Emotion States and Changes Following Rumination in Nonsuicidal Self-Injury and Eating Disorder Behaviours

by

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EMOTION STATES AND CHANGES FOLLOWING RUMINATION IN NONSUICIDAL SELF-INJURY AND EATING DISORDER BEHAVIOURS

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Nonsuicidal self-injury (NSSI) and eating disorder behaviours (EDB) may share a similar emotion dysregulation mechanism. This study examined the relations between repeated rumination episodes and emotions in NSSI and EDB within the context of the Emotional Cascades Model (Selby, Anestis, & Joiner, 2008), which suggests that ruminating on negative events increases the intensity of negative emotion; negative emotion prompts continued rumination, which further increases the intensity of the negative emotion. Individuals with a history of NSSI and/or EDB reported higher levels of negative emotions and lower levels of positive emotions, relative to individuals without a history of these behaviours. Similarly, a history of NSSI was associated with greater initial increases in negative emotions, and a history of EDB was associated with greater initial decreases in positive emotions, following rumination. While these results support the presence of emotion dysregulation in NSSI and EDBs, it only partially supports the emotional cascades model.
Dedication

For the ones I love always, and who love me back so well.
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Emotion States and Changes Following Rumination in Nonsuicidal Self-Injury and Eating Disorder Behaviours

Eating disorder behaviours (EDBs) and nonsuicidal self-injury (NSSI) are important mental health issues among young adults. While NSSI and EDB may occur independently, they often co-exist (Sansone & Levitt, 2004). A review of extant research suggests that NSSI and EDBs may have a common proximal antecedent, namely, dysregulated emotion regulation (Selby, Anestis, & Joiner, 2008). However, the extent to which NSSI and EDBs share a similar affect regulation mechanism has not yet been examined. Of particular interest for this study is the role of rumination as a maladaptive affect regulation strategy in NSSI and EDBs.

Dysregulated Behaviours

Nonsuicidal Self-Injury. Nonsuicidal self-injury (NSSI) refers to direct and deliberate damage to one’s own body tissues without intending to die (Nock & Favazza, 2009). Among other behaviours, this includes cutting, burning or embedding objects in the skin, and, in more severe cases, breaking bones (Gratz, 2001). This definition does not include self-injurious behaviour among individuals with developmental disabilities. Behaviours indirectly resulting in harm (e.g., smoking and disordered eating behaviours), that are accidental, that are done with lethal intent, or that are culturally or religiously sanctioned (e.g., tattoos and body piercings) are also not considered NSSI (Nock & Favazza, 2009).

NSSI is particularly prevalent among adolescents and young adults, with the average age of onset in the early-to-mid teen years (Rodham & Hawton, 2009; Skegg, 2005; Whitlock, Eckenrode, & Silverman, 2006). Within a 12-month period, up to 13% of high school students report engaging in NSSI (Miller, Muehlenkamp, & Jacobson, 2009). However, the highest age risk for NSSI may extend from 18 to 25 years (Rodham & Hawton, 2009). A random sampling
approach found that 17% of college students had a history of NSSI, and of these about 70% had engaged in this behaviour more than once (Whitlock et al., 2006). A study of university students found over 14% of students had engaged in NSSI in the past year (Serras, Saules, Cranford, & Eisenberg, 2010).

Rates of NSSI are relatively equal between males and females in university students (e.g., Heath et al., 2008; Serras et al., 2010; Whitlock et al., 2006). Similarly, studies in clinical settings have found similar proportions of males and females presenting to hospitals following NSSI episodes (e.g., Hawton, Harriss, Simkin, Bale, & Bond, 2004; Rodham & Hawton, 2009). Although general sex differences in lifetime rates of NSSI may not be found, important sex differences do exist in young adults. For example, relative to young adult males, females are more likely to engage in multiple NSSI episodes, and may engage in more frequent NSSI. Similarly, the methods and locations of NSSI in young adults also differs between the sexes, as females are more likely to scratch, pinch, and cut the wrists and thighs, whereas males are more likely to punch themselves and injure the hands (Whitlock et al., 2006).

A large range of factors may elevate risk for NSSI. For instance, specific externalizing behaviours (e.g., hyperactivity and inattention, conduct problems, smoking, and the use of alcohol or drugs), cognitive factors (e.g., rumination and negative thinking), and relationship factors (e.g., negative feelings towards parents or peers) have been identified as risk factors for NSSI (Bjärehed & Lundh, 2008). A study of middle school students found lower quality relationships with parents among students who engage in NSSI compared to those who do not (Hilt, Nock, Lloyd-Richardson, & Prinstein, 2008). Exposure to abuse in relationships—particularly emotional abuse—may also be associated with an increased risk for NSSI (Croyle & Waltz, 2007; Glassman, Weierich, Hooley, Deliberto, & Nock, 2008), however, this link may be
mediated by other factors such as how the individual copes with the abuse (Cha & Nock, 2009; Glassman et al., 2008; Deliberto & Nock, 2008). Family history of alcohol or drug abuse, violence, and suicide ideation are also predictive of NSSI (Deliberto & Nock, 2008).

Individuals who engage in NSSI are more likely than those who do not engage in NSSI to have at least one diagnosed mental illness (Janis & Nock, 2009), and to have experienced outpatient or inpatient psychiatric treatment (Lloyd-Richardson, Perrine, Dierker, & Kelley, 2007). NSSI is associated with higher levels of depressive and anxious symptoms (Ross & Heath, 2002), as well as a history of suicide ideation and suicide attempts (Janis & Nock, 2009; Lloyd-Richardson et al., 2007). It is also common for NSSI to be present in individuals who engage in other dysregulated behaviours such as substance abuse, binge drinking, and disordered eating (e.g., Bjärehed & Lundh, 2008; Hilt et al., 2008; Anderson, Simmon, Martens, Ferrier, & Sheehy, 2006; Serras et al., 2010). Of particular interest to this study is the relationship between NSSI and eating disorder behaviours.

**Eating Disorder Behaviours.** Eating disorders are currently classified in the Diagnostic and Statistical Manual of Mental Disorders-IV-TR (DSM-IV-TR; American Psychiatric Association [APA], 2000) into three categories: anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified. Anorexia nervosa involves an intense fear of gaining weight, body distortions, the refusal to maintain a minimally normal body weight, and the absence of menstruation. Individuals with bulimia nervosa engage in a pattern of binge eating and compensatory measures to prevent weight gain at least twice a week for three months. A diagnosis of eating disorder not otherwise specified is given when an individual engages in eating disorder behaviours (EDBs), but does not meet the full criteria for either anorexia nervosa or bulimia nervosa (e.g., restricting food intake but continuing to menstruate and thus not
meeting the criteria for anorexia nervosa, or binge eating and purging food but not at the
frequency required for a diagnosis of bulimia nervosa), and these behaviours result in distress or
impairment in functioning.

An EDB is defined as an unhealthy consumption level of food and/or attempt to regulate
weight using different behaviours. These include: restricting food intake beyond what the body
requires to function, binge eating, and purging food through self-induced vomiting, laxatives,
diuretics, enemas, fasting, and excessive exercise (APA, 2000). While these behaviours may be
part of a clinically significant eating disorder (e.g., anorexia nervosa, bulimia nervosa, or eating
disorder not otherwise specified), an individual may engage in EDBs at a subclinical level (i.e.,
the individual has some eating disorder behaviours and attitudes, but the full criteria for an eating
disorder is not met).

Lifetime rates of anorexia nervosa and bulimia nervosa range from 3.3% to 3.8% and
from 2.3% to 4.6%, respectively (Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006). Rates of
subclinical EDBs are high, and most women may engage in one or more EDBs at some point in
their lives (Murray 2003). A literature review highlighted that up to 80% of girls under the age of
18 years had restricted their intake, 35% had engaged in binge eating, and 8% had made
themselves vomit to control their weight (Murray, 2003).

There are several age and sex differences associated with specific EDBs. Adolescents are
at the greatest risk for behaviours related to anorexia and bulimia nervosa (i.e., restricting food
intake, binge eating, and engaging in compensatory behaviours), whereas binge eating alone is
typically engaged in well into adulthood (Striegel-Moore & Bulik, 2007). Girls are four times
more likely than boys to have symptoms of anorexia and bulimia (Reijonen, Pratt, Patel, &
Greydanus, 2003), while rates of binge eating are equal between males and females (Striegel-
Moore & Bulik, 2007). Aside from risks related to age and gender, other correlates with EDBs include personality factors (e.g., perfectionism, impulsivity, dependency), low self-esteem and body-dissatisfaction, and negative affect (Murray, 2003; Stice, Ng, & Shaw, 2010; Striegel-Moore & Bulik, 2007). Mood disorders, anxiety disorders, substance abuse disorders, and personality disorders frequently co-exist with eating disorders (Murray, 2003; Reijonen et al., 2003). The comorbidity of EDBs with NSSI appears to be especially high, as discussed below.

