

Investigating Self-Criticism as a Transdiagnostic Predictor of  
Self-Harm Behaviors

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## Chapter I

### INTRODUCTION

Self-harm represents a growing public health concern, generating psychological and medical morbidity and mortality across the lifespan. Nock (2010) noted that research on direct self-harm (i.e., suicide and nonsuicidal self-injury) has typically occurred in isolation from inquiry on more indirect, yet still devastating behaviors (e.g., disordered eating, substance abuse, risky sexual behavior). Clinicians and researchers agree that direct and indirect forms of self-harm frequently co-occur (e.g., Esposito-Smythers & Spirito, 2011; Goldberg & Israelashvili, 2017; Kostro, Lerman, & Attia, 2014; Nock et al., 2013). Nock and others have called on researchers to integrate knowledge from both sets of literature to identify common drivers of these behaviors, thus facilitating prevention and intervention efforts.

#### **Nonsuicidal Self-Injury and Disordered Eating**

This dissertation focuses on nonsuicidal self-injury (NSSI) as an exemplar of direct self-injury and disordered eating (DE) as a form of indirect self-harm and examines self-criticism (SC) as a transdiagnostic risk factor for the same.<sup>1</sup> Both NSSI and DE are associated with substantial psychiatric and medical sequelae (e.g., Cipriano, Cella, & Cotrufo, 2017; Jacobson & Gould, 2007; Treasure, Claudino, & Zucker, 2010). Most alarmingly, research shows that both NSSI and DE may increase risk for suicidality (Franklin et al., 2017; Smith, Velkoff, Ribeiro, &

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<sup>1</sup> NSSI refers to deliberate damage of body tissue absent intent to die (Nock, 2009). Cutting and burning are prototypical forms of NSSI, although the term may encompass a multitude of behaviors. DE refers to the group of behaviors associated with DSM-5 eating disorder diagnoses, including food restriction, binge eating, and purging (e.g., via self-induced vomiting, laxative use, compulsive/excessive exercise, etc.; American Psychiatric Association, 2013).

Franklin, 2018). We have previously reviewed the empirical and theoretical literatures on comorbidity between NSSI and DE (Zelkowitz & Cole, 2018). Briefly, NSSI and DE show high comorbidity. Svirko and Hawton (2007) estimated the prevalence of eating disorders among patients exhibiting some form of NSSI as between 54 and 61 percent and the prevalence of NSSI among patients with eating disorders as between 25.4 and 55.2 percent. As noted in Zelkowitz and Cole (2008), a meta-analysis by Cucchi et al. (2016) estimated a 27.3 percent lifetime prevalence of NSSI among patients with eating disorders, with higher rates of NSSI among individuals diagnosed with bulimia nervosa (BN) versus anorexia nervosa (AN). Taliaferro and Muehlenkamp (2015) also showed that negative attitudes toward one's weight distinguished undergraduates who currently engage in NSSI from those with more distal histories of the behavior. Notably, the research on NSSI and DE has largely focused on DE diagnoses (which encompass attitudes and cognitions about weight and body) or such weight/body-related cognitions themselves. As such, we know less about the association of NSSI with DE behaviors specifically. Moreover, research to date has typically focused on lifetime prevalence of NSSI. This makes sense from a pragmatic standpoint, given relatively low base rates of NSSI, but leaves unclear to what extent NSSI and DE may be associated over shorter time periods.

Understanding drivers of these two behaviors will be critical in the development of effective treatments; indeed, understanding the “need for self-destructive behavior” among individuals with disordered eating has been identified as a “top 10” research priority in that field (van Furth, van der Meer, & Cowan, 2016, p. 706). As noted in Zelkowitz and Cole (2008), conceptual models of DE and NSSI offer potential points of departure for understanding psychopathological processes that may operate across both behaviors. Svirko and Hawton (2007) proposed self-criticism, affect dysregulation, and dissociation as psychological processes that

may contribute to multiple forms of DE in the context of other underlying risk factors (e.g., trauma, invalidating family environments, etc.). Claes and Muehlenkamp (2014) similarly highlight the roles of self-criticism and emotion dysregulation in relation to NSSI and DE, although they consider the role of other constructs as well (e.g., disregard of one's body). Their model also emphasizes the salience of life stress in combination with other risk factors to promote NSSI and DE.

This dissertation will investigate SC as a transdiagnostic risk process in NSSI and DE. My emphasis on SC stems, in part, from a new conceptual model of self-harm that proposes that NSSI may serve an emotion regulation function via satisfying desires for self-punishment (Hooley & Franklin, 2017). The model also proposes that positive self-regard serves as one of several key barriers preventing most individuals from engaging in NSSI. In this model, self-criticism thus serves to erode a key protective barrier to the behavior (and/or reflects the consequence of such erosion). The authors argue that diminished positive regard toward oneself (and specifically one's body) render individuals more apt to select NSSI over indirect self-harm methods such as emotion regulation, let alone more adaptive coping strategies. The authors acknowledge the association of SC with poor body image/body dissatisfaction, a construct with established relevance for DE individually and as a comorbid behavior to NSSI (e.g., Claes and Muehlenkamp, 2014; Muehlenkamp, Peat, Claes, & Smits, 2012; Stice, 2002). I thus contend that the positive self-regard aspect of the benefits and barriers model may also hold relevance to DE and the comorbidity of NSSI and DE. Finally, as described in Zelkowitz and Cole (2018), we note conceptual linkages between increasingly severe self-criticism/self-hatred and actual self-harm behaviors (Horney, 1950).

## Conceptualizing Self-Criticism

Before examining the relation of self-criticism to NSSI and DE, I will clarify what I mean by the term “self-criticism” and how it relates to constructs such as self-esteem, guilt, and shame. Self-criticism is a universal experience with a long history in the clinical literature. Clinical references to self-criticism begin in the early 20<sup>th</sup> century. Psychoanalysts attributed self-criticism to the conscience and saw it as a means of developing inhibition and self-censure (Stärcke, 1929). Beck (1963) focused on clinical self-criticism when he delineated faulty cognitive processes in depressed versus non-depressed patients. He identified self-criticism as a major theme in cognitions of depressed patients and further distinguished low self-regard from self-criticism, the former reflecting an individual’s low appraisals of themselves compared to others or their own internal standards. The latter consisted of “reproaches...leveled against themselves for their perceived shortcomings” (p. 327), and Beck observed how these cognitions generally triggered consistent negative emotions such as guilt. Correcting the distortions inherent in such self-criticisms (i.e., getting the patient to judge themselves by more objective standards) became a major focus of his cognitive therapy approach (Beck, 1979). Later in psychotherapy research, Driscoll (1989) deconstructed self-criticism (in his terms, self-condemnation) into six key components, beginning with perceiving and appraising some aspect of the self as inferior or unacceptable. This would lead to a condemnation response, which might be “a spoken comment, an unspoken thought or rumination, or merely a feeling” (p. 105).

