HOW DO WE TARGET NSSI IN BPD? EXPLORING THE RELATIONSHIP BETWEEN EMOTION DYSREGULATION, INTERPERSONAL DYSFUNCTION, AND NON-SUICIDAL SELF-INJURY

by

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How Does Non-Suicidal Self-Injury Remit in BPD? Exploring the Relationship between Emotion Dysregulation, Interpersonal Dysfunction, and Non-Suicidal Self-Injury

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Abstract

The current research investigated: 1) the trajectory of changes in emotion dysregulation, interpersonal dysfunction, and nonsuicidal self-injury (i.e., NSSI) over the course of DBT, and 2) whether changes in emotion dysregulation mediate the recovery of other features of BPD in treatment. Individuals with BPD (N = 120) enrolled in a multi-site study were assessed at five timepoints over 12 months of dialectical behaviour therapy (i.e., DBT). Results indicated that interpersonal dysfunction and NSSI decreased linearly over the course of DBT. Emotion dysregulation decreased in a quadratic manner; most of the gains in emotion dysregulation may occur in earlier phases of DBT. Results also revealed that although changes in emotion dysregulation was not a significant mediator of the relationship between changes in interpersonal dysfunction and in NSSI, changes in interpersonal dysfunction predicted changes in emotion dysregulation. Future research directions regarding NSSI, emotion dysregulation, and interpersonal dysfunction within DBT are discussed.

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Introduction

Borderline Personality Disorder (BPD) is a severe mental disorder with an estimated lifetime prevalence of 2-6% in the population that is characterized by significant behavioural, emotional, and interpersonal dysregulation (Ekselius et al., 2001; Zimmerman, Rothschild, & Chelminski, 2005). BPD is associated with intensive and costly healthcare service utilization and suicidality, and individuals with this disorder are often hospitalized due to the severity and instability of the associated behaviours (Bender et al., 2001; Evren et al., 2011; Scott et al., 2017; Clarke, Hafner, & Holme, 1995). One of the criteria found to contribute most highly to the high hospitalization and psychiatric health-care utilization rates in BPD is non-suicidal self-injury (NSSI) behaviours (Sansone, McLean, & Widerman, 2008). NSSI is estimated to occur in approximately 65-80% of individuals with BPD (Soloff, Lis, Kelly, Cornelius, & Ulrich, 1994). Previous research has found that participants endorsing current NSSI had greater psychological distress (Mars et al., 2014; Zielinski, Hill, & Veilleux, 2013) and increased depression, panic, anxiety, and alcohol misuse compared with individuals who last self-injured over a year prior (Zielinski, Hill, & Veilleux, 2013). Additionally, NSSI is associated with an increased risk of suicide (Baetens et al., 2014; Klonsky, May, & Glenn, 2013; Muehlenkamp & Gutierrez, 2007; Whitlock et al., 2013). A review of intentional injury in Canada in 2004 found that the total costs of intentional self-injury, including suicide and self-inflicted injuries, amounted to $2.442 billion in direct (i.e., hospitalization, health services) and indirect (i.e., mortality, permanent disability) costs (SMARTRISK, 2009). Thus, it is of utmost importance that further research be dedicated to understanding this BPD feature in greater detail, in order to minimize future economic, social, and emotional costs. Toward this end, the present study examined changes in the frequency of

NSSI, as well as other behaviours theoretically proposed to contribute to NSSI, over the course of 12-months of DBT for individuals with BPD.

Dialectical Behavior Therapy (DBT; Linehan, 1993), a comprehensive, 12-month cognitive-behavioural treatment for BPD, is arguably the most empirically-supported treatment for BPD and suicidal behaviors. DBT adopts a dialectical approach, which emphasizes acceptance and validation of the patient, while maintaining a focus on changing maladaptive behaviours such as NSSI. This dialectical balance stems from a recognition that not only is acceptance of one's experience integral to their psychological well- being, but that a primarily change-focused therapy would feel invalidating to patients with BPD as well (Linehan, 1993). While change-based strategies within DBT involve traditional behavioural modification strategies via behavioural skills training and the extinction of maladaptive behavioural responses, the acceptance-based strategies within DBT are derived from client-centered therapeutic approaches and Zen practice.

To date, there are 17 published randomized controlled trials (RCT) that test the comparative efficacy of standard, comprehensive, or modified DBT for BPD or BPD traits and suicidal behaviours (Linehan, 2016). Extant research provides substantial support for the efficacy of DBT in reducing a range of behaviours associated with BPD, such as self-harm, suicide attempts, and severity of suicidal ideation. Indeed, DBT and DBT skills training groups have been found in several RCTs and effectiveness trials to be effective in reducing non-suicidal self-injury outcomes (e.g., van den Bosch, Koeter, Stijnen, Verheul, & van den Brink, 2005; Koons et al., 2001; Linehan et al., 1991; Linehan et al., 2002; Linehan et al., 2006; McMain et al., 2009; McMain et al., 2017; Mehlum et al., 2014; Priebe, Bhatti, Barnicot, Bremmer, Gaglia et al., 2012; Verheul et al., 2003) compared to treatment-as-usual, up to 6-months and 12-months post-

treatment (Koons et al., 2001; Linehan et al., 2002; Linehan, Heard, & Armstrong, 1994; Verheul et al., 2003; Priebe et al., 2012).

Despite extensive research on DBT and its effects on non-suicidal self-injurious behaviours for individuals with BPD, the specific underlying process through which DBT leads to a reduction in the frequency of NSSI remains largely unexamined. At present, little is known about the pathway through which NSSI decrease in DBT; hence, an important step in the effort to further target NSSI requires a more nuanced investigation into the trajectory of NSSI, as well as associated processes or areas of dysfunction, over the course of treatment.

Characteristics of NSSI

Non-suicidal self-injury (NSSI) are self-directed and deliberate behaviours causing harm or destruction to bodily tissue, performed without the intent to die (Brickman, Ammerman, Look, Berman, & McClosky, 2014; Yates, 2004). NSSI is often chronic and occurs in the form of methods such as cutting, head banging, burning, scratching, or biting (Bracken-Minor & McDevitt-Murphy, 2014). NSSI is associated with a wide range of psychopathologies, including eating disorders and depression, but also may be present in the absence of a psychiatric diagnosis (Bentley et al., 2015; Claes et al., 2012). Other research has found that most adolescent inpatients who engage in NSSI also meet criteria for major depressive disorder (MDD; 42%), posttraumatic stress disorder (24%), or substance use disorder (60%; Nock et al., 2006).

Notably, NSSI is particularly associated with BPD (Bracken-Minor & McDevitt-Murphy, 2014; Cerutti et al., 2011; Klonsky, Oltmanns, & Turkheimer, 2003). Indeed, 50-73% of individuals with BPD endorse a history of NSSI (Chapman, Specht, & Celluci, 2005; Snir, Rafaeli, Gadassi, Berenson, & Downey, 2015; Zanarini et al., 2007). Chapman, Specht, and Cellucci (2005) also found that women with BPD may have a higher lifetime frequency and

earlier onset of NSSI than those who do not have BPD. Moreover, in a study conducted by Glenn and Klonsky (2011), BPD features were among the variables that were found to prospectively predict future engagement in NSSI. BPD severity has also been associated with higher likelihood of repetitive engagement with NSSI (Muehlenkamp, Ertlelt, Miller & Claes, 2011). The extensive economic, emotional, social, and behavioural costs of NSSI, along with the strong relationship between BPD criteria and NSSI, necessitate further study into identifying the particular factors, which serve as the triggers and mechanisms of NSSI for individuals with this disorder.

Understanding NSSI: Interpersonal Dysfunction as a Key Precipitant of NSSI

Extant research suggests that interpersonal dysfunction may be one of the key precipitants leading to NSSI. Interpersonal dysfunction is one of the core features of BPD and is the most endorsed and persistent criterion (Zanarini et al., 2007; Choi-Kain et al., 2010). It is defined as a pattern of unstable and intense interpersonal relationships, characterized by vacillation between extremes of idealization and devaluation of others, as well as frantic efforts to avoid real or imagined abandonment (APA, 2013). Interpersonal dysfunction in BPD is strongly associated with negative outcomes such as depression and difficulty forming long-term intimate relationships (Gremaud-Heitz et al., 2014; Daley, Burge & Hammen, 2000). Most remarkably, it is implicated in contributing to NSSI (Jeung & Herpertz, 2014).

Firstly, cross-sectional studies have reported that difficulties in interpersonal effectiveness and interactions are correlated with NSSI engagement. For instance, individuals engaging in NSSI report greater social skills deficits and poorer social problem solving than individuals who do not engage in NSSI (Nock & Mendes, 2008; Claes et al., 2010, as cited in Muehlenkamp, Brausch, Quigley, & Whitlock, 2013). In Nock and Mendes's (2008) study,

adolescents with a history of NSSI were found to select more negative solutions to social problems and rate their self-efficacy for performing adaptive solutions to these social problems as significantly lower than adolescents who did not self-injure. Kim and colleagues (2015) found that adolescent psychiatric inpatients with a history of NSSI without a history of suicide attempts experienced greater subjective interpersonal distress when given a behavioural task that simulated interpersonal conflict than psychiatric adolescent inpatients with a recent suicide attempt (and no history of NSSI), and community-based, typically developing controls (Kim et al., 2015). Consistent with these findings, Muehlenkamp, Ertlelt, Miller, and Claes (2011) found that, in a sample of outpatient adolescents from a depression and suicide clinic, participants who reported greater levels of interpersonal chaos (i.e., fear of abandonment and unstable relationships) were more likely to engage in NSSI. Additionally, poor relationships with close others has been found to correlate with NSSI. For instance, Muehlenkamp and colleagues' (2013) found that, in a sample of college students, individuals who engaged repetitively in NSSI reported receiving significantly lower perceived social support from family members (Muehlenkamp, Brausch, Quigley, & Whitlock, 2013).