**Comorbidity of NSSI and EDB.** The presence of comorbid eating disorders is common in individuals who engage in NSSI; up to 61% of individuals with NSSI also report a current or past eating disorder, and 50% report a history of either anorexia or bulimia specifically (Sansone & Levitt, 2004). Similarly, NSSI is frequently observed in individuals with eating disorders. One literature review found that approximately 25% of both outpatient and inpatients with bulimia, and 22% of outpatients with anorexia, report a history of NSSI (Sansone & Levitt, 2004).

Overall, between 35% (Paul, Schroeter, Dahme, & Nutzinger, 2002) and 45% (Claes, Klonsky, Muehlenkamp, Kuppens, & Vandereycken, 2010) of all female eating disorder patients report a lifetime history of NSSI, while 21% report engaging in NSSI within the past six months (Paul et al., 2002). In terms of NSSI onset, 49% of the patients with an eating disorder reported that NSSI began after the eating disorder, 25% reported NSSI started before the eating disorder, and 26% reported that NSSI and the eating disorder began simultaneously (Paul et al., 2002). Among patients with eating disorders, the most common methods of self-injury include cutting and scratching oneself, followed by self-bruising and self-burning (Claes et al., 2010).

In clinical settings, NSSI appears to co-occur with binge eating and purging more frequently than with other EDBs (Peebles, Wilson, & Lock, 2011; Sansone & Levitt, 2004). Relative to other eating disorders, lifetime rates of NSSI are highest for individuals diagnosed
with eating disorder not otherwise specified, bulimia, or binge eating/purging subtype of anorexia (Paul et al., 2002; Sansone & Levitt, 2004). In a study of patients with bulimia, patients who self-injured reported more binge eating and vomiting than patients with no history of NSSI; however, there was no difference in general eating pathology between those with bulimia who had self-injured versus those with bulimia who had not self-injured (Muehlenkamp et al., 2009).

Individuals who engage in NSSI consistently show higher levels of EDBs on standardized measures compared to individuals who do not engage in NSSI (e.g., Bjärehed & Lundh, 2008; Croyle & Waltz, 2007; Hilt et al., 2008; Ross, Health, & Toste, 2009). This trend is particularly salient for females compared to males (Bjärehed & Lundh, 2008), and is consistent among both adolescents (Bjärehed & Lundh, 2008; Hilt et al., 2008; Ross et al., 2009) and adults (Croyle & Waltz, 2007). The high co-occurrence rate of NSSI with EDBs has led researchers to suggest that these behaviours may share similar antecedents and mechanisms. The current study focuses on a shared affect dysregulation antecedent and affect regulation mechanism.

**Emotional Cascades**

The emotional cascades model (Selby et al., 2008) postulates that focusing attention on negative emotional stimuli through cognitive processes (e.g., rumination, thought suppression, catastrophizing) increases the intensity of the negative emotion. A cyclical pattern develops where the increasingly intense negative emotion prompts the individual to focus further cognitive attention on the negative emotional experience, which then increases the intensity of the emotion. Dysregulated behaviours such as NSSI and EDBs are used as means to escape this cycle after the emotion has escalated to an unbearable level (Selby, Anestis, Bender, & Joiner, 2009; Selby et al., 2008; Selby & Joiner, 2009).
The emotional cascades model of dysregulated behaviour is consistent with and complementary to other models of NSSI. For example, the experiential avoidance model of NSSI (Chapman, Gratz, & Brown, 2006) explains NSSI as an attempt to avoid, reduce, and escape unwanted negative emotions. According to the experiential avoidance model, individuals who engage in NSSI experience high emotion intensity, poor distress tolerance, difficulties regulating emotions when aroused, and deficits in emotion regulation skills. According to research, NSSI is negatively reinforced through self-reported temporary relief from unwanted emotions (e.g., Claes et al., 2010; Klonsky, 2009). Thus, in the presence of unbearable emotions, NSSI may represent a temporarily effective, albeit maladaptive, problem-solving behaviour that becomes a conditioned response (Chapman et al., 2006; Miller & Smith, 2008). However, a limitation of the experiential avoidance model is that it does not formally account for the process by which the emotion becomes so unbearable that a method of escape as extreme as NSSI is needed (Selby et al., 2008).

A unique advantage of the emotional cascades model as a model of dysregulated behaviour is that it explains the nature of the interaction between cognitive and emotional variables on dysregulated behaviour (Selby et al., 2008). Furthermore, this model is not limited to explaining NSSI, but rather focuses on different types of dysregulated behaviour sharing a common function. To date, support for this model has been found in samples of participants with borderline personality disorder, which is known to include a variety of dysregulated behaviours such as NSSI and EDBs (Selby et al., 2009), as well as in specific dysregulated behaviours such as drinking, binge eating, and excessive reassurance seeking (Selby et al., 2008). Although the emotional cascades model has not been tested with NSSI and EDBs other than binge eating,
available research on the emotion and cognitive dysregulation associated with each of these types of behaviours indicates that the model may be applicable to these behaviours as well.

**Emotion Dysregulation.** Both NSSI and EDBs are associated with negative affect and difficulties regulating emotions. For example, individuals with eating disorders consistently score higher on scales of negative affect compared to participants without eating disorders (e.g., Blackburn, Johnston, Blampied, Popp, & Kallen, 2006; Spoor, Bekker, Van Strien, & van Heck, 2007; Sim & Zeman, 2005; Sim & Zeman, 2006; Vansteelandt, Rijmen, Pieters, Probst, & Vanderlinden, 2007). Similarly, in response to stressful events, individuals with a history of NSSI report more intense and more unpleasant emotions compared to individuals with no history of NSSI (Chapman et al., 2006; Glenn, Blumenthal, Klonsky, & Hajcak, 2011; Najmi, Wegner, & Nock, 2007; Nock & Mendes, 2008; Nock, Wedig, Holmberg, & Hooley, 2008). When the emotion reaches an unbearable intensity (Nock & Mendes, 2008), NSSI or EDBs may be used as means to regulate affect (Chapman et al., 2006; Selby et al., 2008).

A considerable amount of research supports an affect regulation mechanism of NSSI and EDBs, indicating that many individuals engage in these behaviours to alter their emotions (Gratz, 2007; Klonsky, 2007; 2009; Lewis & Santor, 2010; Nock & Prinstein, 2004; Nock, Prinstein, & Serba, 2009; Overton, Selway, Strongman, & Houston, 2005). Moreover, negative affect moderates the relation between EDB risk factors and EDBs (Sim & Zeman, 2005; Sim & Zeman, 2006; Vansteelandt et al., 2007), and predicts increases in EDBs over time (Presnell, Stice, Seidel, & Madeley, 2009). Similarly, individuals who report engaging in NSSI to reduce negative affect have a higher frequency of NSSI and a greater intent of engaging in future NSSI episodes (Lewis & Santor, 2010).
Self-reports indicate that engaging in NSSI or EDBs may alter affect states, and presumably renders them more tolerable to the individual. For example, negative mood states in females taking part in a NSSI support group were reported to be highest immediately prior to engaging in NSSI and lowest immediately following an NSSI episode; the effect of NSSI on mood was temporary, with negative mood states reported to have increased by the following day (Kamphius, Ruyling, & Reijntjes, 2007). Furthermore, reporting a reduction of negative affect following NSSI is a significant predictor of lifetime frequency of NSSI (Klonsky, 2009) and intent to self-injure in the future (Lewis & Santor, 2010). Moreover, this affect regulation mechanism appears to be limited to purposeful injury (i.e., NSSI) rather than accidental injury, as one study found that ratings of subjective negative emotions decreased in participants with a history of NSSI after engaging in imagery of NSSI, but not after engaging in imagery of accidental injury (Welch, Linehan, Sylvers, Chittams, & Rizvi, 2008).

The degree to which individuals who engage in NSSI and those who engage in EDB share a similar affect regulation mechanism, and the specific factors associated with this affect regulation, is not yet known. Some inconsistencies surrounding the role of affect regulation among EDBs exist in the literature. Research among individuals who self-injure has found that the act of NSSI results in self-reported decreases in the level of emotion arousal and improvements in emotion valence (Klonsky, 2009). Similarly, one study examining female inpatients with eating disorders who engaged in at least one form of NSSI found that low-arousal positive emotions (e.g., relief) increased and high-arousal negative emotions (e.g., anxious, angry) decreased following engaging in NSSI (Claes et al., 2010). Interestingly, for individuals with eating disorders (Claes et al., 2010) and for those who engage in NSSI (Klonsky, 2009), changes in positive affect following NSSI episodes was related to NSSI topology, including
frequency and the number of functions endorsed for this behaviour. In contrast, a recent meta-
analyses of ecological momentary assessments of changes in emotion states before and after
EDBs (i.e., binge eating, purging behaviours) found that although increases in negative affect
were observed directly prior to episodes of binge eating or purging, only purging was associated
with a decrease in negative affect after the episode; negative affect continued to increase
following episodes of binge eating (Haedt-Matt & Keel, 2011).

