulate these cascades of intense emotion using equally intense, impulsive behavioral strategies, such as cutting or binge-eating, that succeed in downregulating suffering but reinforce the use of self-harm during future instances of emotion dysregulation.

At the physiological level, the cascade of emotional-behavioral dysregulation present in BPD can, like that in bipolar disorder (BD), be considered a source of allostatic load – a neurochemical burden of stress on CNS processes that results in increased levels of oxidative stress and neuroinflammation. As such, findings from the field of BD neuroprogression can inform the physiology of stress in patients with BPD, increasing understandings of how to approach the condition from a medical perspective. For instance, results from studies of BD neuroprogression suggest that acute depression is significantly associated to a surge in pro-inflammatory cytokines (i.e. IL-6, IL-10), anti-inflammatory proteins (i.e. C-reactive protein) and oxidative stress³⁻⁵ in the CNS and peripheral blood circulation. Like BPD, early illness onset and a history of trauma have been found to correlate

significantly with BD clinical severity. However, from the perspective of BD neuroprogression, these clinical correlates also associate with worse illness outcomes, and chronically higher levels of inflammation. As a result, most individuals with mood disorders develop metabolic syndrome – a gateway to medical comorbidities such as diabetes and cardiovascular disease.

Like in BD, a high prevalence of metabolic syndrome is also present in BPD,⁶ suggesting that a similar stress-induced, allostatic load mechanism exists in BPD, a condition characterized by a higher baseline of emotional reactivity, mediated by acute states of amygdala over activation. Notable gender-mediated differences have also been found for the prevalence of metabolic syndrome in BPD populations. Kahl et al.,⁶ found a higher prevalence of hyperglycemia in both genders, but abdominal obesity and hypertriglyceridemia were higher in women with BPD—both associated with chronic stress. Comorbidity with binge eating and major depressive disorders, as well as dysthymia, panic disorder and benzodiazepine dependency, was also significant in the overall sample ($n=135$ BPD). These findings suggest that stress-mediated neuroinflammatory upregulation may also be medically significant in BPD, potentially mediated by dysphoric mood and trait reactivity, leading to detrimental neurophysiological outcomes like higher medical comorbidity in the population.⁷

However, the medical impact of BPD remains largely unexplored in the literature, which this mini review hopes to address by proposing that: 1) chronic issues with emotion dysregulation underlie the prevalence of medical comorbidity in BPD, and 2) The use of suppression and other over-regulatory strategies of emotional management underlie the prevalence of somatization and medical

variants of self-harm in BPD. Emotional suppression was chosen as the focus of this study because it is, reportedly, one of the regulatory strategies most frequently used by BPD patients to manage thought rumination – the tendency to overthink about a problem. Emotional suppression is often ineffective, however, because it increases stress *in tandem* with cognitive load, resulting in increased distractibility/impulsivity and dysphoric mood – risk factors for the self-harm which often occurs in BPD (Figure 1).

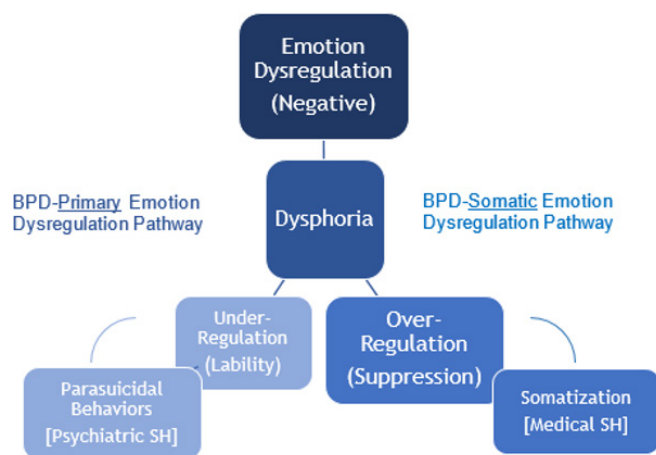


Figure 1 Potential BPD emotion dysregulation pathways leading into psychiatric or medical self-harm.