In sum, the correlational nature of these studies indicates that interpersonal dysfunction and NSSI are related. However, what is a little less clear is the directionality of this relationship. Thus, in order to further examine the temporal relationship between interpersonal dysfunction and NSSI, emerging research has also investigated whether interpersonal dysfunction may act as a *trigger* or *precipitant* to NSSI. Indeed, extant studies have implicated the role of poor relationships with close others as a predictor of future NSSI engagement. For example, Tatnell and colleagues (2014) conducted a study in an adolescent sample aged 12-18 years where paper-and-pencil questionnaires were administered twice: once at baseline, and again at 12-months

post-baseline (Tatnell, Kelada, Hasking, & Martin, 2014). Within the sample, adolescent participants were classified as having *initiated* NSSI if they did not report a history of NSSI at baseline but reported NSSI at follow-up (i.e., onset of NSSI) and were classified as having *ceased* NSSI if they reported NSSI at baseline but subsequently reported that their most recent episode occurred more than 12 months prior at follow-up (i.e., cessation of NSSI). Tatnell and colleagues (2014) found, through logistic regression, that lower perceived family support at baseline predicted the onset of NSSI over the course of the study, and that higher levels of perceived family support at baseline predicted the cessation of NSSI by the end of the study. Similarly, You and Leung (2012) found that, in a sample of Chinese community adolescents followed over 2 years, family invalidation at Year 1 was significantly associated with the occurrence of NSSI in Year 2. Furthermore, peer victimization has also been found to be predictive of NSSI engagement. In a sample of adolescents aged 13 to 15 years, Jutengren and colleagues (2011) reported a temporal precedence between the engagement of deliberate self-harm after experiencing peer victimization (Jutengren, Kerr, & Stattin, 2011). Relatedly, within a community sample of youth studied longitudinally through adolescence, poor relationship quality, including lack of social support and negative interactions with close others, was one of four factors that predicted prospective onset of NSSI at the 2.5-year follow-up (Hankin & Abela, 2011). Moreover, poor relationship quality continued to persist as a prospective predictor of new NSSI, even after controlling for reported suicidality over the 2.5-year follow-up (Hankin & Abela, 2011). In sum, there is a compelling literature suggesting that in adolescent and young adult populations, interpersonal difficulties and lack of social support act as predictors of NSSI onset and maintenance.

Understanding NSSI: Emotion Regulation and Emotion Dysregulation

Emotion regulation. Emotions are multi-faceted responses, generated by an individual's evaluation of their situation as relevant to a currently active goal (Lazarus, 1991). They are phenomena which, apart from involving changes in subjective and physiological experience, may also inspire or incline us to act (Frijda, 1986; Gross, 2013). *Emotion regulation*, therefore, refers to the actions taken to influence emotion experience, "shaping which emotions one has, when one has them, and how one experiences or expresses these emotions" (Gross, 2013). According to the process model of emotion regulation, first, an emotional response occurs, and then is followed by an assessment of the response as "good" or "bad" (Gross, 2013). This assessment then leads to the activation of 1) a goal to alter the emotion response trajectory and 2) subsequent engagement in the process to reach this goal. This means that, contrary to the belief that the construct of emotion regulation equates to the lack of negative emotion, emotion regulation goals can actually include efforts to down-regulate or up-regulate either negative or positive emotion (Gross, 2013). Thus, the ability to emotionally regulate indicates an ability to maintain goal-directed behaviour regardless of the valence of one's emotions (Fruzzetti, Crook, Erikson, Lee, & Worrall, 2009).

Emotion dysregulation. In contrast, *emotion dysregulation* is a broader construct that captures abnormalities in emotional responding (i.e., emotional reactivity and baseline emotional intensity), as well as difficulties with adaptive emotion regulation (Gross, 2013). According to several theoretical models, emotion dysregulation is the fundamental feature that underlies NSSI.

Perhaps the most prominent of these models is Linehan's (1993) Biosocial model of BPD. According to this model, emotion dysregulation underlies *all* dysregulated behaviours in BPD, including NSSI (Linehan, 1993). That is, BPD-relevant criteria are either a direct result of one's emotion dysregulation (e.g., intense anger, interpersonal chaos) or function as maladaptive

emotion regulation strategies (e.g., self-harm, dissociation). Linehan proposes that individuals with BPD have a biological vulnerability to emotion dysregulation that, in transaction with an invalidating environment, leads to escalations in emotion dysregulation, and eventually, BPD (Linehan, 1993). An invalidating environment refers to a chronic rearing environment in which caregivers are intolerant and/or dismissive toward the individual's behaviours (both overt and covert behaviours – e.g. emotions, internal experiences) independent of the actual validity of the behaviour. For the individual with BPD, this transaction of biological vulnerability and an invalidating environment results in an inability to problem-solve when faced with aversive emotions or internal experiences, and subsequent deficits in emotion regulation skills, and dysregulation in emotional response. This inability to regulate emotions in an adaptive way leads individuals with BPD to rely on dysfunctional emotion regulation strategies, such as NSSI, to help alter their emotional experience to be more bearable. NSSI is thus conceptualized as a dysfunctional behaviour that functions to regulate negative emotion (Linehan, 1993).

In contrast to the Biosocial model, rumination, the cognitive process of perseverating on a negative situation, is highly featured within the Emotional Cascade Model, proposed by Selby and Joiner (2009). According to this model, rumination in response to intense negative affect leads to further intensification of said negative affect, which leads to further rumination, and so on – until the individual decides to distract themselves with impulsive dysregulated behaviour, such as self-harm (Selby & Joiner, 2009). This dysregulated behaviour functions to interrupt the rumination-emotion intensification cycle and provides emotional relief from painful internal experiences (Selby & Joiner, 2009). Thus, within this model, Selby and Joiner also propose that NSSI engagement in BPD is a result of attempts to regulate emotion.

Although the previous two models describe NSSI in the context of a BPD diagnosis, the Experiential Avoidance Model, proposed by Chapman, Gratz, and Brown (2006), proposes a model about the functions of NSSI independent of a clinical diagnosis. In this model, the key feature is experiential avoidance, described as an individual's unwillingness to process private internal events, and their subsequent motivation to deliberately change their experience of these private internal events. Much like the Biosocial model, the Experiential Avoidance model postulates that it is the relief that individuals experience from the discomfort of aversive emotional arousal and internal experiences that lead to increased deliberate self-harm. In this model, deliberate self-harm is one of many forms of escape or avoidance of internal experiences (i.e., a form of emotion regulation) (Chapman et al., 2006).

Thus, these models converge in their conceptualizations that emotion dysregulation is a primary mechanism that contributes to NSSI. With the Biosocial model, Linehan (1993) proposed that emotion dysregulation is the core feature that directly impacts all criteria of BPD, and therefore impacts NSSI. Much like the Biosocial model, the Experiential Avoidance model posits that NSSI serves as an escape from internal covert behaviours. Finally, in the Emotional Cascade Model, rather than proposing that emotion dysregulation is the central component of BPD, Selby and Joiner (2009) contend that emotion dysregulation is a core element of a broader emotional cascade, which ultimately leads to behavioural dysregulation, which manifests itself in behaviors such as NSSI.

The theoretical relationship between NSSI and emotion dysregulation within BPD is further supported by empirical evidence. Self-report research suggests that, although individuals who engage in NSSI do so for a variety of reasons, the primary function of NSSI is to regulate intolerable emotional arousal or tension (McKenzie & Gross, 2014; Kleindienst et al., 2008;

Brown, Comtois, & Linehan, 2002). For instance, Nock and Prinstein (2004, 2005) found that the most frequently endorsed reasons for engagement in NSSI were: 1) intrapersonal self-regulation, 2) the reduction of painful or aversive internal sensations (i.e., "to stop bad feelings" and "to relieve feeling numb or empty") or 3) to generate desired internal sensations (i.e., "to punish yourself", "to feel relaxed", and "to feel something, even if it was pain"). These findings were replicated across adolescent and adult BPD clinical samples (Garcia-Nieto, Carballo, de Neira Hernando, de Leon-Martinez, & Baca-Garcia, 2015; Kleindienst et al., 2008; Sadeh et al., 2014). Kleindienst et al. (2008) found that 51% of BPD adults with a history of NSSI specified the reduction of "aversive tension" as their primary motivation for NSSI, followed by reduction of unpleasant feelings (13%). Sadeh et al. (2014) also found that, among youth seeking treatment for NSSI and BPD behaviours, one of the most-endorsed functions of NSSI was the intrapersonal function of emotion regulation (Sadeh et al., 2014). Thus, extant data are consistent with theory proposing that NSSI functions as a maladaptive emotion regulation strategy.

These self-report findings are further supported by psychophysiological laboratory evidence. Nock and Mendes (2008) found that individuals with a past history of NSSI, and more specifically, those who reported engaging in NSSI to decrease aversive emotional arousal, experienced greater physiological arousal in response to a stressful task relative to those with no past history. Given that these individuals endorse using NSSI as an emotion regulation strategy, and also experience high emotional reactivity in response to stress, it follows that these individuals would likely utilize NSSI to regulate strong physiological arousal. Additionally, studies using samples of individuals with BPD (Welch et al., 2008) and NSSI (Haines et al., 1995) have found that participants exhibit a decrease in autonomic emotional arousal following an imagined episode of NSSI.