Individuals who engage in NSSI or EDBs not only experience more negative affect, they
also use less effective strategies for regulating emotions (e.g., Aldao, Nolen-Hoeksema, &
Schweizer, 2010; Heath, Toste, Nedellec, & Charlebois, 2008; Lavender & Anderson, 2010;
Whiteside et al., 2007). While NSSI and EDBs may serve as an attempt to regulate intense
emotions, other regulation strategies—particularly cognitive strategies—may be used prior to
engaging in these behaviours. Unfortunately, some cognitive emotion regulation strategies, such
as rumination, may exacerbate rather than relieve emotional distress.

**Rumination.** The maladaptive emotion regulation strategy of particular interest for this
study is *rumination*. Rumination is the process of responding to distress by repeatedly directing
one’s attention towards the causes, consequences and feelings of distress (Nolen-Hoeksema,
Wisco, & Lyubormirsky, 2008). Active problem solving and attempts to change the
circumstances leading to the distress are generally absent during rumination. According to the
emotional cascades model, the process of ruminating increases, rather than decreases, distress
and negative emotions (Selby et al., 2008; Selby et al., 2009; Selby & Joiner, 2009).

Induced rumination is associated with increases in depression, anxiety, and distress in
general (Aldao et al., 2010; Etu & Gray, 2010; Joormann, Dkane, & Gotlib, 2006; Nolen-
Hoeksema, 1991; Nolen-Hoeksema et al., 2008). Furthermore, rumination may affect not only
subjective experiences of emotions, but also physiological reactions to stress. For example in a study of individuals with major depression, induced rumination both prolonged the dysphoric mood and increased cortisol levels in response to the stress generated through rumination (Kuehner, Huffzinger, & Liebsch, 2009). Since depression and anxiety are commonly comorbid with NSSI (Klonsky & Glenn, 2009; Ross & Heath, 2002) and EDBs (Murray, 2003; Reijonen et al., 2003) rumination may affect emotions associated with NSSI and EDBs in a manner similar to other psychological difficulties.

**Rumination and Emotion Dysregulation in NSSI and EDB.** Research supporting the emotional cascades model in terms of NSSI and EDB is predominantly correlational using self-report measures of both rumination and behaviours. Scores on measures of rumination differentiate between individuals with and without a history of NSSI (Borrill et al., 2009; Croyle & Waltz, 2007; Hoff & Muehlenkamp, 2009) and EDB (Rawal, Park, & Williams, 2010). Rumination is also a strong predictor of NSSI frequency (Armey & Crowther, 2008; Bjärehed & Lundh, 2008; Selby, Connell, & Joiner, 2010) and EDB severity (Aldeo et al., 2010; Harrell & Jackson, 2008), and may mediate the relation between depressive symptoms and engaging in NSSI and EDBs as ways to regulate emotions (Harrell & Jackson, 2008; Hilt, Cha, & Nolen-Hoeksema, 2008).

There has been very little research conducted on the effects of rumination on emotion dysregulation in NSSI and EDBs. In one study, researchers found that instructing participants to ruminate about an imagined negative body image event led to greater body image dissatisfaction and anxiety compared to when participants are instructed to distract themselves from the same event (Etu & Gray, 2010). However, the researchers did not investigate whether the heightened anxiety resulting from the rumination translated into actual EDB. A novel real-time ecological
assessment found that immediately following episodes of NSSI, adolescents reported engaging in
NSSI as an attempt to escape not only from negative emotional states (i.e., anxiety, sadness, and
anger), but also from unwanted thoughts (Nock et al., 2009). Thus, dysregulated behaviours
such as NSSI and EDBs may be means to escape from the process of rumination in addition to
the escalating emotions driven by the process of rumination.

**Current Study**

The purpose of this study was to test one component of the emotional cascades model
within NSSI and EDB populations. Of particular interest was the effect that repeated induced
rumination had on emotion type and intensity for people who engage in NSSI and/or EDB.
Participants were asked to self-report the type and intensity of the emotions they experienced at
the start of the experiment and following each of five rumination induction trials. Analyses
sought to determine the relation between NSSI and/or EDB history and emotion states and
changes throughout the rumination induction trials.

**Hypothesis I.** The first hypothesis was that participants with a history of NSSI or EDB
would report more negative and less positive emotional states both initially (i.e., at baseline) and
in the presence of negative cognitions (i.e., immediately after each rumination induction trial)
when compared to controls with no history of NSSI or EDB. This hypothesis supports past
research indicating that individuals with a history of either NSSI or EDB demonstrate more
negative affect (e.g., Blackburn et al., 2006; Chapman et al., 2006; Glenn et al., 2011; Najmi et
al., 2007; Nock & Mendes, 2008; Nock et al., 2008; Spoor et al., 2007; Sim & Zeman, 2005; Sim
& Zeman, 2006; Vansteelandt et al., 2007) and less effective emotion regulation skills (e.g.,
Aldao et al., 2010; Heath et al., 2008; Lavender & Anderson, 2010; Whiteside et al., 2007)
relative to those individuals without a history of either of these behaviours.
**Hypothesis II.** A second hypothesis was that participants with a history of NSSI or EDB would report greater increases in negative emotions and decreases in positive emotions following each rumination induction trial when compared to controls with no history of NSSI or EDB. This hypothesis supports the emotional cascade model’s assumption that focusing on negative emotional stimuli increases the magnitude of the emotion, particularly for individuals with a history of NSSI or EDB (Selby et al., 2008). Furthermore, this result was expected based on previous research demonstrating that rumination magnifies the experience of negative emotions (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010; Etu & Gray, 2010; Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008). If NSSI and EDB are used as methods of escape from intense unwanted emotions and cognitions (Nock, 2009; Selby et al., 2008), then it is expected that the individuals who use these behaviours would demonstrate the greatest emotional responses to rumination. As NSSI and EDBs are believed to share a similar emotion regulation mechanism (Selby et al., 2008, Selby et al., 2009, Selby & Joiner, 2009), individuals who engage in both of these behaviours to regulate affect were expected to demonstrate greater emotional responses to rumination.

**Methods**

**Participants**

This study was approved by the university Research Ethics Board. Participation was voluntary; participants either received 1.5 percentage points towards their final introductory psychology grade, or were entered into a draw for an iPod Nano.

A total of 437 university students (78.9% females) between 17 and 36 years of age ($M = 18.61, SE = .08$) participated. Most indicated a Caucasian ethnicity (84.7%), however, participants with Asian (6.9%), African (2.1%), Hispanic (1.1%), East Indian (1.1%), mixed
(2.3%), and other (1%) ethnicities were also present. Participants indicated their sexual attraction as follows: straight (81.9%), mostly straight (12.4%), bisexual (3.7%), mostly gay/lesbian (0.7%), gay/lesbian (0.5%), and other (0.7%). Four participants did not indicate ethnicity, and one participant did not indicate sexual attraction.

A history of at least one episode of NSSI was reported by 230 (52.6%) participants, including 51.0% (n = 176) of female participants and 58.7% (n = 54) of male participants. A more detailed description of the specific NSSI behaviours endorsed can be found in Table 1. Of those participants who self-injured, 34.9% (n = 80) had engaged in NSSI within the past 12 months, 62.0% (n = 142) had not engaged in NSSI within the past 12 months, and 3.1% (n = 7) left this question blank. Lifetime frequencies of NSSI among those with a history of NSSI were as follows: 42.8% (n = 98) had engaged in 1 to 4 episodes of NSSI, while 56.3% (n = 129) had engaged in 5 or more episodes of NSSI; two participants (0.9%) did not report NSSI frequency. There were no differences in NSSI history between males and females, $F(1, 435) = 1.26, p = .26$, partial $\eta^2 = .03$, or ethnic groups, $F(7, 425) = 1.00, p = .43$, partial $\eta^2 = .02$. A statistically significant difference was observed for sexual attraction, $F(5, 430) = 2.58, p = .03$, partial $\eta^2 = .03$, whereby participants who were heterosexual had lower NSSI rates than those participants who were mostly heterosexual, $q = .25, p = .04$.

A history of at least one episode of EDB was reported by 252 (57.7%) participants, including 62.0% (n = 214) of female participants and 41.3% (n = 38) of male participants. A more detailed description of the specific EDB behaviours endorsed can be found in Table 2. Of those participants with a history of one or more EDB, 70.6% (n = 178) had engaged in an EDB within the past 12 months, 23.4% (n = 59) had not engaged in an EDB within the past 12 months, and 6.0% (n = 15) left this question blank. Lifetime frequencies of EDB among those
with a history of EDB were as follows: 19% \((n = 48)\) had engaged in between 1 and 4 episodes of EDB, while 78.6% \((n = 198)\) had engaged in 5 or more episodes of EDB; six participants (2.4%) did not report EDB frequency. More females than males indicated a history of EDB, \(F(1, 435) = 13.10, p < .001, \) partial \(\eta^2 = .03\). Neither ethnicity, \(F(7, 425) = 1.68, p = .11, \) partial \(\eta^2 = .03\), nor sexual attraction, \(F(5, 430) = .16, p = .66, \) partial \(\eta^2 = .01, \) predicted a history of EDB.