In contrast to these earlier, relatively narrow definitions of self-criticism, Blatt (1974, 1976) used the term to encompass negative self-evaluations, discrepancies between a perceived ideal self, and feelings of guilt. His conception of self-criticism dominated the field of personality research and psychopathology for decades. He contended that all individuals

experience some level of self-criticism but those who were particularly susceptible would experience depression characterized by “intense feelings of guilt and worthlessness and the sense that one has failed to live up to expectations and standards” (pp. 383-384). Shahar, Henrich, Blatt, Ryan and Little (2003) later clarified that imbalance between focus on self-definition (i.e., competencies, sense of control, self-esteem) at the expense of interpersonal relationships during development might foster self-criticism. Bagby and Rector (1998) argued that Blatt’s conception of self-criticism was redundant of the personality construct of neuroticism. Subsequent factor analyses and tests of incremental validity supported a distinction between the two constructs (Clara, Cox, Enns, Murray, & Torgrudc, 2003; Thompson & Zuroff, 2004). Other work demonstrated that individuals high in trait self-criticism actually expressed stronger negative emotions (disgust, hatred) toward themselves when prompted to criticize themselves in a laboratory setting (Whelton & Greenberg, 2005).

Gilbert (2000) examined self-criticism from an evolutionary perspective. He argued that because our brains evolved to support social relations, self-criticism reflects an internalized social exchange in which one “hostile dominant” inner voice attacks a “fearful submissive” aspect of the self. He observed, “In severe cases it is as if there is an inner war going on with ‘enemies’ and hated parts of the self” (p. 284). The pain of self-criticism results from the inability to defend oneself against these attacks (Gilbert, 2002; Greenberg, Elliott, & Foerster, 1990). Exploration of Gilbert’s conception of self-criticism (and associated measure; Gilbert, Clarke, Hempel, Miles, & Irons, 2004) revealed two key components: thoughts about one’s inadequacy and thoughts of hatred directed at the self (Gilbert et al., 2004; Kupeli, Chilcot, Schmidt, Campbell, & Troop, 2013).

From this brief overview of conceptions of self-criticism, several themes emerge. First, self-criticism is primarily a cognitive phenomenon. It consists of verbalized and nonverbalized statements directed toward and about the self. These statements reflect general opprobrium and highlight the individual's sense of failure to perform according to a set standard. Second, although primarily a cognitive construct, self-criticism also reflects negative emotions. Several theorists (i.e., Beck, 1963; Gilbert, 2000) have described self-criticism as an attack on oneself in which painful affects such as shame and guilt act as shrapnel. Other scholars have noted how elevated levels of shame and guilt may promote additional self-criticism (Blatt, 1974, 1976). Self-criticism can thus be conceptualized as distinct from but tightly linked to these emotions (cf. Gilbert, 2002). A third key component of self-criticism is that it generally consists of discrete thoughts (e.g., "I suck at this, I really messed that up, I am so dumb") which may or may not align with an individual's more global thoughts, feelings, and attitudes about the self. This component closely reflects what Rosenberg, Schooler, Schoenbach, and Rosenberg (1995) termed "global self-esteem." In the studies comprising this dissertation, I endeavored to select measures that explicitly focus on self-criticism rather than the constructs with which it is so closely linked (e.g., guilt, shame, self-esteem, etc.)<sup>2</sup>

### **SC in Relation to NSSI and DE**

Our previous meta-analysis identified significant associations of SC with both NSSI and DE (Zelkowitz & Cole, 2018). These associations were relatively consistent across age groups, sample type (i.e., clinical versus community), and assessment methods. Within the DE literature, we found evidence for stronger association of SC among individuals with BN compared to AN,

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<sup>2</sup> Although see Porter, Gist, Cole, & Zelkowitz, under review, for an investigation of convergent and discriminant validity of SC and self-esteem.

mirroring higher prevalence rates of NSSI in this diagnostic group. Our past work was limited in two key aspects in that 1) we only examined cross-sectional associations of SC and NSSI and SC and DE, and 2) we examined the relation of SC to each behavior independently without consideration for their comorbidity.

As noted in Zelkowitz and Cole (2008), Nolen-Hoeksema and Watkins (2011) laid out a heuristic framework for evaluating transdiagnostic processes. A key element of their framework is the notion that the proposed process actually cause, rather than simply correlate with, the outcomes in question. Similarly, the theoretical frameworks of NSSI and DE described previously both assume causality of the proposed mechanisms. Longitudinal research is critical for establishing causality, yet literature on long-term effects of SC on either NSSI or DE is sparse. You, Lin, and Leung (2015) assessed self-criticism, negative emotions, NSSI, and other personality/psychological variables in a sample of 3,600 adolescents from Hong Kong followed over 18 months. They found evidence of an interaction between SC and negative emotions and SC and past NSSI in prediction of subsequent NSSI. Although an important contribution to the literature, they largely focused on SC as a moderator rather than on its unique effects on NSSI. In an ancillary analysis, they found no significant effect of baseline SC on NSSI at 18-month follow-up after controlling for baseline NSSI and other psychological variables such as depressive and anxiety symptoms and emotion reactivity. Both the long-term follow-up period and inclusion of covariates with potential overlap with SC may have influenced their findings, however. More recently, Fox et al. (2018) found that SC significantly predicted NSSI at one-month follow-up among a sample of adults with histories of recent NSSI (i.e., two or more episodes in the past month). Their findings are notable in that SC was a significant predictor of NSSI at follow-up even after controlling for baseline behavior and using both implicit and

explicit (i.e., self-report) methods of assessing SC. Unclear, however, is whether their results would generalize to less clinically severe samples or how the inclusion of DE behaviors in the model would influence the findings.

As referenced in Zelkowitz and Cole (2008), Boone et al. (2014) examined self-critical perfectionism, psychological need frustration and binge eating in 566 Belgian adolescents. Self-critical perfectionism correlated significantly with binge eating over time. The authors also found that needs frustration at time 2 mediated the relation between self-critical perfectionism at time 1 and binge eating at time 3. Though intriguing, generalizing from the Boone et al. study is difficult, as it involved a particular variant of self-criticism, focused on adolescents, and assessed only binge eating and not the full range of DE behaviors. Procopio, Holm-Denoma, Gordon, and Joiner (2006) examined self-esteem and perfectionism (related constructs to SC) as predictors of bulimic symptoms over 2.5-year follow-up period among middle-aged women. They found significant correlations in the expected directions between Wave 1 perfectionism and self-esteem and bulimic symptoms at Wave 2 but nonsignificant relations after controlling for baseline bulimia symptoms and anxiety. Although noteworthy, the study did not disentangle binge eating behaviors from other food-related cognitions and was not designed to isolate the unique contribution of an SC-related construct on subsequent DE symptomatology.

Ecological momentary assessments of NSSI and DE offer more fine-grained data on the role of SC in both behaviors. In one of the first studies of its kind, Nock, Prinstein, and Sterba (2009) documented the affective, cognitive, and environmental contexts of NSSI urges and behaviors in a sample of adolescents. They found that self-hatred/anger at self significantly predicted acting on NSSI urges. They did not examine the context of binge/purge thoughts and behaviors, however. Shingleton et al. (2013) asked a sample of adolescents to report on the



cognitive and affective context surrounding urges to either binge, purge, or engage in NSSI. Participants reported that they more commonly experienced criticism prior to thoughts of bingeing/purging versus engaging in NSSI. Unclear is whether the criticism originated from the self or another or how such criticism related to actual NSSI or binge/purge behaviors. More recently, Turner, Yiu, Claes, Muehlenkamp, and Chapman (2016) sought to explore cognitive and affective precipitants of NSSI and DE behaviors using a daily diary study. They found that NSSI and DE behaviors co-occurred roughly 30 percent of the time. Their results indicate that participants were significantly more likely to act on binge/purge but not NSSI urges when they experienced self-hatred. The authors acknowledged small sample size ( $n = 25$ ) as a key limitation of their study. Clearly, further investigation of the role of SC in both NSSI and DE is required.