Over-regulation of emotion as a possible pathway to comorbidity, somatization and Medical Self-Harm (MSH)

The cycle of emotional suppression and Non-Suicidal Self Harm (NSSH) in BPD

Mortality risk associated with BPD is significant: up to 10% of patients complete suicide and regularly practice **non-suicidal self-harm (NSSH)**.⁸ NSSH is a cardinal symptom of BPD which often manifests as impulsive, physically self-injurious behavior (i.e. cutting), practiced with the intention of seeking immediate relief from intense, negative emotions that emerge from a distressing event or as an ineffective coping skill to regulate the pervasive emotional instability endogenous to BPD.¹ Other impulsive behaviors, such as binge-eating and intentional interruption of medical treatment,⁹ can be considered long-term NSSH due to their impact on somatic health and mortality risk. Long-term NSSH may affect somatic health by 1) Triggering physiological reactions, such as increased pro-inflammatory response in blood serum and CNS regions associated to impulse control; 2) Reinforcing the use of ineffective cognitive-affective regulation skills associated to negative health outcomes. According to the Emotional Cascade Model,¹⁰ intense emotion dysregulation typical of BPD is primarily mediated by rumination. Because rumination tends to elicit strong emotional responses, emotional *suppression* – the attempt to inhibit the expression of emotion¹² – is often engaged with to “break the cycle” of rumination and avoid the unpleasant experience of dysphoric mood. However, like a dam suppressing a raging river, the suppressed emotion is more likely to grow in intensity the longer it is suppressed, leading to self-harming behaviors as an equally intense attempt to contain suffering.

Biopsychosocial impact of emotional suppression in BPD

NSSH is not the only consequence of suppressed emotion among BPD patients. Regarding its neurophysiological effects, emotional

suppression in BPD has also been associated to increased sympathetic activation of the cardiovascular system,¹¹ pro-inflammatory responses¹² and decreased heart rate variability (HRV), a marker of cardiovascular health and adaptability to stress.^{12,13} Emotional suppression has also been linked to the clinical severity of personality disorders in general,¹⁴ and in BPD to increased report of physiological symptoms¹² and co-occurrence of somatoform disorders.^{15–17} Ethnicity has also been associated to increased use of emotional suppression,¹⁸ albeit in unexpected ways: both Hispanic and African American women with BPD and co-occurring medical disorders reported significantly less physical health symptoms than Caucasian women with BPD.¹² Therefore, the intersection between ethnicity and BPD diagnosis warrants additional consideration when interpreting the association between emotional suppression and physical health outcomes in at-risk, vulnerable populations, such as Hispanic women with BPD.

Increased baseline arousal and decreased heart rate variability (HRV)

Polyvagal Theory (Porges, Doussard-Roosevelt, & Maiti, 1994) suggests that vagal tone, “the parasympathetic influence on the heart”,¹⁹ is a potential biomarker of emotion regulation in BPD. Operationalized as heart rate variability (HRV), HRV is modulated by attention and emotion regulation strategies like mindful breathing, by activating the myelinated vagal system terminating at the sinoatrial node of the heart.¹⁹ In a study of baseline intensity and reactivity to emotionally evocative stimuli in subjects with BPD, Kuo & Linehan¹⁹ measured HRV by assessing Respiratory Sinus Arrhythmia (RSA), while sympathetic reactivity was assessed through Skin Conductance Response (SCR). Subjects with BPD presented significantly lower basal RSA compared to subjects with social anxiety and healthy controls, along with heightened baseline electrodermal responses and reported more negative emotions, suggesting an increased baseline of negative emotional intensity. Of note, no evidence for heightened reactivity was found in BPD, suggesting that the physiological vulnerability inherent in this condition stems from a higher baseline of emotional intensity as opposed to emotional hyper reactivity. Furthermore, persistent difficulties in emotion regulation account for the intensity of negative emotions experienced by subjects with BPD. Other studies assessing vagal tone in BPD have consistently found decreased HRV in their samples, suggesting decreased cardiovascular resiliency and increased risk for heart disease in this population.