A history of both NSSI and EDB was reported by 152 (34.8%) participants, 77 (17.6%) participants had a history of NSSI but not EDB, 100 (22.9%) participants had a history of EDB but not NSSI, and 108 (24.7%) participants had no history of either behaviour. The proportion of males and females in each of the above groups can be found in Table 3. Among participants with a history of both NSSI and EDB, 31.6% \((n = 48)\) had engaged in both NSSI and EDB within the last year, 5.3% \((n = 8)\) had engaged in only NSSI within the last year, 42.8% \((n = 65)\) had engaged in only EDB within the last year, and 13.8% \((n = 21)\) had last engaged in both NSSI and EDB more than a year ago; 10 participants (6.6%) left this question blank. Lifetime frequencies of NSSI and EDB among those with a history of both behaviours showed that 54.6% \((n = 83)\) of participants had engaged in both behaviours five or more times, 7.9% \((n = 12)\) had engaged in NSSI five or more times and EDB fewer than five times, 28.3% \((n = 43)\) had engaged in EDB five or more times and NSSI fewer than five times, and 5.9% \((n = 9)\) had engaged in both NSSI and EDB fewer than five times; 5 participants (3.3%) left this question blank.

**Measures and Tasks**

**Demographics.** Participants self-reported age, sex, ethnicity, and sexual attraction.

**Deliberate Self-Harm Inventory** (DSHI; Gratz, 2001). The DSHI was used as a measure of NSSI behaviours. The DSHI is a self-report questionnaire assessing the presence, frequency, recency, and severity of 16 common specific NSSI behaviours, with the option to
report other forms of NSSI behaviour not already assessed (Gratz, 2001). The DSHI has good
test-retest reliability over periods of 2 to 4 weeks ($r = .92, p < .001$), and good construct,
convergent, and discriminant validity (Gratz, 2001).

**Bulimia Test—Revised** (BULIT-R; Thelen, Farmer, Wonderlich, & Smith, 1991). The
BULIT-R was used as a measure of EDB. The BULIT-R is a 36-item self-report questionnaire
frequently used in research as a measure of eating pathology in both clinical and non-clinical
populations (Thelen et al., 1991). Of the 36 items, 28 items assess the core criteria for a
diagnosis of bulimia nervosa, while 8 additional items measure the frequency of using
compensatory measures (i.e., laxatives, diuretics, fasting, and exercising). Participants use a 5-
point Likert scale to rate the frequency with which he or she identifies with each item. This scale
was chosen over other empirically validated scales because of the scale’s focus on specific
behaviours related to bulimia nervosa (i.e., binge eating and several types of compensatory
measures) which are frequently associated with NSSI (Sansone & Levitt, 2004). The BULIT-R
has high internal consistency ($\alpha = .94$ in the current study), adequate test-retest reliability over a
period of 2 months ($r = .95, p < .001$), and good construct, convergent, and discriminant validity
(Thelen et al., 1991; Thelen, Mintz, & Vander Wal, 1996).

**Eating Disorder Behaviour Inventory** (EDBI). Currently there is no eating disorder
measure assessing the lifetime history (i.e., presence, frequency, recency, and severity) across
EDBs. The EDBI was developed by combining the wording of the BULIT-R with the structure
of the DSHI to assess lifetime EDB history. This allowed the authors to compare EDB with
NSSI behaviours in a consistent manner, while retaining some of the credibility from the well-
established BULIT-R. While the format of this measure limited the ability to test for reliability,
BULIT-R scores significantly predicted EDB history, $F(1, 412) = 244.11, p < .001, r^2 = .37,$
EDB frequency, $F(1, 405) = 299.73, p < .001, r^2 = .43$, and EDB recency, $F(1, 412) = 255.61, p < .001, r^2 = .39$, on the EDBI, which provides support for adequate convergent validity for the EDBI.

**Rumination Induction Task** (Emotional Event Disclosure Task; EED). The procedure for inducing rumination was modified from that of Selby et al. (2009), where rumination was induced in participants with borderline personality disorder. For the current study the following text appeared before the participant on the computer screen:

*Now write about something in your life currently or in the past that is upsetting to you. Try to focus all of your concentration on your feelings about that problem. Consider what these feelings mean and why you feel this way. Analyze the events surrounding this problem and try to understand how they contribute to your feelings. Please do this for the next three minutes. Use the text box provided.*

In the Selby et al. (2009) study, individuals with borderline personality disorder indicated higher levels of negative affect and lower levels of positive affect following rumination induction compared to baseline. Whereas Selby et al. (2009) instructed participants to *think* about an upsetting event, the current study instructs participants to *write* about the upsetting event. Writing about the event may allow for the authors to examine the content of the rumination at a future date, and may help to prevent effects from mind wandering during the task.

**Positive and Negative Affect Schedule** (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS is a self-report measure of positive and negative mood states. An abridged PANAS (Nock & Mendes, 2008) was used in this study and participants were asked to rate on a 5-point Likert scale the extent to which they are currently experiencing each of 10 different emotions.
The positive moods assessed were: happy, confident, satisfied, calm and at ease. The negative moods assessed were: frustrated, sad, confused, angry and irritable. Both positive and negative affect scales have good internal consistency (Cronbach’s $\alpha = .88$ and $\alpha = .84$, respectively, in the current study). The original PANAS (Watson et al., 1988) also demonstrates adequate construct, convergent, and discriminant validity (Crawford & Henry, 2004).

**Procedure**

All measures and tasks were provided to the participants online via a computer. Participants completed a demographic questionnaire and self-reported NSSI and EDB history (DSHI, BULIT-R, EDBI). A baseline index of mood was also obtained (PANAS). Rumination was then induced through a single task (EED) asking participants to reflect on a past negative event, following which another index of mood (PANAS) was taken. The rumination induction task and index of mood were alternated five times. As this study induced rumination, which is known to induce a negative mood, it was important that the participant’s mood returned to baseline prior to ending the study; participants were thus asked to watch a nature video following the fifth rumination induction task and measurement of mood. A nature video was chosen for this purpose because nature has been shown to restore attention, increase self-regulation, and reduce stress (see review in Kaplan & Berman, 2010). At the conclusion of the study, participants were debriefed and provided information and resources for NSSI and EDBs.

**Results**

**Participant Retention**

Although the EED was administered five times consecutively, only three EED trials were used in the analyses below. Both negative and positive emotions tended to plateau around the third EED trial for all participants, albeit at different levels of the emotions between groups.
Those participants with a history of NSSI or EDB typically exhibited a plateau at more negative and less positive levels of emotions relative to those participants without a history of NSSI or EDB. However, it is unclear to what extent this captures the magnitude of actual between-group differences or is clinically significant. While it is possible that the emotions for all participants actually plateau, it may also be the case that the emotions in the control group plateaued while the emotions in the NSSI and EDB groups reached the ceiling (for negative emotions) and floor (for positive emotions) of the modified PANAS measure used to assess emotions.

Participation in the EED trials decreased throughout the study. Writing samples were screened for some degree of thought about a negative event. Cases were excluded (see Table 4) if nothing was written, if the text was excessively short (i.e., less than 12 words), if a spoiled response or responder bias was indicated (e.g., the participant commented on what they thought the study was trying to assess), or if the participant wrote about a positive event. Of the original 437 participants, a total of 371 participants (84.5%) completed the first three EED tasks. There were no significant differences in sex, $F(1, 435) = .03, p = .85$, partial $\eta^2 = .00$, age, $F(1, 435) = .22, p = .64$, $r^2 = .001$, NSSI history, $F(1, 435) = 2.44, p = .12$, partial $\eta^2 = .01$, EDB history, $F(1, 435) = .07, p = .79$, partial $\eta^2 = .00$, baseline negative emotions, $F(1, 432) = .95, p = .33, r^2 = .002$, or baseline positive emotions, $F(1, 431) = .11, p = .75, r^2 = .00$, between participants who completed the first three EED trials versus those who were excluded.

Among participants who completed the first three EED trials, a few failed to complete the modified PANAS at all time points. Due to the impracticalities of estimating changes in emotions over a series of time points from a very limited number of observations, participants with missing PANAS data were excluded from these analyses. Thus, analyses were conducted using 342 participants; 20.7% of the original sample was excluded for missing data (i.e., they did
not complete at least one of the first three EED trials, or did not rate at least one positive or negative emotion on the PANAS at some point up to and including the PANAS administration following the third EED trial). There were no significant differences in sex, \( F(1, 435) = .08, p = .78 \), partial \( \eta^2 = .00 \), age, \( F(1, 435) = .01, p = .95, r^2 = .003 \), NSSI history, \( F(1, 435) = .17, p = .68 \), partial \( \eta^2 = .00 \), or EDB history, \( F(1, 435) = .17, p = .68 \), partial \( \eta^2 = .00 \), between participants whose data were and were not included in the analyses.

**Analyses**

Both hypotheses were examined using the same omnibus tests; two repeated measures analyses of variance (ANOVAs) were conducted to assess the influence of behaviour history on emotion valence (positive, negative) following repeated rumination trials. NSSI (history, no history) and EDB (history, no history) served as fixed factors, and four emotion measurements (at baseline, and after EED1, EED2, and EED3) were the repeated measures. Separate ANOVAs were conducted for positive (Figure 1) and negative (Figure 2) emotions. A Greenhouse-Geisser correction was used to account for sphericity. Means and standard errors are provided in Table 5. Between-group differences, where applicable, were further assessed using one-way ANOVAs.