The first two aims of this dissertation are designed to fill the gaps in the literature described above. Specifically, Aim 1 is to investigate the association of SC and concurrent NSSI and DE behaviors and evaluate the proportion of the association between NSSI and DE attributable to SC. Aim 2 is to test SC as a transdiagnostic predictor of NSSI and DE. I will first test the unique associations of baseline SC with NSSI and DE, then repeat the analyses controlling for baseline behavior and covariates of negative affect and neuroticism. (These covariates were selected to set up a highly conservative test of SC, given strong correlations of SC with these constructs; Bagby & Rector, 1998). For both aims, I will conduct analyses among a sample of undergraduates and a sample of high-risk adults (defined as adults reporting, at minimum, lifetime experience of NSSI and/or DE behaviors). This will facilitate exploration and comparison of the effects of SC amid samples with differing levels of severity/engagement in NSSI and DE.

## **Life Stress as a Moderator of SC**

Claes and Muehlenkamp (2014) posited that it is not SC alone, but rather SC in combination with stressful life events, that increases risk of NSSI and DE. A recent meta-analysis indicated that life stress is associated with increased rates of NSSI, with stronger effects observed among community samples compared to clinical samples (Liu, Cheek, & Nestor, 2016). The authors noted that their analysis was largely based on cross-sectional studies and examined main effects of life stress rather than as a moderator of any underlying psychological process. In one of the few studies of this sort, Guerry and Prinstein (2010) found evidence of an interaction effect between life stress and negative attributional style in a sample of adolescents followed over 18 months. This finding offers preliminary support for the proposed Stress x SC interaction given the relevance of negative attributional style to SC. More research is necessary to assess whether the effect is specific to SC and replicates to other age groups, however.

Within the DE literature, considerable evidence points to the role of life stress in relation to these behaviors (e.g., Horesh et al., 1995; Pike et al., 2006; Welch, Doll, & Fairburn, 1997; see Ball & Lee, 2000, for a comprehensive review). Less attention has focused on stress as a moderator of the longitudinal effects of underlying psychological processes. This applies certainly to SC but also to perfectionism, a closely related construct that has received considerable attention in relation to DE (Bardone-Cone et al., 2007). Sassaroli and Ruggiero (2005) found that exam-related stress moderated the associations of low self-esteem and parental criticism and DE symptoms in a sample of high school students. Ruggiero et al. (2008) later identified significant associations of drive for thinness and body dissatisfaction under stressful conditions (versus non-stressful conditions) among undergraduates. Although intriguing, these works studied only specific form of stress (i.e., exam-related) and did not focus on DE behaviors

specifically. In summary, more research is clearly needed to study life stress as a moderator of SC for both NSSI and DE. Thus, Aim 3 is to test stressful life events as moderators of the association between SC and self-harm behaviors. I will again examine this relation in both an undergraduate and high-risk sample, facilitating comparisons across differing levels of clinical severity.

### **The Role of Reactive SC (Trait vs. State)**

The literature reviewed through this point has generally treated SC as a global, relatively static *traitlike* characteristic. Evidence exists, however, for a meaningful distinction between such traitlike SC and statelike fluctuations of SC within an individual (Zuroff, Sadikaj, Kelly, & Leybman, 2016). Such statelike fluctuations of SC call to mind Beck's (1979) concept of cognitive reactivity, the tendency to engage in maladaptive, self-critical thinking in response to negative affect. This is an important consideration, given substantial theoretical and empirical evidence that both NSSI and DE behaviors may serve or be perceived as serving an affect regulation function (e.g., Aldao, Nolen-Hoeksema, & Schweizer, 2010; Klonsky, 2007; Stice, 2001). Selby and Joiner (2009) proposed a model in which negative affect and cognition (specifically rumination) interact to promote an "emotional cascade." They theorized that individuals use maladaptive, often painful behaviors (e.g., self-harm, disordered eating, substance use, etc.) in an effort to disrupt the cascade. Ecological momentary assessment supports the role of fluctuations in both negative affect and rumination in prediction of NSSI and other dysregulated behaviors, including binge eating (Selby, Franklin, Carson-Wong, & Rizvi, 2013; Selby & Joiner, 2013). In a laboratory task, Arbuthnott, Lewis, and Bailey (2015)

observed significant increases in negative affect among individuals with a history of NSSI and significant decreases in positive affect among DE individuals following a rumination induction.

Taken together, these findings point to the interplay between cognition and emotional reactivity with potential relevance for NSSI and DE. This literature has so far focused on rumination in the context of intensifying negative affect. Whether and how reactive SC relates to self-harm behavior has yet to be explored. Thus, the fourth and final aim of this dissertation is to investigate the association of reactive SC to NSSI and DE in an undergraduate sample.

## Chapter II

### STUDY AIMS

I conducted a series of three studies to address the following aims. See Table 1 for a schematic of aims addressed by each study.

Aim 1 is to confirm and extend previous research identifying an association of NSSI, DE, and SC.

Hypothesis 1a. NSSI, DE (i.e., restriction, bingeing, and purging), and SC will show significant cross-sectional associations.

Hypothesis 1b. SC will account for a significant proportion of covariance between NSSI and each form of DE.

Aim 2 is to test SC as a transdiagnostic predictor of NSSI and DE.

Hypothesis 2a. Wave 1 (W1) SC will significantly predict increases in NSSI and DE at Wave 2 (W2).

Hypothesis 2b. W1 SC will significantly predict increases in NSSI and DE at W2 after controlling for baseline levels of each behavior.

Hypothesis 2c. W1 SC will significantly predict increases in NSSI and DE at W2 after controlling for baseline levels of each behavior, baseline negative affect, and baseline neuroticism.

Aim 3 is to examine life stress as a moderator of the relation between self-criticism and self-harm behaviors.

Hypothesis 3a: Increased life stress at W1 will interact significantly with W1 SC to increase risk of both NSSI and DE at W2.

Hypothesis 3b: The Stress x SC interaction will remain significant in the prediction of NSSI and DE after controlling for W1 levels of each behavior.

Aim 4 is to explore the relation of reactive self-criticism to NSSI and each form of DE using explicit and implicit assessment methods.

Hypothesis 4a: Reactive SC will relate significantly and positively to NSSI and each DE. The relation will be stronger for NSSI than for each DE.

## Chapter III

### STUDY 1

#### Summary of Aims

Study 1 aimed to confirm the association of SC with NSSI and DE and estimate the proportion of covariance between NSSI and DE accounted for by SC (Aim 1), test SC as a predictor of NSSI and DE (Aim 2), and test life stress as a moderator of the longitudinal relation between SC and NSSI and DE (Aim 3).

#### Participants

Participants consisted of 251 university students (79.5% female, 19.5% male, 0.4% transgender) recruited from a mid-sized Southern private school in fall 2016 and spring and fall 2017. Average age was 19.07 ( $SD = 1.23$ ). Eighty percent of participants at Wave 1 completed follow-up assessments.<sup>3</sup> Participants were recruited through the general university study subject pool (82.9%) and via study advertisements posted throughout campus (16.7%).<sup>4</sup> The sample showed moderate racial/ethnic diversity (65.7% Caucasian, 11.2% African American, 24.3% Asian or Asian American, 7.6% Hispanic or Latino, 1.6% another; participants could select more than one option for race/ethnicity, thus percentages do not add to 100%). Additionally, 10.8% of the sample identified as non-heterosexual.