Moreover, studies examining the self-reported emotional states before and after NSSI engagement further demonstrate that, after an episode of NSSI, negative emotions tend to decrease. For instance, in a sample of adults endorsing a history of self-harm, Gordon and colleagues (2010) reported that individuals who endorsed more deliberate self-harm episodes also endorsed feeling calmer and more attentive, and less fear and distress, following their most recent self-injury episode. Indeed, these findings suggest that individuals with a greater history of self-injury episodes feel more positive and less negative internal experiences following their most recent episode. Likewise, Kleindienst and colleagues (2008) compared self-reported emotional states before and after NSSI episodes in a sample of women with BPD and found that guilt, shame, and several other self-reported negative feelings (i.e., strong tension, strong pressure, emptiness, loneliness, depression, dejection, sadness, anger, disgust, numbness, mortification, dissociative feelings) significantly decreased with the engagement of NSSI. Eight-five percent of patients reported feeling more "relieved" after engaging in NSSI, and almost no participants reported having positive feelings before engaging with NSSI (Kleindienst et al., 2008). These findings are consistent with those reported in a study by Chapman and Dixon-Gordon (2007), which found that, in a sample of female inmates with a history of deliberate self-harm, suicide attempt, or ambivalent suicide attempts, the largest proportion of individuals reported feelings of relief immediately following deliberate self-harm episodes. An ecological momentary assessment study conducted by Vansteelandt and colleagues (2017) found that individuals who engaged in NSSI demonstrated more variability in affect valence and activation than individuals who did not, suggesting that individuals who engaged in NSSI experienced more affect instability than those who did not (Vansteelandt et al., 2017). However, the authors found that individuals who engaged in NSSI more frequently demonstrated *less* variability in

affect valence and activation than individuals who did not engage in NSSI as frequently. Thus, collectively, these findings suggest that, among individuals who *do* engage in NSSI, those who engage *more frequently* with NSSI may find that the behaviour actually helps them stabilize their affect and reduce variability in their emotions.

Finally, longitudinal research has also examined the relationship between initial emotion dysregulation and NSSI over time. A longitudinal study in a sample of community-based adolescents found that continuation of NSSI was associated with lower baseline cognitive reappraisal (i.e., poorer ability to change how one thinks about a distressing situation to reduce feelings of distress) and higher emotional suppression (i.e., limiting emotional expression) (Andrews, Martin, Hasking, & Page, 2013). Given that cognitive appraisal and emotional suppression are both key emotion regulation strategies, these results suggest that the use of maladaptive emotion regulation strategies and the deficit in adaptive emotion regulation strategies are related to NSSI maintenance. This finding was replicated in Peters and colleagues' (2016) study, which found that baseline affective instability predicted future NSSI in a general population sample, where affective instability not only predicted the onset of NSSI in participants who did not have a previous history of NSSI, but also predicted the continuation of NSSI in participants who did have a history of NSSI at the 18-month follow-up (Peters, Baetz, Marwaha, Balbuena, & Bowen, 2016). Additionally, Baetens and colleagues' (2014) longitudinal study found that higher psychological distress at baseline was associated with subsequent NSSI (one year and two years later) (Baetens et al., 2014). These longitudinal studies further establish the temporal precedence of emotion dysregulation prior to NSSI. Although there are no experimental longitudinal studies which indicate an explicitly causal relationship between emotion dysregulation and NSSI (e.g., through directly manipulating emotion dysregulation), the

longitudinal correlational evidence does point to emotion dysregulation or emotion regulation difficulties preceding NSSI, which indicates a directional relationship. In sum, both theory and evidence suggest that one of the core mechanisms underpinning NSSI is its function as an emotion "regulation" strategy.

Emotion Dysregulation, Interpersonal Dysfunction, and Their Relationship with NSSI.

Research also suggests that emotion dysregulation is highly related to, and may theoretically underlie, BPD features such as interpersonal dysfunction. For instance, in studies of couples, there has been an established relationship between difficulties regulating negative emotions and lower relationship satisfaction (Bodenmann & Cina, 2006; Bodenmann, Ledermann, & Bradbury, 2007). Consistently, extant data suggest that, among individuals with BPD features, emotion dysregulation is the mechanism through which interpersonal dysfunction and other BPD features are related. For instance, Herr and colleagues (2013) found that emotion regulation difficulties mediated the relationship between BPD feature severity and interpersonal dysfunction, indicating that emotion regulation difficulties accounted for the pathway through which individuals with BPD experience more interpersonal dysfunction (Herr, Rosenthal, Gieger, & Erikson, 2013). In a qualitative study examining video-recorded conversations between women with BPD and their partners, Miano, Grosselli, Roepke, and Dziobek (2017) found that women with BPD were more emotionally reactive and experienced higher stress levels during threatening conversations with a romantic partner (regarding the relationship or personal matters) than women in the healthy control condition. These higher stress levels for women with BPD predicted more negative communication behaviours and feelings compared to women in the healthy control condition (Miano, Grosselli, Roepke, & Dziobek, 2017). This also suggests that interpersonally threatening situations may lead to emotional reactivity and higher

stress levels, which then lead to poorer communication behaviour. Thus, collectively, emerging evidence suggests that, within BPD, there exists a relationship between emotion dysregulation and maladaptive interpersonal behaviours, and specifically, that emotion dysregulation may mediate the relationship between interpersonal dysfunction and other BPD features such as NSSI. Thus, based on the current literature reviewed above, one could expect greater interpersonal dysfunction would lead to greater NSSI, as mediated by greater emotion dysregulation (i.e., an intrapersonal model of NSSI), as depicted in Figure 1.

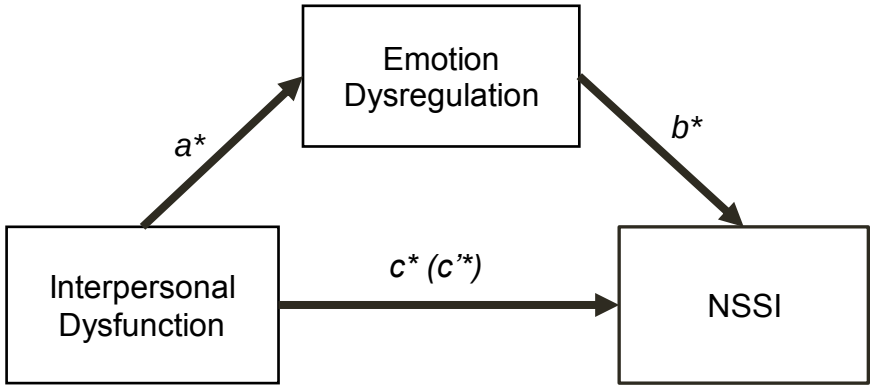

Figure 1. Proposed pathways (i.e., mediation model) between interpersonal dysfunction, emotion dysregulation, and NSSI.

* The *a* path is the effect of interpersonal dysfunction on emotion dysregulation, the *b* path is the effect of emotion dysregulation on NSSI, the *c* path is the direct effect of interpersonal dysfunction on NSSI. The *c'* path in parentheses is the indirect effect of interpersonal dysfunction and NSSI through the indirect path of emotion dysregulation.

In sum, extant literature suggests that emotion dysregulation is the mediator through which interpersonal dysfunction influences NSSI. Understanding this pathway will ultimately

allow us to understand the mechanism through which NSSI remits over the course of DBT treatment.

Changes with Treatment

Based on the proposed models illustrated in Figure 1, this thesis seeks to examine how these three variables of interest *change* over the course of DBT, and the relationships between changes among these variables. Currently, theoretical models and the extant literature suggest that emotion dysregulation may be the hypothesized mechanism to underpin BPD criteria, including interpersonal dysfunction and NSSI (Figure 1). Thus, it is proposed that *changes* in emotion dysregulation will mediate the relationship between *changes* in interpersonal dysfunction and *changes* in NSSI. That is, by studying the variables within the context of change through treatment, it is hypothesized that the pathway through which NSSI decrease over the 12-months of DBT would first begin with improvements in interpersonal dysfunction, followed by a reduction in emotion dysregulation, with, finally, a reduction in NSSI frequency (i.e., an intrapersonal regulation model of changes in NSSI in treatment). This new pathway is highlighted in Figure 2.

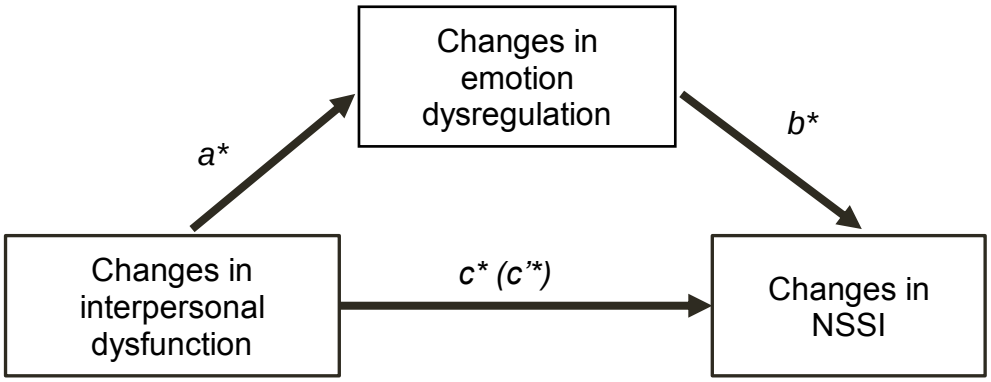

Figure 2. Proposed pathways (i.e., mediation model) between changes in emotion dysregulation, changes in interpersonal dysfunction, and changes in NSSI.

* The *a* path is the effect of changes in interpersonal dysfunction on changes in emotion dysregulation, the *b* path is the effect of changes in emotion dysregulation on changes in NSSI, the *c* path is the direct effect of changes in interpersonal dysfunction on changes in NSSI. The *c'* path in parentheses is the indirect effect of changes in interpersonal dysfunction and changes in NSSI through the indirect path of changes in emotion dysregulation.