**Behaviour History and Emotional States**

**NSSI.** There were significant between-group main effects for NSSI history for both positive, \( F(1, 338) = 15.11, p < .001 \), partial \( \eta^2 = .043 \), and negative, \( F(1, 338) = 12.12, p = .001 \), partial \( \eta^2 = .035 \), emotions. Post hoc analyses found that participants with a history of NSSI exhibited lower levels of positive emotions at baseline, \( F(1, 340) = 16.78, p < .001 \), partial \( \eta^2 = .047 \), and following all three EED trials (EED1: \( F(1, 340) = 23.61, p < .001 \), partial \( \eta^2 = .065 \); EED 2: \( F(1, 340) = 19.32, p < .001 \), partial \( \eta^2 = .054 \); EED3: \( F(1, 340) = 13.22, p < .001 \), partial \( \eta^2 = .037 \)) relative to participants without a history of NSSI. Similarly, participants with a history
of NSSI had higher levels of negative emotions at baseline, $F(1, 340) = 8.28, p = .004$, partial $\eta^2 = .024$, and following all three EED trials (EED1: $F(1, 340) = 22.52, p < .001$, partial $\eta^2 = .062$; EED 2: $F(1, 340) = 19.56, p < .001$, partial $\eta^2 = .054$; EED3: $F(1, 340) = 5.66, p = .02$, partial $\eta^2 = .016$) when compared to participants with no NSSI history.

**EDB.** There were significant between-group main effects for EDB history for both positive, $F(1, 338) = 13.60, p < .001$, partial $\eta^2 = .039$, and negative, $F(1, 338) = 8.22, p = .004$, partial $\eta^2 = .024$, emotions. Participants with a history of EDB had lower levels of positive emotions at baseline, $F(1, 340) = 10.99, p = .001$, partial $\eta^2 = .031$, and following each of the three EED trials (EED1: $F(1, 340) = 19.25, p < .001$, partial $\eta^2 = .054$; EED 2: $F(1, 340) = 20.03, p < .001$, partial $\eta^2 = .056$; EED3: $F(1, 340) = 16.59, p < .001$, partial $\eta^2 = .047$). Similarly, relative to participants without a history of EDB, those with a history of EDB also exhibited higher levels of negative emotions at baseline, $F(1, 340) = 6.47, p = .01$, partial $\eta^2 = .019$, and following each of the three EED trials (EED1: $F(1, 340) = 9.60, p = .002$, partial $\eta^2 = .027$; EED2: $F(1, 340) = 15.59, p < .001$, partial $\eta^2 = .044$; EED3: $F(1, 340) = 8.32, p = .004$, partial $\eta^2 = .024$).

**Interaction between NSSI and EDB.** No significant interactions were observed between NSSI and EDB for either positive, $F(1, 338) = .12, p = .73$, partial $\eta^2 = .000$, or negative, $F(1, 338) = .01, p = .91$, partial $\eta^2 = .000$, emotions.

**Behaviour History and Cumulative Emotion Changes**

A significant within-subjects main effect was observed for both positive, $F(3, 1014) = 111.02, p < .001$, partial $\eta^2 = .247$, and negative, $F(3, 1014) = 34.07, p < .001$, partial $\eta^2 = .092$, emotions across the four emotion measurements. However, this main effect should be interpreted with caution in light of significant interactions between emotion measurements and behaviour.
history, as described below. As the Emotional Cascades Model postulates emotions become increasingly negative following rumination (Selby et al., 2008), the post-hoc analyses focused on cumulative changes (e.g., between baseline and each EED trial) in emotions between history and no history groups, rather than exploring differences observed between all emotion measurements.

**NSSI.** There was a significant interaction between NSSI history and emotion measurement for negative emotions, $F(3, 1014) = 4.40, p = .008$, partial $\eta^2 = .013$, but not positive emotions, $F(3, 1014) = 1.41, p = .24$, partial $\eta^2 = .004$. Post hoc analyses found that participants with a history of NSSI had greater cumulative increases in negative emotions following the first, $F(1, 340) = 9.99, p = .002$, partial $\eta^2 = .029$, and second, $F(1, 340) = 5.30, p = .02$, partial $\eta^2 = .015$, EED trials, but not after the third EED trial, $F(1, 340) = .03, p = .87$, partial $\eta^2 = .000$, when compared to participants without a history of NSSI.

**EDB.** There was a significant interaction between EDB history and emotion measurement for positive emotions, $F(3, 1014) = 2.75, p = .05$, partial $\eta^2 = .008$, but not negative emotions, $F(3, 1014) = 1.30, p = .28$, partial $\eta^2 = .004$. Post hoc analyses found that relative to participants without a history of EDB, participants with a history of EDB had greater cumulative decreases in positive emotions following the first, $F(1, 340) = 6.63, p = .01$, partial $\eta^2 = .019$, and second, $F(1, 340) = 6.27, p = .01$, partial $\eta^2 = .018$, EED trials, but not after the third EED trial, $F(1, 340) = 3.60, p = .06$, partial $\eta^2 = .010$.

**Interaction between NSSI and EDB.** No significant interactions between EDB history, NSSI history, and emotion measurements were found for either negative, $F(3, 1014) = .36, p = .74$, partial $\eta^2 = .001$, or positive, $F(3, 1014) = 1.69, p = .18$, partial $\eta^2 = .005$, emotions.
Discussion

The results of this study highlight several trends between behaviour history and emotional states and changes following repeated rumination induction trials. These trends are discussed below in relation to the study’s hypotheses and in the context of current research in the field. Theoretical and clinical implications, as well as study limitations, are also highlighted.

Hypothesis 1: Emotional States

Participants with a history of NSSI or EDB were hypothesized to report more negative and fewer positive emotions following each rumination induction task when compared to controls with no history of NSSI or EDB. Indeed, this hypothesis was supported as a history of NSSI or EDB was associated with a higher level of negative emotions and a lower level of positive emotions at baseline and after each of the three EED trials. These results indicate that individuals with a history of NSSI or EDB may routinely experience (i.e., both in the presence and absence of stressors) greater levels of negative and lower levels of positive emotions relative to those without a history of these behaviours. These observed differences in emotions states between groups support past research indicating that those who engage in NSSI and EDB generally experience greater levels of negative emotions (e.g., Chapman et al., 2006; Najmi et al., 2007; Nock & Mendes, 2008; Nock et al., 2008; Presnell et al., 2009; Sim & Zeman, 2005; Sim & Zeman, 2006) and lower levels of positive emotions (Gratz, 2006), relative to individuals who do not engage in NSSI or EDB.

It is unclear whether this reported heightened negative emotion and reduced positive emotion is a physiological difference in arousal or whether those individuals who engage in NSSI or EDBs rate the same level of emotion more or less intensely compared to individuals who do not engage in these behaviours. Both NSSI (Borrill et al., 2009) and EDBs (Ridout,
Thom, & Wallis, 2010; Rozenstein, Latzer, Stein, & Eviatar, 2011) are associated with higher levels of alexithymia (i.e., difficulties in identifying, distinguishing, and communicating emotions); past research indicates that alexithymia is associated with impaired emotion recognition while physiological arousal remains intact (Stone & Neilson, 2001). Thus, physiological and subjective experiences of arousal are two distinct facets of emotion that may or may not be consistent with each other among individuals who engage in NSSI or EDBs. For example, adolescents who engage in NSSI have greater physiological responses during a distressing task and report poorer tolerance of this distress (Nock & Mendes, 2008). In contrast, individuals who engage in binge eating or purging behaviours also report greater difficulties with distress tolerance (Anestis, Selby, Fink, & Joiner, 2007), but do not exhibit greater physiological responses to stressful situations (Tuschen-Caffier & Vögele, 1999) relative to individuals who do not engage in these EDBs.

**Hypothesis II: Cumulative Emotion Changes**

Participants with a history of NSSI or EDB were also hypothesized to report greater changes in emotions relative to those participants without a history of NSSI or EDB, respectively. This hypothesis was partially supported. While a significant main effect was observed among all participants, relative to those participants without a history NSSI or EDB, respectively, participants with a history of NSSI demonstrated significant increases in negative emotions, and participants with a history of EDB demonstrated significant decreases in positive emotions. Furthermore, changes in emotions were observed following only the first two of the three EED trials relative to baseline. These results support the emotional cascades model as negative cognitive processes, such as rumination, are shown to lead to increasingly intense negative emotions (Selby, Anestis, Bender, & Joiner, 2009; Selby et al., 2008; Selby & Joiner,
2009), particularly among those with a history of NSSI or EDB relative to those without a history of these behaviours. This further supports research indicating that NSSI and EDBs are associated with emotional dysregulation (e.g., Aldao et al., 2010; Heath et al., 2008; Lavender & Anderson, 2010; Whiteside et al., 2007), especially in the presence of negative cognitive processes (Nock et al., 2009; Selby, Anestis et al., 2009; Selby et al., 2008; Selby & Joiner, 2009). The unique contribution of this study is the use of an experimental paradigm to link the increasingly negative emotions directly to negative cognitive processes, thus implicating a greater degree of emotion dysregulation among those individuals with a history of NSSI or EDB relative to those without a history of these behaviours.