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<sup>3</sup> Participants who did not complete Wave 2 assessments did not significantly differ from those who completed the study on age, gender, baseline SC, recruitment source, baseline NSSI, or any baseline DE variable.

<sup>4</sup> There were no significant baseline differences in SC or past-month NSSI or DE by recruitment method.

Table 2 shows baseline rates of NSSI and DE. Of note, scraping the skin, cutting, and self-biting were the most commonly endorsed forms of NSSI in the sample.

## **Methods**

Descriptions of the study in both the research subject pool and in on-campus flyers noted that the goal was to understand “how people think about themselves” and specifically referenced that the study included questions about NSSI and DE. Eligibility criteria included being between the ages of 18 and 25, enrolled at the university, and fluent in English. As part of the enrollment process, all participants received a brief orientation call with a research assistant, who explained the timeline and procedures associated with the study and answered questions. Participants received a link to complete Wave 1 study measures via the Qualtrics web-based survey system. They received a second link to complete Wave 2 measures after two months. All participants received either course credit or an \$8 Amazon.com gift credit (per wave) as compensation for their participation. Additionally, participants were entered into a raffle for an additional \$50 Amazon.com gift card if they completed both waves of the study. All participants were shown a list of university-based and national mental health resources after completing each survey. The first author or another graduate research assistant reviewed survey responses and contacted participants reporting elevated clinical symptoms (i.e., moderate to severe depressive symptoms, clinically significant eating concerns) to offer additional referrals to the university counseling center and wellness center.



## Measures

**NSSI.** The *Self-Injurious Thoughts and Behavior Interview, Self-Report version* (SITBI, Nock et al., 2007) is a structured interview designed to assess the frequency and function of various forms of self-injury. The full measure consists of five modules tapping various aspects of suicidality and NSSI. In the present study, a web-based version of the NSSI module was administered. This module asks about engagement in NSSI over a variety of time frames (i.e., past month, past year, lifetime), specific methods used, and other aspects of the behavior. The web-based NSSI module has shown both adequate test-retest reliability and construct validity in a validation study among a sample of older adolescents ( $M = 17.1$  years,  $SD = 1.9$  years; Nock et al., 2007).

**DE.** *Eating Disorder Examination-Questionnaire-6.0* (EDE-Q, Fairburn & Beglin, 2008). This is the most recent version of the EDE-Q (Fairburn & Beglin, 1994), a well-validated, widely used self-report measure of disordered eating. The 28-question instrument asks about DE behaviors (i.e., binge eating, fasting, and purging via self-induced vomiting, laxative misuse and excessive exercise) and DE-associated cognitions and emotions that an individual may have experienced in the past month.<sup>5</sup> The measure has a well-established factor structure, has been validated among undergraduate men and women, and has acceptable reliability across subscales in both groups (Lavender, De Young, & Anderson, 2010; Luce, Crowther, & Pole, 2008). We focused on the DE behavior items only to ensure the most salient comparison to NSSI (i.e., another behavior). Focusing on the DE behavior items (versus the subscales or total score) also safeguarded against artificially inflating the DE-SC correlation due to negative cognitions about one's weight or shape. Test-retest stability of the DE behavior items ranged from moderate to

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<sup>5</sup> To parallel assessment on the SITBI and more comprehensively document DE pathology in the sample, we added items assessing frequency of DE behaviors over the past year. See Table 2.

good in a validation study with a follow-up period of approximately two weeks (Luce & Crowther, 1999). As expected, stability was much poorer over a follow-up period of approximately 10 months (Bardone-Cone & Boyd, 2007).

*SC. Depressive Experiences Questionnaire* (DEQ; Zuroff, Quinlan, & Blatt, 1990). The DEQ is a 66-item measure developed by Blatt et al. (1976) to measure feelings and cognitions associated with depression (as distinct from more normative negative affect). It uses 1 (strongly disagree) to 7 (strongly agree) Likert scales. Factor analysis reveals three subscales: self-criticism, dependency, and efficacy. The measure has been validated among undergraduates and shown acceptable internal consistency in this population (alphas range from .73 to .81). Multiple item selection and scoring paradigms exist for the DEQ. We used the scoring system described in Bagby, Parker, Joffe, & Buis (1994), as this was shown to be psychometrically superior to other published methods (Desmet et al., 2007). The SC subscale based on this scoring method consists of nine items (e.g., “I often find that I don’t live up to my own standards or ideals.”) Internal consistency in the present sample was 0.72 at Wave 1 and 0.84 (both Cronbach’s  $\alpha$ ).

*Self-Rating Scale* (SRS; Hooley et al., 2002). The SRS is an eight-item measure of self-criticism that asks people to rate their agreement with statements such as “others are justified in criticizing me.” The measure has been used in a community sample, where it distinguished between self-injurers and non-self-injurers (St. Germain & Hooley, 2012). In the present sample, Cronbach’s alpha was 0.88 at Wave 1 and 0.91 at Wave 2.

*Forms of Self-Criticism/Self-Reassurance Scale* (Gilbert et al., 2004). This is a 24-item measure examining types of self-criticism in which individuals may engage (i.e., “Hated Self” and “Inadequate Self”). Participants note their agreement with each statement on a 0 (not at all like me) to 4 (extremely like me) scale. It was validated in a female undergraduate population,

showing adequate internal consistency (Cronbach's alpha > .85). It has subsequently been validated among both men and women in this population (Baiao, Gilbert, McEwan, & Carvalho, 2015). In the present study, we removed one item conceptually related to NSSI (i.e., “I become so angry with myself that I want to hurt or injure myself”) and combined the remaining items of the Hated Self and Inadequate Self subscales into a single self-criticism score. Internal consistency of the combined score was excellent (Cronbach’s  $\alpha = 0.88$  &  $0.90$  at Waves 1 and 2 respectively).

*Affect Misattribution Procedure* (AMP; Payne, Cheng, Govorun, & Stewart, 2005). The AMP is a computer-based task designed to tap implicit affect and has been used in assessments of disparate topics including NSSI (Franklin, Lee, Puzia, & Prinstein, 2014; Franklin, Puzia, Lee, & Prinstein, 2014). Individuals briefly view an affective stimulus followed by a blank screen and finally an ambiguous prompt (e.g., a Chinese pictograph). Respondents are asked to rate whether the pictograph is more pleasant or more unpleasant than the average pictograph. Before the task, they are told to ignore any affect provoked by the initial stimuli. Previous research indicates that, despite these admonitions, affective associations with the initial stimuli strongly impact participants’ ratings of the neutral prompt. In the proposed study, the affective stimuli will consist of six different self-focused words and short phrases (e.g., “me,” “myself,” “I,” “my own,”) and six different body-focused words and short phrases (e.g., “my body,” “my weight,” “my shape”). These will be interspersed with six neutral, six positive, and six negative terms (as characterized by the Affective Norms for English Words; Bradley & Lang, 1999). Scores are based on the proportion of positive evaluations of the target prompt. Lower scores for self-focused stimuli, for example, are thus thought to be reflective of greater SC. In the present sample, AMP scores showed small-to-medium correlations with self-report SC measures

(Pearson's  $r$  range from  $-.21$  to  $-.24$  for the self-focused stimuli and  $-.26$  to  $-.38$  for body-focused stimuli. All correlations were significant at  $p < .01$ ). This is consistent with past research on the correlation of implicit and explicit assessments (Hoffman, Gawronski, Gschwendner, Le, & Schmitt, 2005.)