Consistent with the proposed model (Figure 2), emerging research has begun to explore whether changes in emotion regulation lead to changes in NSSI (Gratz, Levy, & Tull, 2012; Voon, Hasking, & Martin, 2014). Gratz, Levy and Tull (2012) examined the efficacy of Emotion Regulation Group Therapy (EGRT) in a sample of individuals meeting criteria for BPD and endorsing a history of deliberate self-harm and found that changes in emotion dysregulation mediated the reductions in deliberate self-harm frequency over the course of the treatment. They also found that the growth patterns of emotion dysregulation and deliberate self-harm were related, and that changes in emotion dysregulation over the course of EGRT mediated changes in deliberate self-harm frequency (Gratz, Levy, & Tull, 2012). Research has also begun investigating how changes in emotion regulation might act as a mechanism of change for interpersonal problems. Keating, Muller, and Classen (2017) conducted a study with 36 adult women from the Women Recovering from Abuse Program (WRAP; Duarte Giles et al., 2007), examining their levels of attachment, emotion dysregulation, and interpersonal problems pre- to post-treatment. They found that changes in emotion dysregulation mediated the relationship between changes in attachment and changes in interpersonal problems (Keating, Muller, & Classen, 2017).

Present Aims

The proposed study had three study aims. The first aim was to examine how emotion dysregulation, NSSI frequency, and interpersonal dysfunction each change over the course of DBT. Specific hypotheses are below:

- **Hypothesis 1a:** There will be a reduction in emotion dysregulation over the course of 12 months of comprehensive DBT.
- **Hypothesis 1b:** There will be a reduction in the interpersonal dysfunction over the course of 12 months of comprehensive DBT.
- **Hypothesis 1c:** There will be a reduction in NSSI frequency over the course of 12 months of comprehensive DBT.

The second study aim was to test a mediational model of the relationship between changes in emotion dysregulation, interpersonal dysfunction, and NSSI frequency, with changes in emotion dysregulation as a mediator. As illustrated in Figure 2, the specific hypothesis is:

- **Hypothesis 2**: The association between reductions in interpersonal dysfunction and reductions in the frequency of NSSI will be mediated by reductions in emotion dysregulation.

Method

This study consisted of secondary analysis from a multi-site (Toronto and Vancouver), single blind, two-arm RCT (McMain et al., 2018) examining the clinical and cost-effectiveness of 6 months of DBT versus 12 months of DBT.

Participants

Participants were recruited through treatment and research waiting lists at the Centre for Addiction and Mental Health (CAMH) in Toronto, Ontario, and the Personality and Emotion Research Laboratory (PERL) in Burnaby, British Columbia, advertisements at health service

centres, and through word of mouth referrals. Participants included the subset of individuals randomized to the 12-month DBT condition (*n*=120). Given that 12 months of DBT is the standard and most common iteration of DBT administered to individuals with BPD who engage in NSSI, and it is currently unclear whether it is effective within an abridged format, we examined change in the three variables of interest in the 12-month condition only.

Inclusion criteria. Participants were deemed eligible to participate if they fulfilled the following inclusion criteria: a) aged 18-65; b) met criteria for Borderline Personality Disorder; c) engaged in at least two episodes of self-injury or suicide attempts in the last 5 years, including one of which must have occurred in the 8 weeks prior to study screening; d) were proficient in English; e) did not receive more than 8 weeks of standard DBT in the past year; and f) had either Ontario Health Ontario Health Insurance Plan (OHIP) coverage or British Columbia Medical Services Plan (MSP) health insurance for one year.

Exclusion criteria. Exclusion criteria included those that might interfere with the participant's ability to engage with and understand the treatment being offered. If the participant met the following criteria, they were excluded from the study: a) DSM-IV criteria for specific psychotic disorder, bipolar disorder I, or dementia; b) an IQ of less than or equal to 70; c) had chronic or serious physical health problem requiring hospitalization within the next year; and d) had plans to move to another province other than Ontario or British Columbia within the duration of the study.

Procedure

Intervention. The data used for the proposed study included only the participant data from those enrolled in the DBT-12-month condition. DBT-12 is a 12-month comprehensive treatment (Linehan, 1993), and consists of four components. Each participant attended a weekly

one-hour individual therapy session, where the therapist's goal is to improve client motivation to change, and to assist the client in practicing behavioural skills in daily life and received telephone consultation to receive between-sessions skills coaching from their therapist as needed, to assure generalization of behavioural skills to their natural environment. These participants also participated in a two-hour weekly behavioural skills training group, with skills modules in mindfulness, emotion regulation, interpersonal effectiveness, and distress tolerance, and which operates in 6-month cycles (thus, 12-month participants received two cycles of this skills training group). Participants were enrolled in these ongoing groups depending on availability, meaning that skill modules were not presented sequentially. Therefore, the timing of, or ordering in presentation of skill modules was not a confounding factor impacting trajectory of change for any of the three variables of interest. Finally, study therapists participated in weekly therapist consultation meetings to provide support for therapists and to enhance therapist motivation and capabilities.

Therapists. The treatment was administered by doctoral- and master's-level therapists who had a minimum of 2 years of experience with DBT and have participated in advanced-level DBT workshops. Therapists were not crossed over treatment condition; however, to control for possible confounding effects of therapist characteristics, therapists across both 6-month DBT and 12-month DBT conditions were matched on a number of factors, including expertise, training in DBT, and availability of supervision. To ensure treatment fidelity, supervision and consultation in the parent study were provided by certified practitioners with the Linehan Board of Certification and Accreditation.

Treatment Adherence. Treatment fidelity (i.e., treatment competence and treatment adherence) were evaluated using treatment adherence ratings in the *Dialectical Behaviour*

Therapy Adherence Rating Scale (Linehan & Korslund, 2003). This scale, which evaluates sessions across a range of DBT strategies, were used by treatment-masked coders trained to reliability. A random selection of 5% of sessions (individual and group) from each therapist-client dyad were rated. As well, 5% of the coded sessions underwent calibration checks with the Gold Standard rater to prevent against coding drift.

Measures

Participants were assessed at 5 time points, at 3-month intervals: from baseline (i.e., pre-treatment) to post-treatment (i.e., 12 months).

Assessment of inclusion/exclusion criteria. Personality disorder criteria was assessed using the *International Personality Disorder Exam – BPD Module* (IPDE-BPD; Loranger, 1995). The IPDE-BPD is a semi-structured interview of personality disorders based on the World Health Organization (WHO) International Classification of Diseases, 10th revision (ICD-10; cite), and the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV; cite). The IPDE assesses for traits and behaviours related to BPD pathology through a series of open-ended questions, for which a rating of 0 (absent or normal), 1 (exaggerated or accentuated), or 2 (pathological), is then assigned. In order for a diagnosis of BPD to be assigned, a minimum of five BPD-related behaviours must have been present over the past 5 years, with one behaviour required to have been present before the age of 25 (Loranger, Janca, & Sarlorius, 1997). The IPDE-BPD is considered a reliable measure of BPD pathology, with a high temporal stability of .82, and high inter-rater reliability of .90 (Mann et al., 1999). The IPDE is also considered highly valid, with IPDE-BPD scores correlating highly with self-report measures of BPD pathology (i.e., Inventory of Clinical Personality Accentuations BPD subscale; Schroeder, Andresen, Naber, and Huber, 2010).

To assess total frequency of NSSI over the lifespan, the *Lifetime Suicide Attempt – Self-Injury Interview* (L-SASII; Linehan & Comtois, 1996) was administered at baseline. The L-SASII is a 20-minute interview used to obtain a lifetime history of the frequency, medical severity, intent, and precipitants of self-injurious acts. It has been used in numerous experimental studies in the assessment of NSSI (Carter, Willcox, Lewin, Conrad, & Bendit, 2010; Chapman & Dixon-Gordon, 2007; Chapman, Specht, & Cellucci, 2005; Dougherty et al., 2009; Jacobson, Muehlenkamp, Miller & Blake Turner, 2008).

DSM-IV criteria for specific psychotic disorder, bipolar disorder I, and dementia, was assessed using the *Structured Clinical Interview for the Diagnostic and Statistical Manual – IV, Axis 1, patient version* (SCID-I; First, Spitzer, Gibbon, & Williams, 1995). The SCID-I is a semi-structured interview with inter-rater reliability values for Axis 1 modules ranging from .60 to .83 (Lobbestael, Leurgans, & Arntz, 2011). In this study, the SCID-I was used to not only assess exclusion criteria, but also to assess for comorbid diagnoses, as comorbidity tends to be the norm for individuals with BPD (Tomko, Trull, Wood, & Sher, 2014).

Cognitive functioning was assessed using the *Peabody Picture Vocabulary Test – Revised* (Dunn, 1981). The PPVT-R is a brief screening measure of verbal cognitive functioning, where a participant is asked to examine a group of pictures and identify the correct depiction of a stimulus named by the test administrator (Golden, Espe-Pfeifer, & Wachsler-Felder, 2000). The PPVT-R was initially normed on a nation-wide sample and has moderate internal consistency of .61 to .88 (Dunn & Dunn, 1981). It has a concurrent validity ranging from .40 to .60 (Dunn & Dunn, 1981; McCallum, 1985).

Outcome Measures. Assessors were masked to treatment condition assignment and calibrated with a gold-standard assessor on all semi-structured interviews.

The *Suicide Attempt Self-Injury Interview* (SASII; Linehan, Comtois, Brown, Heard, & Wagner, 2006) was used to measure changes in suicidal and non-suicidal self-injury throughout the course of the trial and will assess the frequency and severity of suicide and self-harm behaviors since the previous assessment (i.e., 3-month period). It takes approximately 30 minutes to administer. The SASII has been considered to be the most robust and comprehensive instrument available to measure self-harming behaviours in adults (Borschmann, Hogg, Phillips, & Moran, 2012) and has a high inter-rater reliability, ranging from .85 to .98, and high convergent validity, ranging from .75 to .99 (Linehan et al, 2006). The SASII was initially created as an offshoot of the L-SASII (Linehan & Comtois, 1996; Linehan, Comtois, Brown, Heard, & Wagner, 2006), described above. For the present study, the L-SASII frequency score was used to assess the inclusion criterion of self-injury behaviours prior to treatment, while the SASII frequency score for all timepoints (i.e., baseline, 3 months, 6 months, 9 months, and 12 months post-treatment) was used to measure frequency of self-injury behaviours over the previous 3-month interval.