Interestingly, this effect may apply only to initial exposure to negative cognitions. The cumulative change after the third EED trial was not significantly different between those with a history of NSSI or EDB, and those without. This indicates that while those participants with a history of these behaviours had greater changes in emotions earlier in the rumination induction trials compared to those participants without a history of NSSI or EDB, the overall amount of emotion change was roughly the same for both those participants with and without a history of these behaviours. While these results conflict with the emotional cascades model of dysregulated behaviours, in which emotions are expected to continue to become increasingly negative following rumination, they are consistent with research on emotion reactivity in NSSI and eating disorders. More specifically, individuals who engage in NSSI or EDBs self-report greater emotion reactivity compared to controls without a history of NSSI or EDB (Glenn et al., 2011; Nock et al., 2008). Thus, those individuals with a history of NSSI or EDB may have a faster emotional response to the same stimulus compared to those without a history of these behaviours. Those individuals without a history of these behaviours may require a greater degree
of negative cognitive processes before their emotions are affected to the extent seen among those with a history of NSSI or EDB. While this current study did not formally examine emotion reactivity, those participants with a history of NSSI or EDB did demonstrate significantly greater changes in emotions earlier in the trials relative to those participants without a history of these behaviours, indicating that they may more easily be triggered by negative cognitive processes.

Although those individuals who engage in both NSSI and EDB had the highest levels of negative emotions, the lowest levels of positive emotions, and the greatest initial changes in emotions (Table 5), the interactions between behaviours were not statistically significant. Indeed, the mechanism(s) behind the interaction between emotions and multiple dysregulated behaviours is (are) not yet known. It is possible that the degree and type of emotion dysregulation (e.g., reactivity, intensity, distress tolerance) and behaviour consequences (e.g., physical or social consequences) may not be comparable between behaviours. For example, past research indicates that changes in affect following NSSI in patients with eating disorders is greater for patients who engage in cutting, scratching or burning, relative to those who engage in bruising as a form of NSSI (Claes et al., 2010), indicating that not all NSSI behaviours are entirely comparable. Similarly, not all EDBs may be entirely comparable, as research has shown that changes in negative affect following EDBs is different for purging versus binge eating behaviours (Haedt-Matt & Keel, 2011). As NSSI involves direct and immediate damage to body tissues, whereas the physical consequences of EDBs accumulate slowly over time, NSSI may be considered a more extreme behaviour compared to EDB. Future research should examine the comparability of different methods of NSSI and EDBs as well as differentiating correlates (e.g., emotion dysregulation, physical consequences) between these methods.
Theoretical Implications

These results provide partial support for a shared affect regulation mechanism for NSSI and EDB (Selby et al., 2008, Selby et al., 2009, Selby & Joiner, 2009). Those individuals who engage in these behaviours had higher levels of negative emotions and lower levels of positive emotions relative to those individuals without a history of these behaviours; greater increases in negative emotions were also observed among individuals with a history of NSSI, as well as greater initial decreases in positive emotions among individuals with a history of EDB. Contrary to expectations, no significant interactions were observed between NSSI and EDBs for either emotion states or emotion changes following rumination.

Although NSSI and EDBs share some similarities in dysregulated affect, the extent to which they share a similar affect regulation mechanism is unclear, as different emotion valences are affected by rumination in NSSI versus EDB. There may be an element of emotion dysregulation present among those who engage in NSSI that is subtly different from those who engage in some forms of EDBs. To date, research formally examining differences in emotion regulation between NSSI and EDBs is scant. However, impulse control may be one personality component that interacts with affect regulation in the presence of emotional distress to lead to dysregulated behaviours such as NSSI and EDBs (Tice, Bratslavsky, & Baumeister, 2001); furthermore, impulse control characteristics may differ for NSSI and EDBs. For example, one study found that males with eating disorders who also engage in NSSI reported greater difficulties with impulse control relative to males with eating disorders who did not engage in NSSI (Claes et al., 2011). Indeed, while both NSSI (Claes et al., 2011; Glenn & Klonsky, 2010; Ross et al., 2009) and bulimia nervosa (Anestis et al., 2007; Heilbrun & Bloomfield, 2006) are
associated with self-reported deficits in impulse control, anorexia nervosa is not (Heilbrun & Bloomfield, 2006).

Furthermore, while affect regulation is a primary function for both NSSI and EDB (e.g., Gratz, 2007; Klonsky, 2007; 2009; Klonsky & Glenn, 2009; Sim & Zeman, 2005; 2006; Vansteelandt et al., 2007), other functions of NSSI are reported (see Klonsky, 2007; 2009 for overview). These other functions may include intrapersonal functions other than affect regulation (e.g., to punish the self, to feel something in the presence of numbness, or to quell suicidal thoughts and urges), as well as interpersonal functions (e.g., to communicate internal distress, to fit in with others, or to get revenge against others). Indeed, the number of functions for NSSI reported by individuals who engage in both NSSI and EDB is associated with changes in positive affect following NSSI (Claes et al., 2010). Very little research has examined the functions of EDBs, and no published research to date has formally compared the functions of EDBs and NSSI; however, preliminary research suggests that while there are similarities in functions endorsed in both behaviours, there are also clinically useful differences (Peyerl, Muehlenkamp, & Claes, 2011). While an affect regulation function may be the most widely endorsed function for engaging in NSSI among those who engage in NSSI only (Gratz, 2007; Klonsky, 2007; 2009; Klonsky & Glenn, 2009), and those who engage in both eating disorders and NSSI (Claes et al., 2010), some research implicates social functions as having a larger role to play in EDBs (Lieberman, Gauvin, Bukowski, & White, 2001). Individuals may also have multiple functions for NSSI and EDBs, and these may evolve over time. Thus, more research needs to be conducted to better understand the interaction between intra- and interpersonal functions in EDBs, and to understand how functions of EDBs and NSSI are similar and different.
Clinical Implications

Findings from this study have several clinical implications. First, NSSI and EDBs are associated with more negative emotional states, as well as greater increases in negative emotions and decreases in positive emotions (for NSSI and EDB, respectively), following negative cognitive processes. This means that individuals who experience emotion dysregulation may be at a greater risk for NSSI and EDBs as means to attempt to regulate affect. This is consistent with research showing that individuals engage in NSSI and EDBs in order to alter negative affect (Klonsly, 2009; Lewis & Santor, 2010; Nock & Prinstein, 2004; Nock, Prinstein, & Serba, 2009; Overton et al., 2005), and that this negative affect predicts EDBs (Presnell et al., 2009) and NSSI (Lewis & Santor, 2010) over time. Moreover, individuals with poor emotion regulation skills may be at an increased risk for eating disorders and more frequent and medically severe NSSI. Thus, in line with current assessment guidelines (e.g., Klonsky & Lewis, in press; Klonsky & Weinberg, 2009), emotional regulation skills should be assessed in individuals who present with NSSI or EDBs. In individuals with poor emotion regulation skills, NSSI and EDB topology (i.e., methods, frequency, medical severity) should also be assessed and monitored for changes. Furthermore, a functional assessment of NSSI or EDB may provide insight into the extent that these behaviours are used to regulate affect for the individual (Klonsky et al., 2011; Klonsky & Weinberg, 2009).

Second, treatments for both NSSI and eating disorders should include increasing emotion regulation skills and targeting maladaptive cognitions. Treatments involving cognitive behavioural techniques have received empirical support for NSSI (see Klonsky & Muehlenkamp, 2006; Muehlenkamp, 2006), eating disorders (see Wilson, Grilo, & Vitousek, 2007), and for targeting rumination among other psychopathologies such as depression (Watkins et al., 2007).
and obsessive-compulsive disorder (Freestone et al., 1997). Dialectical behaviour therapy (DBT; Linehan, 1993) in particular, which draws on cognitive behavioural techniques, has been found to be effective in reducing both NSSI (Linehan, 2006; Turner, 2000) and EDBs (Safer, Telch, & Agras, 2001; Telch, Agras, & Linehan, 2001) in clinical samples. A central goal of DBT is to reduce maladaptive behaviours by increasing emotion regulation skills. This is accomplished by enhancing mindfulness, distress tolerance, emotion regulation, and interpersonal effectiveness (Linehan, 1993)—all areas that are known to be deficient among individuals who engage in NSSI and EDB. Through DBT, an individual with poor emotion regulation skills will learn to accept and tolerate more intensely negative emotions without engaging in dysregulated behaviours such as NSSI and EDB. Thus, for individuals who engage in NSSI and EDB for affect regulation purposes, dialectical behaviour therapy may be a well-suited treatment approach.