Alexithymia or difficulty identifying emotions

BPD subjects who attempt suicide have been found to have significantly reduced gray matter volume in the left insula, a region associated with interoceptive awareness—the accurate perception of bodily states.²⁰ A lack of interoceptive awareness is known as alexithymia, and it often underlies somatic preoccupation¹⁵ physical health symptoms¹² and clinical severity in personality disorders.¹⁴ Individuals who experience alexithymia are more likely to register emotional experience as a more diffuse type of physical discomfort, and to habitually employ experiential avoidance strategies like emotional suppression to regulate the discomfort. Beblo et al.,²¹ found that individuals with BPD practiced more emotional suppression than non-BPD subjects, irrespective of emotional valence. In other words, they suppressed negative and positive emotions alike, and this suppression was significantly associated with alexithymia and increased negative emotions. Notably, these findings were independent of psychiatric comorbidity with Major Depressive Disorder (MDD) and emphasize the counterproductive effects of suppression as a reinforcer of aversion to negative stimuli.

Conversely, the use of effective emotion regulation strategies has been associated with less emotional distress and decreased blood serum levels of inflammation.²² Reappraisal, or the ability to change how a situation is perceived, has been associated to decreased peripheral blood serum levels of c-reactive protein when compared to subjects who habitually practiced emotional suppression.²² It is relevant to note that skills-training for emotion regulation is a core feature of Dialectical Behavior Therapy (DBT),² the gold standard of BPD treatment, and improved biopsychosocial outcomes may be partly mediated by the health-positive effects of these skills. DBT has been successfully adapted for binge-eating disorder²³ and preliminary results from a novel adherence-focused adaptation of DBT for adolescents with chronic medical illness (DBT-CMI)²⁴ indicate that the intervention is effective, emphasizing the importance of effective emotion regulation in the attainment of positive somatic health outcomes.

Increased use of medical services

According to the DSM-5 (2013), the estimated prevalence of BPD in primary care settings is ~6%. A prevalence of 30% has been reported among patients with chronic pain syndromes,^{25,26} while non-remitted BPD patients frequently present with fibromyalgia, temporal-mandibular joint syndrome, and back pain.²⁷ Likewise, arteriosclerosis or hypertension, obesity, osteoarthritis and cardiovascular disease are significantly associated with BPD.²⁸ Non-remitted patients with BPD have shown higher rates of medical emergency room visits, medical hospitalizations, use of sleep and pain medications,²⁹ and increased suicide attempts in those with any medical condition versus those with BPD alone.²⁸

Sansone et al.,^{9,26} proposed that these patients have developed a somatic form of BPD (S-BPD), characterized by a significantly higher use of healthcare services and high medical comorbidity. Possibly, a more significant prevalence of alexithymia in these individuals has led them to experience somatization or the “abnormal body experience”²⁹ more strongly than individuals with typical BPD. Besides learning to manage emotional dysregulation through over-regulation, also prevalent in somatoform disorders (SD), individuals affected by S-BPD would have a 2-fold increase in medical comorbidity risk due to both the allostatic effects of over-regulation and its distraction-driven behaviors seeking to regulate dysphoric mood (Figure 2).