The *Inventory of Interpersonal Problems – 64* (IIP-64; Horowitz, Alden, Wiggins, & Pincus, 1988, 2000) is a self-report measure that was used to assess interpersonal dysfunction. The IIP-64 is theoretically based on Wiggins's Interpersonal Problems Circumplex (Wiggins, 1979; Alden, Wiggins, & Pincus, 1990) and derived from the original Inventory of Interpersonal Problems (Horowitz, Rosenberg, Baer, Ureno, & Villasenor, 1988). The original IIP was a measure of problems with interpersonal functioning that was relevant to individuals with personality disorders (Scarpa et al., 1999). The IIP-64 consists of seven interpersonal problem subscales: Domineering/Controlling, Vindictive/Self-Centered, Cold/Distant, Non-assertive, Overly Accommodating, Self-Sacrificing, and Intrusive/Needy, and is divided into two sections:

behaviours that individuals find "hard to do" and behaviours that individuals "do too much." These behaviours are rated on a 5-point Likert scale ranging from 0 (not at all) to 4 (extremely). The psychometric properties of the IIP-64 have been demonstrated to be moderate to good. The Cronbach's alpha for subscales of the IIP-64 range from .72 to .88 (Vittengel, Clark, & Jarrett, 2003), and test-retest reliabilities for the subscales of the IIP-64 range from .58 to .84 (Horowitz, Alden, Wiggins, & Pincus, 2000). Correlations between the IIP-64 subscales with the Beck Anxiety Inventory and the Beck Depression Inventory range from .31 to .48, and correlations between IIP-64 subscales and the Brief Symptom Inventory range from .57 to .78 (Akyunus & Gencoz, 2016). The IIP-64 total score was used to measure interpersonal dysfunction.

The *Difficulties in Emotion Regulation Scale* (DERS; Gratz & Roemer, 2004) is a self-report measure that was used to assess general emotion dysregulation using six emotion-dysregulation subscales: Non-Acceptance of Emotional Responses, Difficulties Engaging in Goal-Directed Behaviour, Impulse Control Difficulties, Lack of Emotional Awareness, Limited Access to Emotion Regulation Strategies, and Lack of Emotional Clarity. The DERS has shown to have good test-retest reliability for the overall DERS score, with a kappa of .88, and good internal consistency for the total DERS scale and its subscales (.93 and a range of .80 to .89 respectively) (Gratz & Roemer, 2004; Neumann et al., 2010). The DERS has been shown to have adequate construct and predictive validity (Gratz & Roemer, 2004). There are strong relationships between scores on the DERS and maladaptive emotion regulation strategies as well, such as relational aggression, substance use, and NSSI (Fox et al., 2007; Gratz, Paulson, Jakupcak, & Tull, 2009; Gratz & Roemer, 2008). Finally, Gratz and Roemer (2004) reported that the DERS has good construct validity, demonstrating that DERS scores were significantly

correlated with emotion regulation measures and measures of experiential avoidance and emotional expressivity. The total DERS score was used to measure emotion dysregulation.

Analytic Strategy

Hypothesis 1. The first hypothesis that there will be an overall reduction in frequency of NSSI, interpersonal problems, and emotion dysregulation over the course of DBT (baseline, 3-months, 6-months, 9-months, and 12-months), was tested using latent growth curve modelling using Mplus (LGM; Cheong, MacKinnon, & Khoo, 2003; Selig and Preacher, 2009). Multilevel growth curve modelling is a type of structural equation modeling used to study change in variables over time, for nested data. In the current study, time points were nested within individuals, as each participant was assessed five times over the course of study. Growth curve models with a 2-level hierarchical structure [time points (level 1) nested within participant (level 2)] were evaluated. Using the data measured at all five timepoints, the latent growth curve model calculated three parameters of interest for each variable: the estimated score at baseline (the intercept), the rate at which the variable changes over time (the slope mean), and the interindividual variability in that rate (the slope variance; Selig and Preacher, 2009). That is, to measure the trajectory of emotion dysregulation change, the LGM calculated the three parameters of interest using the total DERS score at each of the five timepoints. Models were specified to have random intercepts and slopes (i.e., the intercept and slope of the variables will be permitted to vary among participants) and were estimated using a robust maximum likelihood estimation (MLR). Separate latent growth curve models were tested to determine the function that best represents the change in each variable over the five timepoints. This analytic method allowed for the study of the individual differences in the patterns of change for each of the variables of interest.

Hypothesis 2. The second hypothesis was that a reduction in interpersonal dysfunction will have an effect on the frequency of NSSI, mediated by a reduction in emotion dysregulation in DBT. This hypothesis was tested through a parallel process mediation (Bollen & Curran, 2004; Cheong et al., 2003; MacCallum, Kim, Malarkey, & Kiecolt-Glazer, 1997; Singer & Willett, 2003). This analytic method, which combines both traditional mediation models (e.g., Baron & Kenney, 1986) and latent growth modeling (e.g., Selig & Preacher, 2009), allows researchers to plot the trajectory of change in two or more variables simultaneously (i.e., in parallel), thus examining whether the intercept (i.e., the variable as measured at baseline) and slope mean (i.e., the average rate at which the variable changes) in one variable is related to the intercept and slope in another.

For the purposes of the present study, a *slope-only* parallel process mediation model was estimated, such that only the slopes of DERS, IIP, and frequency of NSSI will be included in the model. This model assessed whether changes in total IIP scores (slope 1) will lead to changes in total DERS scores (slope 2), and in turn, whether changes in DERS scores (slope 2) will lead to changes in frequency of NSSI (slope 3) to find the indirect effects pathway. Finally, the model tested whether changes in total IIP scores (slope 1) will lead to changes in NSSI (slope 3) to find the direct effects pathway. The parallel process mediation model addressed whether the relationship between changes in IIP and changes in NSSI is mediated by changes in DERS. This analytic method was chosen for its ability to preserve the parallel change processes (i.e., the trajectory of variable change over time), as the research hypotheses at hand sought to explore the mediating effect of *changes* in emotion dysregulation on the relationship between changes in interpersonal dysfunction and changes in NSSI throughout the course of treatment.

To estimate the direct and indirect effects, bias corrected standard errors and bias-corrected bootstrapped confidence intervals were estimated using maximum likelihood estimation using 500 bootstrap draws (Deng et al., 2013; Efron & Tibshirani, 1993; MacKinnon et al., 2004).

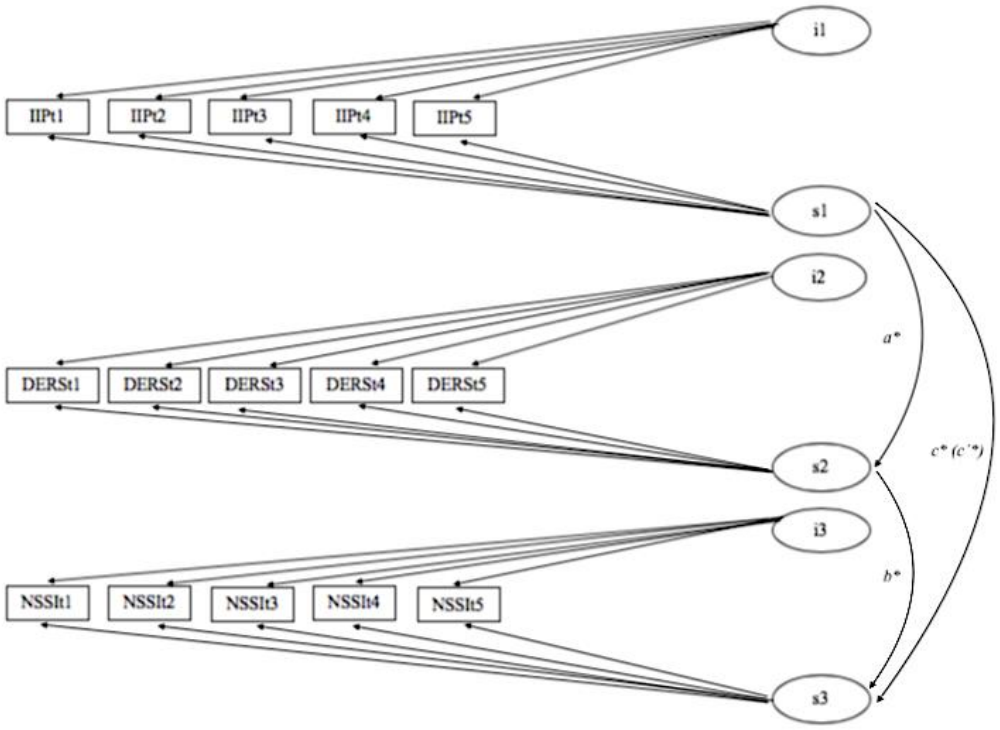

Figure 3. Proposed pathways (within a slope-only mediation model) between changes in emotion dysregulation, changes in interpersonal dysfunction, and changes in NSSI.

* The *a* path is the effect of changes in interpersonal dysfunction on changes in emotion dysregulation, the *b* path is the effect of changes in emotion dysregulation on changes in NSSI, the *c* path is the direct effect of changes in interpersonal dysfunction on changes in NSSI. The *c'* path in parentheses is the indirect effect of changes in interpersonal dysfunction and changes in NSSI through the indirect path of changes in emotion dysregulation.

Results

Descriptive statistics were calculated for all demographic and baseline measurements and can be found in Tables 1 and 2.