**Neuroticism.** *The Big Five Inventory*; Neuroticism subscale (BFI; John, Naumann, & Soto, 2008). The BFI is an instrument designed to assess personality according to a five-factor model. We administered the eight-item Neuroticism subscale (example item: "I am someone who can be tense"). Respondents rate their agreement with each statement on a 1 (strongly disagree) to 5 (strongly agree) scale. This subscale has shown adequate internal consistency ( $\alpha = .81$ ) among a sample of undergraduate students (Thalmayer, Saucier, & Eigenhuis, 2011). Cronbach's  $\alpha$  in the present sample was  $.81$ .

**Negative Affect.** *Positive and Negative Affect Scales*; Negative subscale (PANAS, Watson, Clark, & Tellegen, 1988). The PANAS is a commonly-used measure that assesses the extent to which respondents have specific positive and negative affective states in a given time period. Respondents endorse the extent to which they experienced a particular emotion in that time period on a 1 (not at all) to 5 (extremely) Likert scale. The NA and PA subscales have shown adequate internal consistency and test-retest reliability for time frames up to one year in university samples (Watson, Clark, & Tellegen, 1988). Internal consistency for the NA subscale in the present sample at Wave 1 was excellent (Cronbach's  $\alpha = .87$ ).

**Stressful Life Events.** *Inventory of College Students Recent Life Experiences* (ICSRLE; Kohn, Lafreniere, & Gurevich, 1990). The ICSRLE was developed to measure exposure to stressors relevant to a college population. It consists of 49 items, and respondents indicate the extent to which each item reflects a source of stress for them in the preceding month. It was

developed and validated in an undergraduate population and showed adequate reliability and internal consistency. Cronbach's alpha in the present sample at Wave 1 was .93.

### **Data Preparation and Analytic Approach**

Data were visually inspected for invalid or careless response patterns (e.g., “straight-lining”). We subjected all SC measures to a confirmatory factor analysis to assess whether they were reflective of a single latent SC factor. The model showed excellent fit ( $\chi^2(2, N = 251) = 0.25, p = .88$ ; NFI = 1.00; IFI = 1.01; CFI = 1.00; RMSEA = .00, 90% CI [.00, .90]). The SRS, FSCRS, and DEQ (all explicit measures) showed strong loadings on latent SC ( $\lambda_s > .70$ ), but the self-focused AMP score showed poor loading ( $\lambda = -.27$ ). We thus decided to combine all the explicit SC measures using factor scores for subsequent analyses and analyze relations of NSSI and DE to AMP (self-focused and body-focused scores) in separate models.

We conducted several steps to prepare the DE data for analyses. First, we identified four items that reflected restriction behaviors (see Appendix for specific items). Response choices for these behaviors refer to a range of days during which the respondent engaged in that particular behavior (e.g., “0 days,” “1-5 days,” etc.). We calculated the mean number of days in each range and coded responses accordingly (Note: Participants reporting “0 days” retained a score of 0). This allowed us to calculate an approximate mean count of days in which the participant reported engaging in restriction. Because this restriction score encompassed behaviors that could be interpreted as mild (e.g., excluding favored food items, attempting to limit intake) as well as more severe behaviors (e.g., fasting), we also analyzed responses to the fasting item alone. We created a composite purging item by summing episode counts of laxative misuse, self-induced

vomiting, and excessive exercise.<sup>6</sup> Bingeing was represented by self-reported days on which the participant reported eating an unusually large amount of food while experiencing a sense of loss of control. Finally, we summed scores for fasting, purging, and bingeing to create a composite DE score.

To prepare NSSI data for analyses, we first inspected distributions of past-month NSSI at Waves 1 and 2. As expected, distributions were both zero-inflated and highly skewed (see Table 2). We thus elected to bin the data as follows: Individuals who reported no NSSI received scores of “0,” those reporting 1 to 2 episodes received a score of “1,” those reporting 3 to 4 episodes were scored “2,” and those reporting 5 or more episodes received a score of “3.” Bins were selected to parallel the distribution of raw scores (i.e., bins contained progressively fewer participants and corresponded to engaging in NSSI on a monthly to bimonthly, weekly, and more frequent basis.)

The skewness and zero-inflation of the NSSI and DE variables made traditional regression approaches inadvisable. Past investigation of NSSI has instead turned to zero-inflated approaches, such as the hurdle models (e.g., Zelkowitz, Cole, Han, & Tomarken, 2016). Such models assume a zero-inflated distribution of data and explicitly calculate the relation of a variable to whether a response is zero or non-zero and (within the non-zeros) to the actual count. Hurdle models are not readily adaptable to multivariate responses, however. Because modeling the relation of both NSSI and DE to SC was a key goal of the paper, we instead opted to conduct path analysis using maximum likelihood estimation with robust standard errors (MLR). This

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<sup>6</sup> The choice to combine across purging items was motivated both by low rates of laxative misuse and self-induced vomiting and because all three behaviors are considered forms of “inappropriate compensatory behaviors” in DSM-5.

technique allowed us to examine multivariate outcomes while accounting for the skewness in the data.

To evaluate the association of NSSI and DE (Aim 1), we first examined zero-order correlations between NSSI and the composite DE variable. I then tested correlations between NSSI and each specific form of DE. All zero-order correlations among outcome variables were analyzed using both Spearman's  $\rho$  and Pearson's  $r$  with robust standard error estimates (RSE). To assess the proportion of covariance between NSSI and DE accounted for by SC, I compared the zero-order Pearson's correlation of NSSI and each form of DE at Wave 1 with the residual correlation between those variables regressed on Wave 1 SC.

I conducted a series of path analyses to test SC as a transdiagnostic predictor of NSSI and DE (Aim 2). In brief, I first regressed both Wave 2 NSSI and DE on Wave 1 SC. (I tested each form of DE in separate analyses, such that each model contained NSSI and one form of DE as endogenous variables). I then tested SC as a predictor of both NSSI and DE (examined in separate models), controlling for baseline levels of each behavior. Significant regression path coefficients from SC to NSSI and DE were taken as support for transdiagnostic prediction by SC. I then extended the analyses by testing a series of path analyses in which both Wave 2 NSSI and Wave 2 DE were regressed on SC, controlling for (a) baseline levels of both behaviors, (b) neuroticism, and (c) negative affect (neuroticism and negative affect were tested in separate models). Given the directional nature of hypotheses related to SC, one-tailed tests with  $p < .05$  were used.

To test Aim 3, I first centered the stress variable and formed an interaction term with SC. (Factor scoring already produced an SC variable with a mean of 0, thus no further centering was needed). I entered the Stress x SC interaction term into the model along with all main effects.

Again, I examined NSSI and each DE variable as multivariate outcomes. I first assessed for moderation in the cross-sectional data prior to assessing for longitudinal moderation effects.

In all models, I used full information maximum likelihood estimation in order to retain cases with partial data. All models were thus fully identified; as such, fit indices are uninterpretable. Data preparation was conducted in SPSS v. 24, and analyses were conducted using MPlus v. 7 (Muthén & Muthén, 2012).