Finally, as a history of both NSSI and EDB are, to some extent, associated with more negative emotions relative to participants with a history of only one of these behaviours, clients should be screened for multiple maladaptive behaviours, even if these behaviors are subclinical. This aligns with assessment recommendations for NSSI (e.g., Klonsky & Lewis, in press; Klonsky & Weinberg, 2009). Behaviours such as drinking and gambling are believed to share a similar affect regulation function with NSSI and EDB (Selby et al., 2008; Selby et al., 2009; Selby & Joiner, 2009; Serras et al., 2010); however, more research is needed to determine the extent to which other maladaptive behaviours share a similar affect regulation mechanism with NSSI, and the degree to which they co-occur. Clinicians are cautioned to avoid assuming that all maladaptive behaviours serve the same function for the individual, and are encouraged to conduct a functional assessment of each behaviour in order to best tailor treatment options.
towards the individual (for overviews of NSSI functional assessment see Klonsky, Muehlenkamp, Lewis, & Walsh, 2011; Walsh, 2006).

**Limitations**

Limitations to this study include examining only behaviour history. Interactions between behaviour frequency, recency, poly-method behaviour, and medical severity of NSSI and EDB may be more strongly associated with emotion dysregulation than behaviour history alone. For example, following NSSI, the reduction of negative affect predicts lifetime number of episodes of NSSI (Klonsky, 2009), and—among patients with eating disorders—the increase in positive affect is associated with NSSI frequency (Claes et al., 2010). Due to a limited number of participants, and small participant numbers in some groups, this study was unable to examine the interactions between these other behaviour domains.

The measurement of EDBs may represent an additional limitation in this study. In order to develop a parallel measure of the DSHI (used to assess NSSI) for EDBs, this study focused on six EDBs and then analyzed these EDBs together. However, past research has demonstrated different patterns in affect change before and after EDB episodes for different EDBs (e.g., Haedt-Matt & Keel, 2011). As past research indicates that NSSI is associated more strongly with binge eating and purging (Paul et al., 2002; Peebles et al., 2011; Sansone & Levitt, 2004), as opposed to other EDBs, it could be possible that the behaviours stereotypically central to bulimia (e.g., binge eating, self-induced vomiting) may have a greater association with emotional dysregulation than other compensatory measures examined in this study (e.g., over exercising, restricting caloric intake, using laxatives or diuretics). Future research should examine EDBs independent of each other.
Research shows that other maladaptive behaviours (e.g., drinking, smoking, excessive reassurance seeking) may also be associated with a similar affect regulation function and emotion dysregulation mechanism (Selby et al., 2008; Selby et al., 2009). Thus, while a participant may have a history of NSSI or EDB, they may also have a history of another behaviour that shares a similar affect regulation function but is not captured in this study. In addition, not all of these behaviours have the same level of physical harm or social consequences. Similarly, this study examined NSSI and EDB in university students, and results from a sample of university students may not be generalizable to other populations. To date, support for the emotional cascades model has been found in both clinical samples of individuals with borderline personality disorder (Selby et al., 2009), as well as nonclinical samples of undergraduate students (Selby et al., 2008). The extent to which the emotional cascades model is supported in nonclinical non-university samples is still unknown.

While this study is able to demonstrate that negative cognitive processes lead to more negative emotion states, no conclusions can be made as to the extent to which emotion dysregulation and rumination lead to actual NSSI and EDB behaviours. However, past research has found that rumination predicts NSSI (Borrill et al., 2009; Croyle & Waltz, 2007; Hoff & Muehlenkamp, 2009) and EDB (Rawal, Park, & Williams, 2010) history, NSSI frequency (Armey & Crowther, 2008; Bjärehed & Lundh, 2008; Selby, Connell, & Joiner, 2010), and EDB severity (Aldeo et al., 2010; Harrell & Jackson, 2008). Furthermore, greater decrease in negative emotions (Claes et al., 2010; Haedt-Matt & Keel, 2011; Klonsky, 2009; Welsh et al., 2008) and increases in low-arousal positive emotions (Claes et al., 2010) following engaging in NSSI or EDB have been observed in those who engage in NSSI and EDB relative to those who do not engage in these behaviours.
Similarly, as the content of the texts written during the rumination induction task were not examined in great detail, it is not know to what extent participants engaged in true rumination. While writing about a negative event may have elicited rumination among some participants, others may have inadvertently used other emotion regulation strategies to distract from or even resolve the negative event (Aldeo et al., 2010, Selby et al., 2008). Both catastrophizing and thought suppression also involve focusing attention on negative events and are believed to increase negative emotions in a manner consistent with rumination (Selby et al., 2008). Similarly, all types of rumination may not affect emotions equally; for example, brooding may be a more maladaptive form of rumination compared to reflective pondering (Joormann et al., 2006). While individuals who engage in NSSI may engage in more brooding (Selby et al., 2010) and reflective rumination (Hoff & Muehlenkamp, 2009; Selby et al., 2010) relative to those individuals without a history of NSSI, reflective rumination may be a stronger predictor of NSSI history (Hoff & Muehlenkamp, 2009) and frequency (Selby et al., 2010) than brooding, and may not always be adaptive in this population. In contrast to the type of rumination best predictive of NSSI, EDB may be more strongly linked to brooding rather than to reflective rumination (Rawal et al., 2010).

Rumination is typically associated with thoughts that unintentionally enter the consciousness (Joorman et al., 2006; Nolen-Hoeksema, 1987); however, this study asked participants to intentionally ruminate. It is not currently known whether intentional rumination affects emotions differently from unintentional rumination. Thus, while the intent of this task was to induce rumination, and all participants included in these analyses wrote about a negative event, the actual emotion regulation strategies or type of rumination indicated in these texts will need to be examined at a later date. Furthermore, future work should examine the valence and
intensity of emotions associated with different emotion regulation strategies, and rumination in particular.

Finally, because this study was administered over the Internet, and participants were able to take a break at any point during the study, conclusions involving timing of events must be made cautiously. This study does not predict emotion reactivity, or how quickly emotions change following a negative event. Similarly, it cannot account for factors such as events outside of the study that may have influenced participant emotions. Additionally, only the first three of the five EED trials were analyzed. Thus, no conclusions can be made about the relation between induced rumination and emotions beyond the initial three EED trials. Future research should address the effects of in-situ rumination and temporal length of rumination on emotions.

**Conclusion**

The emotional cascades model suggests that emotions become increasingly negative following negative cognitions. Although emotion states observed in this study were consistently more negative among individuals with a history of NSSI or EDB, relative to those without a history of these behaviours, emotions only initially became more negative following rumination. While these results clearly support the presence of emotion dysregulation in NSSI and EDBs, it only partially supports the emotional cascades model in particular. The presence of emotion dysregulation in both behaviours represents an important avenue for future work.
References


of an interpersonal model. *Journal of Early Adolescence*, 28, 455-469. doi:
10.1177/0272431608316604


10.1016/j.psychres.2008.06.041


10.1097/NMD.0b013e3181593d89


10.1016/j.psychres.2008.02.008


Table 1

Frequency of Participants (N = 437) Reporting a History of NSSI, by Behaviour

<table>
<thead>
<tr>
<th>NSSI Behaviour</th>
<th>N</th>
<th>Females&lt;sup&gt;a&lt;/sup&gt; %</th>
<th>Males&lt;sup&gt;b&lt;/sup&gt; %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cut your wrists, arms, or other areas of your body (without intending to kill yourself)?</td>
<td>110</td>
<td>28.1</td>
<td>14.1</td>
</tr>
<tr>
<td>Burned yourself with a cigarette?</td>
<td>11</td>
<td>2.0</td>
<td>4.3</td>
</tr>
<tr>
<td>Burned yourself with a lighter or a match?</td>
<td>22</td>
<td>4.4</td>
<td>7.9</td>
</tr>
<tr>
<td>Carved words into your skin?</td>
<td>28</td>
<td>7.6</td>
<td>2.2</td>
</tr>
<tr>
<td>Carved pictures, designs, or other marks into your skin?</td>
<td>27</td>
<td>7.3</td>
<td>2.2</td>
</tr>
<tr>
<td>Severely scratched yourself, to the extent that scarring or bleeding occurred?</td>
<td>88</td>
<td>22.4</td>
<td>12.6</td>
</tr>
<tr>
<td>Bit yourself, to the extent that you broke the skin?</td>
<td>17</td>
<td>3.5</td>
<td>5.4</td>
</tr>
<tr>
<td>Rubbed sandpaper on your body?</td>
<td>6</td>
<td>1.5</td>
<td>1.1</td>
</tr>
<tr>
<td>Dripped acid onto your skin?</td>
<td>2</td>
<td>0.0</td>
<td>2.2</td>
</tr>
<tr>
<td>Used bleach, comet, or oven cleaner to scrub your skin?</td>
<td>3</td>
<td>0.0</td>
<td>3.3</td>
</tr>
<tr>
<td>Stuck sharp objects such as needles, pins, staples, etc. into your skin, not including tattoos, ear piercing, needles used for drug use, or body piercing?</td>
<td>48</td>
<td>11.1</td>
<td>11.4</td>
</tr>
<tr>
<td>Rubbed glass into your skin?</td>
<td>2</td>
<td>0.6</td>
<td>0.0</td>
</tr>
<tr>
<td>Broken your own bones?</td>
<td>2</td>
<td>0.6</td>
<td>0.0</td>
</tr>
<tr>
<td>Banged your head against something, to the extent that you caused a bruise to appear?</td>
<td>33</td>
<td>5.2</td>
<td>16.3</td>
</tr>
<tr>
<td>Punched yourself, to the extent that you caused a bruise to appear?</td>
<td>36</td>
<td>7.9</td>
<td>9.8</td>
</tr>
<tr>
<td>Prevented wounds from healing?</td>
<td>44</td>
<td>10.5</td>
<td>8.7</td>
</tr>
<tr>
<td>Done anything else to hurt yourself that was not asked about in this questionnaire?</td>
<td>43</td>
<td>9.0</td>
<td>13.0</td>
</tr>
</tbody>
</table>

Note. Participants may report a history of more than one NSSI behaviour.