Table 1

Participant Demographics

Demographic	Summary Statistics
N	240
Age (mean)	*M* = 27.8 (*SD* = ±8.65)
Gender (%)	
Male	*N* = 38 (15.8%)
Female	*N* = 190 (79.2%)
Non-binary	*N* = 12 (5%)
Education (%)	
Less than High School	*N* = 22 (9.2%)
High School	*N* = 40 (16.7%)
Some College or University	*N* = 78 (32.5%)
>= College degree	*N* = 47 (19.6%)
Marital Status (%)	
Single	*N* = 181 (75.4%)
Widowed	*N* = 0 (0%)
Married/Common-Law	*N* = 38 (15.8%)
Separated	*N* = 12 (5%)
Divorced	*N* = 9 (3.8%)
Children (%)	

0	*N* = 209 (87.1%)
>=1	*N* = 31(12.9%)
Frequency of NSSI (median)	*Mdn* = 4.0 [2.00; 10.00]
DERS score (mean)	*M* = 129.7 (*SD* = ± 19.9)
IIP score (mean)	*M* = 121.3 (*SD* = ± 31.1)

Note. Variables represented as mean (± standard deviations), median [IQR], or frequencies (percentages). NSSI = non-suicidal self-injury; DERS = Difficulties with Emotion Regulation scale; IIP = Inventory of Interpersonal Problems.

Table 2

Lifetime and Current Diagnostic Comorbidities as Totals and Percentages

Diagnosis	Full Sample (Lifetime)	Full Sample (Current)
Major depressive disorder	*N* = 192 (80%)	*N* = 95 (39.6%)
Panic disorder	*N* = 83 (34.6%)	*N* = 71 (29.6%)
Posttraumatic stress disorder	*N* = 118 (49.2%)	*N* = 81 (33.8%)
Any anxiety disorders	*N* = 202 (84.2%)	*N* = 190 (79.2%)
Any eating disorders	*N* = 113 (47.1%)	*N* = 50 (20.8%)
Alcohol abuse	*N* = 41 (17.1%)	*N* = 11 (4.6%)
Alcohol dependence	*N* = 108 (45%)	*N* = 35 (14.6%)
Substance abuse	*N* = 49 (20.4%)	*N* = 14 (5.8%)
Substance dependence	*N* = 129 (53.8%)	*N* = 57 (23.8%)

Missing Data

Out of 120 participants, 14 participants were missing data from four time-points, five from three time-points, six from two time-points, and seven from 1 time-point, with a total of 32

participants with some missing data. This missing data was treated using a full maximum likelihood estimation, as estimated through Mplus 8.0 (Muthen & Muthen, 2017). The full maximum likelihood estimation (FIML) method for missing data has been found to be superior to traditional methods of dealing with missing data, such as pairwise deletion, listwise deletion, and mean imputation (Enders, 2001; Enders & Bandalos, 2001). Additionally, FIML has been compared to multiple imputation, another approach for dealing with missing data also lauded for its superiority over traditional methods; however, research has found that the multiple imputation approach may underestimate the standard error and increase the bias of the estimate (Larsen, 2011).

Hypothesis 1a: Changes in Emotion Dysregulation Over DBT

A linear growth curve model with maximum likelihood estimation with robust (Huber-White) standard errors and a scaled test statistic estimator (MLR) was run. The MLR estimator is a robust, nonparametric estimator that provides a Chi-square statistic that is robust to potential issues such as non-normality and non-independence of observations. Model fit statistics indicated that the model was a poor fit for the data, as evidenced by the CFI (0.944), TLI (0.944), RMSEA (0.109), and SRMR (0.164). Given there was residual variance left in the indicators, a quadratic growth curve model with maximum likelihood estimation was run to determine better model fit. Model fit statistics indicated that the model was an acceptable fit for the data, as evidenced by the CFI (0.999), TLI (0.998), RMSEA (0.021), and SRMR (0.062).

Results from the unconditional quadratic growth curve model of DERS scores revealed an overall decrease in DERS over the course of DBT, $\beta = -11.564$, $SE = 1.559$, $p < 0.01$, with decelerating decreases over assessment ($\beta = 1.005$, $SE = 0.372$, $p < 0.01$). Results also revealed that the variances of the intercept (i.e., the variance of DERS over individuals at baseline, $\beta =$

376.844, *SE* = 78.796, $p < 0.01$), and linear slope (i.e., the variance of growth rate over individuals, β = 144.574, *SE* = 57.86, $p < 0.05$) were significant. This indicates that there was a significant amount of variability (i.e., individual differences) between participants in both baseline levels of DERS and in the rates of change (i.e., trajectory) of DERS over the course of treatment. Additionally, findings revealed that there was no significant relationship between the latent intercept and latent slope of the DERS (β = -46.85, *SE* =71.90, *p* =0.608), indicating that there was no relationship between scoring on the DERS at baseline and subsequent rate of change in DERS.

Hypothesis 1b: Changes in Interpersonal Dysfunction Over DBT

Similar to the DERS data, a linear growth curve model with MLR estimation was run for the IIP data. Model fit statistics indicated that the model was an acceptable fit for the data, as evidenced by the CFI (0.999), TLI (0.999), RMSEA (0.035), and SRMR (0.043).

Results from the unconditional growth curve model of IIP scores revealed a mean slope for the IIP that was negative and statistically significant (β = -5.48, *SE* = 0.97, $p < 0.01$). That is, on average, participants' scores on the IIP significantly decreased over time. Results also revealed that the variance of the intercept was significant (β = 789.474, *SE* = 137.78, $p < 0.01$), as was the variance of the slope (β = 64.21, *SE* = 18.08, $p < 0.01$), indicating that there was a significant amount of variability between participants in both baseline levels of IIP and in the rates of change (i.e., trajectory) of IIP over the course of treatment. Notably, findings indicated a *significant* relationship for IIP scores between the latent intercept and latent slope (β = -84.08, *SE* =32.95, *p* =0.01), indicating that, if a participant scored higher on the IIP at baseline, the participant experienced a reduced rate of change in IIP.

Hypothesis 1c: Changes in NSSI Over DBT

For the present study, NSSI data was treated as a count variable. Count variables typically do not satisfy the assumption of normality; rather, their distributions are more aptly represented by Poisson, zero-inflated Poisson, negative binomial, or zero-inflated negative binomial distributions. Although count variables are not as normally distributed as continuous variables, linear growth curve modeling is able to model count variables two ways: 1) log transformation of the count variable, followed by a nonparametric estimator for a linear growth curve model, or 2) a linear growth model for a count outcome using a Poisson, zero-inflated Poisson, zero-inflated negative binomial, or negative binomial model.

Due to observations of over-dispersion in the distributions (i.e., the variance of a variable is larger than its mean), and a small count mean within the NSSI data, log-transformation is not recommended (O'Hara & Kotze, 2010; St-Pierre, Shikon, & Schneider, 2018). Therefore, all four linear growth curve models for count outcomes were run and the goodness of fit of each model was assessed. The distribution with the lowest loglikelihood, BIC, and AIC values indicates the best fit of the distribution to the observations.

Table 3

Goodness-of-fit values for NSSI count distribution

Distribution	Loglikelihood (df)	BIC	AIC
Poisson	-1282.864 (5)	2589.665	2575.728
Zero-Inflated Poisson	-1354.288 (14)	2775.600	2763.575
Negative Binomial	-1033.512 (10)	2114.898	2087.023
Zero-Inflated Negative Binomial	-1030.631 (15)	2133.075	2091.263

As indicated in Table 3, the negative binomial distribution provided the lowest BIC and AIC values, and the loglikelihood value was only slightly higher than that of the zero-inflated negative binomial distribution. Therefore, a linear growth curve model for a count outcome using a negative binomial model with an MLR estimator was used.

Results from the linear unconditional growth curve model revealed a mean slope for NSSI that was negative and statistically significant (β = -0.836, *SE* = 0.09, $p < 0.01$). That is, on average, participants' average NSSI incidences significantly decreased over time. Results also revealed that the variance of the intercept (i.e., the variance of NSSI incidences over individuals at baseline) was significant (β =1.112, *SE* = 0.24, $p < 0.01$), as was the variance of the slope (i.e., the variance of growth rate of NSSI incidences over individuals) (β = 0.18, *SE* = 0.078, $p <$ 0.05), meaning that there was a significant amount of variability between participants in their baseline number of NSSI incidences and in the rates of change (i.e., trajectory) of their NSSI incidences over the course of treatment. Additionally, findings revealed that there was no significant relationship between the latent intercept and latent slope of the NSSI incidence (β = -0.08, *SE* =0.09, p =0.36), indicating that there was no relationship between number of NSSI incidences at baseline and subsequent rate of change in NSSI incidences.

Hypothesis 2: Testing Changes in Emotion Dysregulation as a Mediator

To test this hypothesis, a longitudinal slope-only mediation model was estimated, as per von Soest and Hagtvet (2011). The slope of NSSI was regressed on the slopes of IIP and DERS and the slope of IIP was regressed on the slope of DERS. The intercepts for each growth model were allowed to covary with the slopes of each growth model. To estimate the direct and indirect effects, bias correct standard errors and bias-corrected bootstrapped confidence intervals were

estimated using the maximum likelihood estimation with 500 bootstrap draws (MacKinnon et al., 2004).

Overall, the model fit the data well [LL = -5574.29 (43), Pearson Chi-square for count variables = 1161.001 (99953), p = 1.00; Likelihood ratio chi-square = 229.326 (99953) p = 1.00]. Consistent with hypothesis, the slope of IIP significantly predicted the slope of DERS (i.e., the alpha path) ($\beta = 0.701$, $p < 0.01$). However, inconsistent with hypothesis, the slope of the IIP did not significantly predict the slope of NSSI ($\beta = 0.002$, $p = 1.00$) (i.e., the direct effect). In addition, the slope of the DERS did not significantly predict the slope of NSSI ($\beta = 0.032$, $p = 0.99$) (i.e., the beta path). Finally, the indirect effect of IIP on NSSI through DERS was not significant ($\beta = 0.022$, $p = 0.99$). This model did not provide support for mediation.