## Results

### Aim 1

Table 3 shows cross-sectional associations of SC, NSSI, and DE at Waves 1 and 2 (significance levels are based on robust standard errors, see Appendix A, Table A1, for values based on Spearman's  $\rho$ ). Explicit SC measures (based on the factor score composite) correlated significantly with NSSI, the DE composite, restriction and fasting at Wave 1; SC correlated significantly with NSSI and all DE behaviors at Wave 2. Implicit SC (i.e., self-focused and body-focused AMP stimuli) did not correlate significantly with any behavior at Wave 1. At Wave 2, scores for self-focused stimuli correlated significantly with the DE composite score and purging ( $p < .05$ ) and fasting ( $p < .10$ ). Scores for body-focused stimuli correlated significantly with NSSI and each DE behavior (correlation with bingeing was significant at  $p < .10$ ).

Table 4 shows both the zero-order correlations of Wave 1 NSSI and each DE variable and the partial correlations controlling for Wave 1 SC. (Due to nonsignificant results for Wave 1 implicit SC measures, I calculated partial correlations based on explicit SC only). NSSI showed small but significant correlations with the DE composite score, bingeing, and purging; the association with bingeing was not in the expected direction. The association of fasting and NSSI



at Wave 1 trended significant and was in the expected direction. After controlling for SC, the correlation of NSSI and the DE composite was no longer significant. Correlations of NSSI with purging, restriction, and fasting dropped by 7 to 29 percent after controlling for SC. The association of NSSI and bingeing became more negative after controlling for SC.

## **Aim 2**

Table 5 shows the longitudinal effects of SC on NSSI, the DE composite, and each specific form of DE. The top portion of the table (i.e., Model 1) depicts the effect of explicit SC on Wave 2 NSSI or DE prior to controlling for baseline levels of the dependent variable; the middle portion (i.e., Model 2) shows results for NSSI and each DE behavior after controlling for that behavior at baseline. The bottom portion of Table 5 (i.e., Model 3) shows the effect of Wave 1 SC on Wave 2 NSSI or DE after controlling for *both* behaviors at baseline. The dependent variable of interest is listed in the far-left column. Estimates for the effect of SC on Wave 2 NSSI varied slightly depending on which DE variable was concurrently assessed. For completeness, we present the range of estimates for the effect of Wave 1 SC on Wave 2 NSSI.

Measured via self-report instruments (i.e., explicit assessment), Wave 1 SC significantly predicted NSSI, composite DE, and each form of DE behavior prior to controlling for baseline levels of the NSSI or DE. Neither implicit SC measure (self-focused stimuli or body-focused stimuli) significantly predicted W2 NSSI prior to controlling for baseline behavior; thus all subsequent analyses focused on SC measured via self-report instruments.<sup>7</sup> SC remained a significant predictor of NSSI, the DE composite, bingeing, and fasting after controlling for baseline behavior. Although not a specific aim of the study, we also noted longitudinal effects of

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<sup>7</sup> Self-focused and body-focused AMP both significantly predicted the DE composite and fasting prior to controlling for baseline levels of each behavior. After controlling for baseline behavior, body-focused AMP significantly predicted the DE Composite at Wave 2 ( $b = -4.63$ ,  $SE = 2.26$ ,  $\beta = -.13$ ,  $z = -2.05$ ). Paths from body-focused AMP to Wave 2 Fasting and paths from self-focused AMP to Wave 2 DE composite and fasting were nonsignificant.

the DE composite and bingeing at Wave 1 on subsequent NSSI (controlling for W1 NSSI).

Baseline NSSI had no significant effect on any form of DE at Wave 2. (See Tables 6 and 7).

Neuroticism and negative affect each showed large and significant correlations with SC ( $r_s = .67$  and  $.70$ , respectively,  $p_s < .001$ ). After controlling for baseline NSSI and DE behaviors and negative affect, however, SC remained a significant predictor of NSSI, DE composite, bingeing, and fasting at Wave 2. SC remained a significant predictor of the same outcomes after controlling for baseline behaviors and neuroticism. See Table 8 for path estimates.

### **Aim 3**

Relations of the SC x Stress interaction term and NSSI and each form of DE were nonsignificant at Wave 1. Thus, I did not proceed with a test of the longitudinal moderation effect. I conducted post-hoc analyses of the main effects of SC and stress in relation to each behavior, again using one-tailed tests at  $\alpha = .05$  to reflect directional hypotheses. NSSI was not positively related to stress after controlling for SC. The NSSI-SC relation remained significant after controlling for stress ( $b = 0.07$ ,  $SE = 0.03$ ,  $\beta = .32$ ,  $z = 2.51$ , 95% CI [0.02,  $\infty$ ]). Controlling for SC, stress was significantly related to the DE composite ( $b = 4.34$ ,  $SE = 1.84$ ,  $\beta = 0.18$ ,  $z = 2.36$ , 95% CI [1.31,  $\infty$ ]), bingeing ( $b = 1.96$ ,  $SE = 0.87$ ,  $\beta = 0.22$ ,  $z = 2.25$ , CI [0.53,  $\infty$ ]) and restriction ( $b = 10.38$ ,  $SE = 5.28$ ,  $\beta = .15$ ,  $z = 1.97$ , 95% CI [0.03,  $\infty$ ]). SC showed a significant, positive relation only with fasting after controlling for stress ( $b = 1.57$ ,  $SE = 0.50$ ,  $\beta = 0.23$ ,  $z = 3.16$ , 95% CI [0.78,  $\infty$ ]).

## **Study 1 Discussion**

Three key findings emerged from Study 1. First, we replicated and extended findings of an association between NSSI and DE by focusing on actual DE behaviors rather than the constellation of affective, cognitive, and behavioral symptoms associated with eating disorders.

Specifically, we noted significant associations between past-month NSSI and a composite of DE behaviors and NSSI and bingeing and purging. We then determined the proportion of the correlation between NSSI and each DE attributable to SC. Estimates of the proportion of correlation due to SC varied by behavior but appeared greatest for the association between NSSI, binge eating, and fasting and lowest for the correlation between NSSI, purging, and a broader compendium of restriction behaviors. Finally, we documented longitudinal effects of SC on both NSSI and DE in a university sample. The results extend previous work in high-risk and clinical samples (Fox et al., 2018) and provide further support for SC as a transdiagnostic risk factor for two important self-harm behaviors. We acknowledge a key limitation of the present study, however, in that participants demonstrated low rates of NSSI over the follow-up period (although rates of DE at follow-up were higher). We thus sought to explore our study aims in a high-risk sample in Study 2.

## Chapter IV

### STUDY 2

#### Summary of Aims

As in Study 1, Study 2 aimed to confirm the association of SC with NSSI and DE (Aim 1), test SC as a predictor of NSSI and DE (Aim 2), and test life stress as a moderator of the longitudinal relation between SC and NSSI and DE (Aim 3). Study 2 examines these aims within a high-risk sample.

#### Participants

Participants consisted of 517 adults aged 18-30 ( $M = 24.65$ ,  $SD = 3.58$ ) recruited from across the United States via Qualtrics Panels, a Web-based research volunteer management system. Volunteers completed surveys in exchange for points redeemable for airline miles, restaurant gift cards, etc. Individuals were eligible to participate if they reported a lifetime history of either NSSI, DE, or both behaviors. We attempted to recruit roughly equal numbers of participants with each behavior history (33.66% NSSI, 30.37% DE, 35.98% NSSI and DE)<sup>8</sup>. The sample was overwhelmingly female (88.78%); 9.09% identified as male, 2.13% identified as transgender or another gender. Participants self-reported race/ethnicity (72.53% Caucasian, 14.31% Hispanic or Latino, 12.19% African American, 6.58% Asian or Asian American, 2.71% American Indian, 1.55% another; percentages do not add to 100 as participants could select more

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<sup>8</sup> Screening categories were to ensure representativeness of each behavior in the study but were not used as actual variables in analyses.

than one option). Among participants, 29.79% described their occupation as “mostly focused on school;” the remainder endorsed a focus on work or other.