<sup>a</sup> n = 345

<sup>b</sup> n = 92
Table 2

*Frequency of Participants (N = 437) Reporting a History of EDB, by Behaviour*

<table>
<thead>
<tr>
<th>EDB behaviour</th>
<th>N</th>
<th>Females&lt;sup&gt;a&lt;/sup&gt; %</th>
<th>Males&lt;sup&gt;b&lt;/sup&gt; %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Engaged in episodes of uncontrolled eating, where you eat to the point of stuffing yourself?</td>
<td>147</td>
<td>37.7</td>
<td>18.5</td>
</tr>
<tr>
<td>Used laxatives or suppositories to help control your weight?</td>
<td>18</td>
<td>5.2</td>
<td>0.0</td>
</tr>
<tr>
<td>Used diuretics to help control your weight?</td>
<td>5</td>
<td>1.2</td>
<td>1.1</td>
</tr>
<tr>
<td>Exercised vigorously for long periods of time in order to burn calories?</td>
<td>150</td>
<td>38.3</td>
<td>19.6</td>
</tr>
<tr>
<td>Intentionally vomited after eating?</td>
<td>47</td>
<td>12.8</td>
<td>3.3</td>
</tr>
<tr>
<td>Fasted or gone on strict diets to lose weight?</td>
<td>120</td>
<td>31.0</td>
<td>14.1</td>
</tr>
</tbody>
</table>

*Note.* Participants may report a history of more than one EDB behaviour.

<sup>a</sup> n = 345

<sup>b</sup> n = 92
Table 3

*Frequencies of Participants Reporting NSSI and/or EDB Histories*

<table>
<thead>
<tr>
<th>Behaviour history group</th>
<th>N</th>
<th>Females(^a) %</th>
<th>Males(^b) %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Both NSSI and EDB</td>
<td>152</td>
<td>36.2</td>
<td>29.3</td>
</tr>
<tr>
<td>NSSI only</td>
<td>77</td>
<td>14.8</td>
<td>28.3</td>
</tr>
<tr>
<td>EDB only</td>
<td>100</td>
<td>25.8</td>
<td>12.0</td>
</tr>
<tr>
<td>Neither NSSI nor EDB</td>
<td>108</td>
<td>23.2</td>
<td>30.4</td>
</tr>
</tbody>
</table>

\(^a\) \(n = 354\)

\(^b\) \(n = 92\)
### Emotional Event Disclosure Task Completion Rates (N) by Behaviour History

<table>
<thead>
<tr>
<th>EED task</th>
<th>Total</th>
<th>EDB history</th>
<th>No EDB history</th>
<th>NSSI history</th>
<th>No NSSI history</th>
</tr>
</thead>
<tbody>
<tr>
<td>EED 1</td>
<td>401</td>
<td>234</td>
<td>167</td>
<td>217</td>
<td>184</td>
</tr>
<tr>
<td>EED 2</td>
<td>386</td>
<td>222</td>
<td>164</td>
<td>206</td>
<td>180</td>
</tr>
<tr>
<td>EED 3</td>
<td>371</td>
<td>214</td>
<td>157</td>
<td>201</td>
<td>170</td>
</tr>
<tr>
<td>EED 4</td>
<td>358</td>
<td>209</td>
<td>149</td>
<td>198</td>
<td>160</td>
</tr>
<tr>
<td>EED 5</td>
<td>323</td>
<td>192</td>
<td>131</td>
<td>181</td>
<td>142</td>
</tr>
</tbody>
</table>
Table 5

Positive and Negative Emotion Means (SE) for each Behaviour Group across EED Trials

<table>
<thead>
<tr>
<th>Behaviour history within each emotion valence</th>
<th>Baseline</th>
<th>EED1</th>
<th>EED2</th>
<th>EED3</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Positive Emotions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of NSSI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of EDB</td>
<td>14.27 (.42)</td>
<td>12.28 (.44)</td>
<td>11.19 (.45)</td>
<td>11.35 (.47)</td>
<td>121</td>
</tr>
<tr>
<td>No history of EDB</td>
<td>15.80 (.57)</td>
<td>14.02 (.66)</td>
<td>13.25 (.70)</td>
<td>12.88 (.69)</td>
<td>60</td>
</tr>
<tr>
<td>Total (history of NSSI)</td>
<td>14.78 (.34)</td>
<td>12.86 (.37)</td>
<td>11.87 (.39)</td>
<td>11.86 (.39)</td>
<td>181</td>
</tr>
<tr>
<td>No history of NSSI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of EDB</td>
<td>16.19 (.48)</td>
<td>14.33 (.57)</td>
<td>13.19 (.63)</td>
<td>12.64 (.64)</td>
<td>78</td>
</tr>
<tr>
<td>No history of EDB</td>
<td>17.25 (.46)</td>
<td>16.61 (.54)</td>
<td>15.53 (.56)</td>
<td>15.24 (.57)</td>
<td>83</td>
</tr>
<tr>
<td>Total (no history of NSSI)</td>
<td>16.64 (.33)</td>
<td>15.52 (.40)</td>
<td>14.40 (.43)</td>
<td>13.98 (.44)</td>
<td>161</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of EDB</td>
<td>15.03 (.32)</td>
<td>13.09 (.36)</td>
<td>11.97 (.37)</td>
<td>11.85 (.38)</td>
<td>199</td>
</tr>
<tr>
<td>No history of EDB</td>
<td>16.64 (.36)</td>
<td>15.52 (.43)</td>
<td>14.57 (.45)</td>
<td>14.25 (.45)</td>
<td>143</td>
</tr>
<tr>
<td>Total (all participants)</td>
<td>15.70 (.24)</td>
<td>14.11 (.28)</td>
<td>13.06 (.29)</td>
<td>12.86 (.30)</td>
<td>342</td>
</tr>
<tr>
<td><strong>Negative Emotions</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of NSSI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of EDB</td>
<td>10.00 (.38)</td>
<td>11.82 (.44)</td>
<td>12.45 (.42)</td>
<td>11.60 (.44)</td>
<td>121</td>
</tr>
<tr>
<td>No history of EDB</td>
<td>9.20 (.51)</td>
<td>10.73 (.55)</td>
<td>10.70 (.53)</td>
<td>10.58 (.53)</td>
<td>60</td>
</tr>
<tr>
<td>Total (history of NSSI)</td>
<td>9.73 (.31)</td>
<td>11.46 (.35)</td>
<td>11.87 (.34)</td>
<td>11.27 (.34)</td>
<td>181</td>
</tr>
<tr>
<td>No history of NSSI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of EDB</td>
<td>9.03 (.46)</td>
<td>9.82 (.46)</td>
<td>10.47 (.49)</td>
<td>10.86 (.50)</td>
<td>78</td>
</tr>
<tr>
<td>No history of EDB</td>
<td>8.04 (.35)</td>
<td>8.66 (.42)</td>
<td>9.04 (.47)</td>
<td>9.41 (.45)</td>
<td>83</td>
</tr>
<tr>
<td>Total (no history of NSSI)</td>
<td>8.52 (.29)</td>
<td>9.22 (.31)</td>
<td>9.73 (.34)</td>
<td>10.11 (.34)</td>
<td>161</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>History of EDB</td>
<td>9.62 (.30)</td>
<td>11.04 (.33)</td>
<td>11.67 (.33)</td>
<td>11.31 (.33)</td>
<td>199</td>
</tr>
<tr>
<td>No history of EDB</td>
<td>8.52 (.30)</td>
<td>9.53 (.35)</td>
<td>9.73 (.36)</td>
<td>9.90 (.35)</td>
<td>143</td>
</tr>
<tr>
<td>Total (all participants)</td>
<td>9.16 (.21)</td>
<td>10.41 (.24)</td>
<td>10.86 (.25)</td>
<td>10.72 (.24)</td>
<td>342</td>
</tr>
</tbody>
</table>
Figure 1. Positive emotion ratings obtained at baseline and following each of the first three EED tasks. There were no significant interactions for NSSI with EDB; thus, emotion states for NSSI history (a) and EDB history (b) are graphed separately.
Figure 2. Negative emotion ratings obtained at baseline and following each of the first three EED tasks. There were no significant interactions for NSSI with EDB; thus, emotion states for NSSI history (a) and EDB history (b) are graphed separately.