Discussion

NSSI is an immensely costly public health issue impacting a high percentage of individuals diagnosed with BPD. This study was designed to further enhance scientific and clinical understanding of the influence of changes in emotion dysregulation as a potential key feature of BPD accounting for changes in NSSI. Firstly, this study tested whether emotion dysregulation, interpersonal dysfunction, and NSSI decrease over the course of DBT. This study was the first to examine the change trajectory of these particular features of BPD over the course of treatment. Secondly, this study tested whether changes in emotion dysregulation was a mediator of the relationship between changes in interpersonal dysfunction and changes in NSSI. Results revealed that all three BPD features decreased over the course of DBT. Furthermore, although changes in emotion dysregulation was not found to be a mediator of the relationship between changes in interpersonal dysfunction and changes in NSSI, results demonstrated that

changes in interpersonal dysfunction predicted changes in emotion dysregulation over the course of DBT.

Changes in Emotion Dysregulation, Interpersonal Dysfunction, and NSSI Over DBT

Consistent with proposed hypotheses, results indicated that emotion dysregulation decreases over the course of DBT. Moreover, the results further suggest that such reductions in emotion dysregulation follow a non-linear trajectory in which these overall decreases in emotion dysregulation decelerate over time. Prior research by Lutz and colleagues (2012) on the patterns of change over the course of general psychotherapy have revealed that approximately 40% of improvements between two successive treatment sessions occur during the early phase of treatment. Findings from the present study are also consistent with a number of other studies, including a growth mixture modeling analysis conducted by Rubel and colleagues (2015), which revealed that the most change in progress occurred early in treatment for individuals receiving outpatient cognitive-behavioural therapy, after which participants experienced change in progress more moderately. Similarly, Macdonald and colleagues (2011), who studied the trajectory of change for individuals undergoing cognitive processing therapy (i.e., CPT) for PTSD, found that participants showed non-linear changes, where PTSD symptoms over CPT first decline more rapidly in earlier phases of treatment, followed then by a slower decline.

However, although prior studies specifically studying emotion dysregulation change over the course of treatment have found that emotion dysregulation decreases over the course of treatment, they specify a *linear* trajectory, rather than a quadratic trajectory. For example, a study examining the trajectory of emotion dysregulation over the course of specialized inpatient psychiatric intervention for adults with severe mental illness found that emotion dysregulation trajectory over the course of treatment was best fit to a linear model (Fowler et al., 2016). These

findings are inconsistent with the results of the present study; however, no research yet has explored the trajectory of emotion dysregulation change over the course of DBT.

Therefore, while the present findings of an overall decrease in emotion dysregulation over the course of DBT that decelerates in the latter timepoints is consistent with extant data about trajectory of *general* treatment outcomes, these findings are inconsistent with research specifically about emotion dysregulation outcomes. Further, there is a dearth of research regarding emotion dysregulation outcomes within the context of DBT. Thus, future research should replicate these analyses and further investigate the trajectory of emotion dysregulation over the course of DBT.

As well, consistent with Hypothesis 1b and 1c, analyses revealed that both interpersonal dysfunction and NSSI incidences significantly decrease over the course of DBT. These results regarding overall decrease in interpersonal dysfunction and NSSI incidences are consistent with the extant literature, which evinces that DBT and derivatives of DBT (i.e., brief 6-month DBT, DBT skills-only training) are efficacious both in reducing NSSI incidences and urges (Cook & Gorraiz, 2016; Kliem, Kroger, & Kosfelder, 2010; Krantz, McMain, & Kuo, 2018; McMain et al., 2012; Pistorello et al., 2012; Stanley, Brodsky, Nelson & Dulit, 2007), and interpersonal dysfunction (Pistorello et al., 2012; Wilks, Korslund, Harned, & Linehan, 2016).

Specifically regarding the finding that interpersonal dysfunction and NSSI incidences decrease *linearly* over DBT, an extant study on the trajectory of interpersonal dysfunction over the course of DBT reported on findings that a *quadratic* growth curve model indicated better fit for interpersonal dysfunction change in the context of DBT (Wilks, Korslund, Harned, & Linehan, 2016). However, this interpersonal dysfunction change is modelled over the course of treatment *and follow-up*, such that participants showed more rapid improvement of interpersonal

dysfunction that plateaued upon the end of treatment. Apart from this singular study on the trajectory of interpersonal dysfunction change in the context of DBT, a large majority of studies examining trajectory of change within DBT do not include NSSI or interpersonal dysfunction as an outcome of interest (McMain et al., 2017). Therefore, results in the literature to date are not comprehensive and should be examined in future studies.

Interestingly, higher interpersonal dysfunction at baseline was associated with less change in interpersonal dysfunction over DBT, suggesting that, for individuals with more severe interpersonal dysfunction, engagement in DBT does not lead to equivalent decreases in interpersonal dysfunction as it does for those who experience more mild or moderate interpersonal dysfunction. This finding is consistent with Ryle and Golynkina (2000)'s study, which found that higher pre-treatment BPD severity was associated with lower chance of recovery from BPD. However, it is inconsistent with a majority of the BPD literature at present, which suggests that pre-treatment BPD feature severity within specific clusters (i.e., impulsivity, feelings of emptiness, unstable relationships at baseline) may be a *positive* predictor of symptom change in DBT. That is, a majority of extant data suggests that individuals with greater BPD severity may have greater potential to achieve changes in psychotherapy (Barnicot et al., 2012; Yen, Johnson, Costello, & Simpson, 2009).

These findings related to interpersonal dysfunction may, in part, be related to the dearth of research specifically examining interpersonal dysfunction as an outcome in the present literature. Yen and colleagues' study examining pre-treatment BPD features as predictors of outcomes, interpersonal dysfunction was not examined as an outcome (Yen et al., 2009). Rather, their findings indicated that individuals who endorsed higher instability in relationships reported greater improvement in *self-injury*; and those who endorsed higher emptiness improved on

general *psychopathology, dissociation, and depression* (Yen et al., 2009). This finding was similarly reported in a systematic review published by Barnicot and colleagues (2012), which explored general pre-treatment BPD feature severity as a predictor of outcomes such as later BPD severity, depression severity, anger severity, dissociation severity, self-harm, suicidality, and general psychiatric severity, but did not examine interpersonal dysfunction itself as an outcome variable. Additionally, the present finding is novel in that it examines pre-treatment *interpersonal dysfunction* as a predictor of *change in interpersonal dysfunction* specifically.

Indeed, this finding that levels of pre-treatment interpersonal dysfunction differentially influence rates of subsequent change in interpersonal dysfunction over the course of DBT is not only theoretically but also clinically relevant. These findings indicate that more severe interpersonal dysfunction at pre-treatment may be associated to factors preventing greater treatment response, such as ambivalence or resistance to change. Additionally, an individual's severity of interpersonal dysfunction may be related to therapeutic alliance (as it has been found in long-term psychodynamic psychotherapy; Ollila, Knekt, Heinonen, & Lindfors, 2016) or treatment-interfering behaviours that disrupt the therapeutic process in DBT.

Testing Changes Emotion Dysregulation as a Mediator

When testing all pathways of the mediation model, the only significant pathway found was that changes in interpersonal dysfunction predicted changes in emotion dysregulation. Taken together with the fact that changes in emotion dysregulation did not mediate the relationship between changes in interpersonal dysfunction and changes in NSSI, this finding is inconsistent with predominant theories holding that emotional dysregulation is a primary mechanism in BPD pathology and treatment. These theories include the Biosocial theory (Linehan, 1993), Emotional Cascade model (Selby & Joiner, 2009), and Experiential Avoidance Model (Chapman, Gratz, &

Brown, 2006). The current findings are consistent with other theories of BPD that place emphasis on the role of interpersonal dysfunction within BPD and implicate interpersonal dysfunction as a critical contributor to emotion dysregulation within BPD. For instance, Gunderson and Lyons-Ruth posit that interpersonal sensitivity drives emotional reactivity in BPD in the Gene-Environment-Developmental Model (Gunderson & Lyons-Ruth, 2008). More specifically, this proposes that interpersonal hypersensitivity is the core feature leading to other features of BPD and arises from a genetic predisposition toward increased interpersonal stress reactivity and high reward value of attachment-related cues, as well as caregiver effects on infant attachment (Gunderson & Lyons-Ruth, 2008; Steele & Siever, 2010).

Similarly, Hughes and colleagues proposed a theory of BPD that primarily focuses on interpersonal functioning as a contributor to emotion dysregulation (Hughes, Crowell, Uyeji, & Coan, 2011). Hughes and colleagues suggested examining BPD, BPD features, and emotion dysregulation through the lens of the Social Baseline Theory, which proposes that all individuals are "hardwired… to utilize social proximity as a baseline affect regulation strategy" (Coan, 2010, p. 213). Essentially, this theory proposes that interpersonal relationships and attachment to others (i.e., social proximity) have traditionally served an evolutionarily adaptive role in distributing the metabolic cost of survival, through the distribution of risk (i.e., distribution of risk of becoming prey), load sharing (i.e., having companions who share the burden of health- and safety-related tasks), and economy of action (i.e., the conservation of energy when the ratio of collective resources expended to acquired reaches an ideal ratio). Therefore, co-regulation of emotion (i.e., attachment and interpersonal relationships) allow individuals to conserve their metabolic resources and is essential to survival (Coan, 2010). The Social Baseline Theory further

suggests that emotion dysregulation is not simply an individual failure to modify or regulate emotions but also occurs in an interpersonal context (Hughes et al., 2011).