First, 307 individuals completed measures at Wave 1. Of these, 139 (45.28) completed follow-up assessments.<sup>9</sup> We then recruited an additional 210 participants at Wave 2 to replace those lost to follow-up (thus generating our total N of 517)<sup>10</sup>. These additional participants were also screened for NSSI/DE history to ensure that each behavioral history pattern (i.e., NSSI only, DE only, NSSI+DE) was equally represented among participants at Waves 1 and 2.

## Methods

Participants meeting our age and English-language requirements received an invitation to complete the pre-screener. Those who qualified were then invited to participate in the full study, which took approximately 17 minutes to complete. Responses were anonymous (thus precluding follow-up), but all participants viewed a page of national mental health resources upon completion of the study. Participants were prompted to complete Wave 2 four weeks after completing Wave 1. As noted, participants received credit redeemable for rewards as compensation for their effort.

## Measures

**NSSI.** We retained use of the online SITBI-NSSI module from Study 1. We added the Deliberate Self-Harm Inventory (DSHI, Gratz, 2001) to facilitate even more comprehensive

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<sup>9</sup> Participants who did not complete Wave 2 assessments were younger ( $M$  difference = 1.05,  $SE$  difference = 0.42,  $t(301.61) = 2.52$ ,  $p = .01$ , 95% CI [.23, 1.87], less self-critical ( $M$  difference = 0.24,  $SE$  difference = 0.11,  $t(305) = 2.24$ ,  $p = .03$ , 95% CI [0.03 – 0.45]). They did not differ significantly on sex, baseline NSSI, or any baseline DE variable.

<sup>10</sup> See Data Preparation for treatment of missing data.

assessment of NSSI. The DSHI assess individuals' engagement in 17 different self-harm behaviors. If respondents endorse a lifetime history of the behavior, they are then asked to estimate their lifetime, past-year, and past-month frequency of engaging in the behavior. The validation study supported its internal consistency (Cronbach's  $\alpha = .82$ ) and test-retest reliability. In the present study, Cronbach's  $\alpha$  was .71 at Wave 1 and .74 at Wave 2.

**DE.** We retained use of the EDE-Q from Study 1 and again focused on the DE behavior items to promote comparability with NSSI. We added two additional DE measures to assess for convergence of results beyond the single-item assessments of DE behaviors from the EDE-Q. Specifically, we added the Dutch Eating Behaviors Questionnaire, Restrained Eating Subscale (DEBQ; Van Strien, Frijters, Bergers, & Defares, 1986). This subscale consists of 10 items designed to measure restrained eating. Responses are scored on a 5-point Likert scale. An example item is "Do you try to eat less at mealtimes than you would like to eat?" Cronbach's  $\alpha$  was .93 at both Waves 1 and 2 in the present study.

We also added specific items related to binge eating from the Bulimia Test-Revised (BULIT; Thelen, Farmer, Wonderlich, & Smith, 1991). Items were selected by rational inspection of the full BULIT-R. Questions about specific items were resolved through consensus among the research team. The final set of binge eating items consisted of eight items (Example item: "I would presently label myself a 'compulsive eater,' (one who engages in episodes of uncontrolled eating).") Internal consistency of the binge eating items was excellent at both Wave 1 (Cronbach's  $\alpha = .89$ ) and Wave 2 (Cronbach's  $\alpha = .91$ ).

We followed a similar process to identify BULIT-R items related to purging. We initially identified eight items that seemed to reflect some aspect of purging (Example item: "How often do you intentionally vomit after eating?") Internal consistency of these items was poor at both Waves 1

and 2 (Cronbach's  $\alpha$ s = .56 and .48). This was largely driven by two items that listed specific frequencies of laxative or diuretic misuse as response options on the Likert scale. After removing these items, the final six-item scale showed good internal consistency at Waves 1 and 2 (Cronbach's  $\alpha$ s = .84).<sup>11</sup> See Appendix B for items comprising the BULIT Binge and Purge subscales.

**SC.** We retained all SC measures from Study 1. Internal consistency for the SRS was .86 at Wave 1 and .89 at Wave 2. For the FSC, internal consistency was .89 at Wave 1 and .92 at Wave 2. Internal consistency for the DEQ was also acceptable (Cronbach's  $\alpha$  = .83 at Wave 1 and .79 at Wave 2).

**Stress.** Participants who reported their occupation as “mostly focused on school” completed the ICSRLE (see Study 1 for measure description). Internal consistency in the Study 2 sample was .95. Participants who reported their occupational focus as “work” or “other” completed the Survey of Recent Life Events (SRLE; Kohn & MacDonald, 1992). This is a 51-item measure developed by the same authors as the ICSRLE. Items are largely consistent with the student-focused tool except for changes in language reflecting the intended population (e.g., references to “boss” versus “professor”). As with the ICSRLE, items are designed to assess normative stress and “hassles” rather than traumatic incidents. Internal consistency in the present sample was also excellent (Cronbach's  $\alpha$  = .95).

### **Data Preparation and Analytic Approach**

An additional 61 participants were screened out for excessively fast responding (determined by the research team as <15 min after examining the mean and median response times to the survey) or for incorrect responses to a series of validity indicators. In the final

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<sup>11</sup> The final purging scale retained items specifying relative frequency of laxative and diurectic misuse (e.g., “seldom,” “frequently”).

sample ( $n = 517$ ), we prepared the DE count variables from the EDE-Q as described in Study 1. We chose to analyze data from the EDE-Q separately from the added DE measures (i.e., the DEBQ and BULIT-R Binge and Purge subscales) to facilitate comparison of our findings with results from Study 1 and to preserve the count nature of the EDE-Q data. See Appendix A for results based on the BULIT and DEBQ subscales.

In contrast, we elected to combine responses to the SITBI and DSHI as data from both measures was in the form of counts. The measures also asked about largely non-overlapping forms of NSSI; combining responses ensured the most comprehensive assessment of the construct. Three behaviors (cutting, burning, and biting) were assessed in the same fashion by the SITBI and DSHI. We averaged responses to each behavior across the two measures (e.g., SITBI cutting and DSHI cutting) to come up with a single count for these three behaviors. We then summed responses for the remaining behaviors across the SITBI and DSHI.<sup>12</sup> Table 9 shows baseline statistics for NSSI and DE variables among Study 2 participants recruited at Wave 1 (bottom panel shows baseline statistics for those entering the study at Wave 2). As in Study 1, the NSSI variable was highly skewed. We thus binned the NSSI data in the following fashion: Participants reporting no past-month NSSI received scores of “0,” those reporting 1, 2, and 3 episodes were scored “1,” “2,” and “3,” respectively. Those reporting 4-5 episodes were scored “4,” 6-7 as “5,” 8-10 as “6,” 11-13 as “7,” 14-16 as “8,” 17-19 as “9,” 20-24 as “10,” 25-36 as “11,” 37-52 as “12,” 59-74 as “13,” 75-107 as “14,” and all remaining values as “15.” Bins were selected to mirror the actual distribution of counts and contain successively fewer numbers of observations. The binned NSSI count variable showed acceptable skew at Wave 1 (skewness = .54, *SE* of skewness = .09) and Wave 2 (skewness = .31, *SE* of skewness = .09).