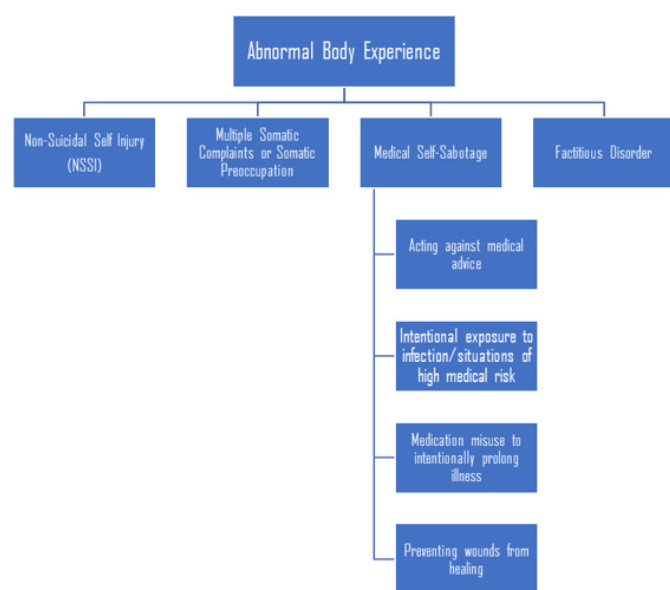


Figure 2 Components of Abnormal Bodily Experience in BPD.²⁹

In lieu of NSSH, medical self-harm appears to healthcare professionals as: a) Perceived “sabotage” of medication adherence and medical advice which prolongs illness course; b) Interfering with healing wounds to elicit or extend caring behaviors from the environment; c) Hostile or aggressive behaviors in the healthcare setting as related to under-regulated stress, dysphoric mood, or in response to stigmatizing attitudes from healthcare personnel. Taken together, these results highlight the insidious burden of medical comorbidity and physical health symptoms on mental health outcomes in individuals diagnosed with BPD and point to a significant need to assess the prevalence of these outcomes in diverse health care settings.

Discussion

Research in the nascent field of BPD medical comorbidity has found significant associations between BPD and increased prevalence of medical comorbidity along with physical health symptoms and frequently co-occurring somatoform disorders. Furthermore, an expansive medical history and increased treatment-seeking behaviors like frequent use of medical services point to a somatic variant of BPD (S-BPD). Less understood are the developmental and psychological mechanisms driving the formation of S-BPD. Possibly, an interaction between the BPD-core component of emotion dysregulation and the over-reliance on emotional suppression mediates the S-BPD pathway. Some study results suggest a link between experiential avoidance, a feature of emotional suppression, and personality disorder severity,¹⁴ leading to an increased risk of mortality. At the physiological level, habitual use of emotional suppression has correlated to increased levels of generalized neuroinflammation.

Likewise, the physiological underpinnings of BPD trait stress can mimic disease states in the CNS. Pro-inflammatory cascades have been found to occur *in tandem* with dysphoric mood symptoms like depression, irritability and impulsive behavior. If pervasive, chronically upregulated states of inflammation can lead to full medical comorbidity with chronic conditions of the musculoskeletal system like osteoarthritis and fibromyalgia, and metabolic disease like hypertension and hyperglycemia. BPD issues are confounded with medical ones as higher rates of emergency room visits and hospitalizations have been found among the BPD population, along with greater use of sleep and pain medications, treatment “sabotage” and an overall disregard for medical advice, suggestive of avoidance-driven behavior and emotional suppression. Thus, the issue of medical comorbidity in BPD is highly relevant to treatment goals that aim to improve quality of life and management of addiction risk. For the clinician, carefully assessing medical history and encouraging the use of reappraisal -the ability to accept a situation or see it in a different way- as an effective emotion regulation strategy can help improve treatment outcomes for BPD patients with somatic issues. Likewise, clinical research targeting the role of alexithymia and emotional suppression in decreased HRV and medical comorbidity can clarify best practices for BPD psychological and medical interventions.³⁰

Conclusion

A significant link exists between the use of emotional suppression as a regulatory strategy and both increased somatization and medical comorbidity among individuals with BPD. Furthermore, a somatic variant of BPD appears to explain a subset of patients with voluminous medical records, number of visits to ER and increased use of sleep or pain medication. Because BPD patients with comorbid somatization disorders tend to score higher on measures of alexithymia, prioritizing emotion recognition as a treatment target can help improve pain management and medical treatment effectiveness in patients with

S-BPD, a more medically prevalent version of the condition, while also decreasing incidence of medical self-harm.

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Conflict of interest

The author declares there is no conflict of interest.

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