Finally, Herpertz and colleagues also proposed a theoretical model of Social Dysfunctioning from the perspective of cognitive neuroscience, where social dysfunctioning (i.e., interpersonal dysfunction) has a bidirectional relationship with three BPD features: poor social cognition, impulsivity/behavioural disinhibition, and importantly, affect dysregulation (Herpertz, Jeung, Mancke, & Bertsch, 2014). Therefore, this model provides theoretical support of interpersonal dysfunction as a contributor to emotion dysregulation (Herpertz et al., 2014).

These theories that posit that interpersonal dysfunction contributes to and predicts emotion dysregulation are further supported by empirical evidence. In Dixon-Gordon and colleagues' (2013) study, which examined the mediating role of interpersonal difficulties in the relationship between borderline personality features and physiological measures of emotional reactivity to an interpersonal stressor in a non-clinical sample, findings revealed that total interpersonal dysfunction, and more specifically, interpersonal ambivalence, were significant mediators (Dixon-Gordon et al., 2013). These findings demonstrate that interpersonal and social deficits or dysfunctions may drive emotional reactivity in BPD. In addition, a study on the differences between perceived parental protection (i.e., interpersonal dysfunction) and cortisol response (i.e., emotional response) for individuals with BPD and healthy controls, found that participants with BPD reported less perceived parental protection in their mother-daughter relationship at lab entry, which was not only associated with higher cortisol levels at lab entry, but also higher distress following an interpersonal conflict discussion between the dyad (Lyons-Ruth et al., 2011). Finally, results from a study examining differences in social interaction diary entries between individuals with BPD, individuals with other personality disorders, and healthy

controls revealed that while the three groups had similar amounts of daily social interaction, individuals with BPD reported having more negative emotional reactions *to social interactions* (Stepp et al., 2009). Therefore, the finding that changes in interpersonal dysfunction predicted changes in emotion dysregulation is consistent with some extant theory and data.

However, none of the other pathways of the parallel process mediation model were shown to be significant. That is, findings demonstrated that changes in interpersonal dysfunction did not predict changes in NSSI, nor did emotion dysregulation, and therefore, contrary to our second hypothesis, we did not find that changes in emotion dysregulation was a significant mediator of the relationship between changes in interpersonal dysfunction and changes in NSSI frequency. A potential reason for these nonsignificant findings in the relationship between changes in interpersonal dysfunction and emotion dysregulation, and changes in NSSI frequency, is that there may be other features of BPD that may be more closely related to NSSI. For example, NSSI is associated with impulsivity, conceptualized as a heterogeneous construct that is organized into five factors: negative urgency (i.e., tendency to act rashly when experiencing negative affect), lack of premeditation (i.e., difficulty reflecting on consequences of an act prior to engaging in the act), lack of perseverance (i.e., difficulty staying focused on a boring or difficult task), sensation seeking (i.e., tendency to seek out new and exciting or dangerous experiences), and positive urgency (i.e., tendency to act rashly when experiencing positive affect; Whiteside & Lynam, 2001). Past research has demonstrated a relationship between these five factors of impulsivity and NSSI (Glenn & Klonsky, 2010; Hamza, Willoughby, & Heffer, 2015; Mullins-Sweatt, Lengel, & Grant, 2013). Alternatively, rumination is a psychological feature that is also strongly associated with NSSI (Selby et al., 2013) and implicated in a theoretical model as being a predictor of NSSI (Emotional Cascade Model; Selby & Joiner, 2009). In fact, Zaki and

colleagues (2013) found that rumination predicted higher rates of NSSI urges and incidences among participants who had difficulty differentiating negative emotions (i.e., emotion dysregulation) (Zaki, Coifman, Rafaeli, Berenson, & Downey, 2013). That is, emotion dysregulation was found to be a moderator for the relationship between rumination and NSSI frequency (Zaki et al., 2013) instead of a *mediator* as was tested in the present study. It is possible that we failed to find a mediating effect between interpersonal dysfunction and NSSI because neither of the chosen predictor or mediator variables, changes in interpersonal dysfunction and changes in emotion dysregulation, have a particularly strong impact on changes in NSSI within a treatment context, or perhaps because changes in emotion dysregulation act as a moderator instead of a mediator for the relationship between BPD features and NSSI.

Limitations and next steps

Although the present study was the first to explore the relationship between the trajectories of emotion dysregulation, interpersonal dysfunction, and NSSI over the course of DBT, it has several limitations. First, one methodological limitation of the present study is that the sample size was small. The present study was a secondary analysis of a randomized clinical trial and involved 6-12 months of treatment and multiple rounds of assessments; therefore, only 120 participants were enrolled in the present study. Given the complexity of the analyses performed on the data, and that parallel process mediation models in the literature have a sample size in the range of 223 (Piehler et al., 2014) to 1339 participants (Cheong, MacKinnon, & Khoo, 2003), the analyses within the present study should certainly be replicated with a larger sample size.

Next, the study uses a parallel process statistical method, which examines the trajectories of the predictor, mediator, and outcome variable over the same timepoints, and therefore is

unable to demonstrate time course to investigate which one variable remits first (i.e., the predictor). Since this statistical design cannot address the temporal precedence assumption of mediation, the present study is limited in the extent to which it can establish causality (von Soest and Hagtvet, 2011). Alternatively, another mediation model that would theoretically allow for investigation into causality is a latent difference score mediation model, which would have allowed for us to represent change in predictor, mediator, and outcome variable as separate epochs of change taking place during adjacent timepoints (Hamagami & McArdle, 2001; McArdle & Hamagami, 2001; Selig & Preacher, 2009; Turnes & Ernst, 2016). However, given the dearth of literature for latent difference score mediation with a count outcome variable, we were unable to utilize this model. As future quantitative knowledge progresses so too should future statistical mediation analyses involving count variables such as NSSI.

A theoretical consideration of the present study is that NSSI, the outcome variable within the present study, was a count variable. That is, NSSI over the course of DBT was conceptualized within the present study as the number of NSSI incidences over the three months prior to a timepoint assessment. While this is a typically-used indicator of NSSI, the present study did not capture factors such as severity of injury or differences in injury method, which, if studied, may have given a more comprehensive picture of NSSI for participants rather than a simple count. Furthermore, summarizing a multifaceted variable into a count variable also captures a low base rate; although some participants reported many incidences of NSSI at baseline, the median and IQR of NSSI incidences at baseline are low (Table 1). Finally, although the present study sought to determine the factors (i.e., emotion dysregulation and interpersonal dysfunction) that led to a reduction in NSSI incidences, participant responses from exit interviews about the mechanisms they perceived to be helpful in DBT or in their recovery that

led to decreases in NSSI, were not included. Future studies that aim to examine NSSI should take these limitations into account for a more comprehensive and qualitative look at NSSI incidences over the course of DBT.

Of note for future studies, previously reviewed theory (Chapman, Gratz, & Brown, 2006; Linehan, 1993; Selby & Joiner, 2009) and empirical evidence (Herr, Rosenthal, Geiger, & Erikson, 2013) implicate emotion dysregulation as a predictor of interpersonal dysfunction. Seeing that the present study evinced findings that changes in interpersonal dysfunction predicted changes in emotion dysregulation, the reverse should also be tested: whether changes in emotion dysregulation in turn predicts changes in interpersonal dysfunction. An investigation into a potential bidirectional relationship between changes in emotion dysregulation and changes in interpersonal dysfunction within BPD may be fruitful.

Future studies should also explore moderators of 1) the individual change trajectories of emotion dysregulation, interpersonal dysfunction, and NSSI, and 2) the relationships between these variables, to investigate whether there are subgroups of clients, for whom treatment impacts differentially, and if so, what factors impact this response pattern. Current literature implicates dissociation (Kleindienst et al., 2011) and DBT skills use (Neacsiu, Rizvi, & Linehan, 2010) as factors that may impact treatment response to DBT specifically, whereas low distress tolerance is a feature found in BPD that have been shown to moderate the relationship between affect intensity (i.e., emotion dysregulation) and NSSI frequency (Slabbert, Hasking, & Boyes, 2018). Therefore, distress tolerance may in fact moderate the relationship between changes in emotion dysregulation and changes in NSSI frequency in treatment. Since the present study did not examine the relationship between distress tolerance and NSSI, emotion dysregulation, or interpersonal dysfunction, the present findings may not be as nuanced, and this may result in

having not captured a relationship between changes in emotion dysregulation and changes in NSSI frequency within the course of DBT. This is especially crucial to inform clinical understanding and potential treatment modifications, as recent findings have indicated that subgroups of shared treatment response patterns may have differential treatment outcomes (Cuijpers et al., 2005; Stulz et al., 2010). Once moderators are identified, DBT therapists may use a typical response pattern in emotion dysregulation as a benchmark with which their client can be compared and make adjustments that may assist their improvement.

Conclusion

The present study was the first to examine the trajectories of emotion dysregulation, interpersonal dysfunction, and NSSI over the course of 12 months of DBT. In this study, findings indicated that emotion dysregulation, interpersonal dysfunction, and NSSI incidences all decrease over the course of a standard comprehensive treatment course of DBT, though the trajectory of emotion dysregulation differs from the trajectory of interpersonal dysfunction and NSSI in that emotion dysregulation decreases more sharply at the beginning of DBT than in the later timepoints, whereas interpersonal dysfunction and NSSI decrease more linearly over the course of DBT.

Additionally, the present study investigated whether changes in emotion dysregulation over these 12 months mediated the relationship between changes in interpersonal dysfunction and changes in NSSI. While no such mediation was found, findings did reveal that changes in interpersonal dysfunction predicted changes in emotion dysregulation. This finding holds implications for the study of the relationship between interpersonal dysfunction and emotion dysregulation. Future research into potential bidirectional relationship between the two variables

over the course of DBT (i.e., whether changes in emotion dysregulation also predict changes in emotion dysregulation), are clearly warranted.

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