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<sup>12</sup> In keeping with established methods in the NSSI literature, we did not include hair-pulling or picking at wounds in the NSSI sum.



We proceeded to assess Aims 1, 2, and 3 according to the analyses described in Study 1. In the present study, we rotated eight DE variables (i.e., all EDE-Q variables, DEBQ, BULIT Binge and Purge) through the models portrayed in Figures 1 and 2. We again testing the longitudinal effect of SC prior to controlling for any baseline behavior, after controlling for NSSI or DE (in separate models), and after controlling for *both* NSSI and DE. As in Study 1, we first tested for a Stress x SC moderation effect in the cross-sectional data before examining the effect in the longitudinal data.

## Results

### Aim 1

Table 10 shows the cross-sectional associations of SC, NSSI, and DE at Waves 1 and 2 (all analyses based on Pearson's  $r$  with robust standard errors, associations based on Spearman's  $\rho$  listed in Appendix A, Table A3).<sup>13</sup> SC showed significant, small-to-medium correlations with NSSI and each DE variable in both waves.

Table 11 compares the zero-order correlations of NSSI and each DE variable with the partial correlations of the same, controlling for SC. NSSI showed small but significant cross-sectional associations with the DE composite and fasting. Correlations with bingeing and purging were not significant, association with restriction trended toward significance.<sup>14</sup> As Table 11 illustrates, controlling for SC decreased the correlation of NSSI and each DE behavior; all NSSI-DE correlations were non-significant after partialling out SC. SC accounted for approximately 40 percent of the correlation between NSSI and the DE composite. The proportion

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<sup>13</sup> For the sake of cogency and to facilitate comparisons with Study 1, I present only DE variables based on the EDE-Q here. See Appendix A for analyses based on the DEBQ, BULIT-Binge, and BULIT-Purge.

<sup>14</sup> But see results for BULIT-Binge, BULIT-Purge, and DEBQ.

of the association between NSSI and specific DEs accounted for by SC was lowest for NSSI and the purging count variable and greatest for NSSI and the bingeing count variable.

## **Aim 2**

Table 12 shows the longitudinal effect of SC on NSSI and each DE variable, prior to controlling for baseline behavior (Model 1), after controlling for baseline NSSI *or* DE only (Model 2), and after controlling for both baseline NSSI *and* DE (Model 3). SC significantly predicted each aspect of DE prior to controlling for baseline behavior. After controlling for baseline DE behaviors only, SC significantly predicted the DE composite, purging, and fasting at Wave 2. Results remained the same after controlling for baseline DE and NSSI (Model 3). SC did not significantly predict NSSI prior to controlling for baseline behavior (Model 1), after controlling for baseline NSSI only (Model 2) or after controlling for baseline NSSI and each DE variable (Model 3).<sup>15</sup>

As in Study 1, we noted the longitudinal reciprocal effects of NSSI and DE (although this was not an explicit aim of the study). Wave 1 purging trended toward significantly predicting Wave 2 NSSI after controlling for baseline NSSI, SC, and purging (see Table 13). Wave 1 NSSI did not significantly predict any DE variable at Wave 2 after controlling for baseline DE (see Table 14).

## **Aim 3**

Stress did not interact significantly with SC in relation to NSSI or any DE variable. As in Study 1, we elected to explore main effects of SC and stress in relation to Wave 1 NSSI and DEs. Wave 1 SC remained significantly associated with Wave 1 NSSI after controlling for stress

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<sup>15</sup> But see Appendix B for results based on the SITBI alone rather than the NSSI composite variable.

( $b = 0.55$ ,  $SE = 0.28$ ,  $\beta = 0.14$ ,  $z = 2.00$ , 95% CI [0.03,  $\infty$ ]). The NSSI-stress relation was not significant.

Among the DE variables, SC remained significantly associated with only fasting and restriction after controlling for stress. In contrast, stress was associated with all DE variables after controlling for SC. See Table 15 for estimates.

### **Study 2 Discussion**

Three key findings emerge from Study 2. First, as in Study 1, we documented significant associations between past-month NSSI and a composite of past-month DE behaviors. We specifically showed a significant association of NSSI and fasting. We again calculated the extent to which SC contributes to the association of NSSI and a range of DE behaviors. Findings indicated that the proportion accounted for by SC was greatest for the NSSI-binge association and smallest for the NSSI-purge association. Second, we did not find evidence of a longitudinal effect of SC on NSSI (either prior to or after controlling for baseline behavior), but SC did emerge as a longitudinal predictor of several forms of DE (controlling for baseline behavior). Finally, findings did not support an interaction effect between stress and SC for NSSI or any form of DE.

## **Chapter V**

### **STUDY 1 AND STUDY 2 GENERAL DISCUSSION**

#### **Aim 1**

Clinicians and researchers alike increasingly recognize substantial comorbidity of DE and NSSI rather than viewing them as wholly distinct, unrelated behaviors. This awareness has been based, in large part, on research documenting significant associations of DE diagnosis and lifetime history of NSSI (Cucchi et al., 2016; Svirko & Hawton, 2007). In doing so, however, the literature has focused on comorbidity with less regard to whether these behaviors presented synchronously or asynchronously (an important consideration in the study of comorbidity; Valderas, Starfield, Sibbald, Salisbury, & Roland, 2009). My first aim was to confirm past work documenting associations of NSSI, DE, and SC, extending this work by a) focusing on NSSI and DE behaviors specifically, and b) assessing both within a specific timeframe.

Few studies have examined the correlations between NSSI and DE behaviors alone (versus the broader constellation of behavior, affective, and cognitive symptoms associated with a DE diagnosis). Arbuthnott, Lewis, and Bailey (2015) showed small but significant correlations between lifetime NSSI and lifetime report of DE behaviors in an undergraduate sample. Our findings of the NSSI-DE association are consistent with their results (and the broader literature on NSSI-DE comorbidity). We extend this previous work, however, in that we documented this effect in a) both undergraduate and high-risk samples and b) by examining past-month behaviors (versus lifetime NSSI and DE).

Cucchi et al. (2016) identified higher rates of NSSI among individuals with bulimia nervosa (BN) compared to anorexia nervosa (AN). Within the undergraduate sample, we observed significant associations of NSSI with bingeing (although this association was negative) and purging. This last, specifically, is a behavior consistent with BN and AN-purging subtype. Within the high-risk sample, however, we observed the opposite pattern, in that the association of NSSI with fasting was significant whereas associations with bingeing and purging were not significant. The explanation for this discrepancy may be more statistical than conceptual: Point estimates for the NSSI-DE associations were fairly comparable across samples. The undergraduate sample had substantially lower rates of participants endorsing the target behaviors than the high-risk sample (as anticipated). Data from undergraduate participants who endorsed NSSI may have been particularly influential, allowing associations to emerge as significant in that sample while similar estimates were not significant in the high-risk sample due to greater variability in the data.

Another explanation could stem from the operationalization of purging in both samples. We collapsed across excessive exercise, self-induced vomiting, and laxative misuse. Doing so allowed us to capture more fully the spectrum of purging behaviors outlined in DSM-5 and increase power to detect effects. However, the notion of “excessive exercise” is inherently subjective. Self-reported responses to this item could have reflected normative forms of exercise, thus diluting the effect in our samples. Post-hoc analyses revealed stronger correlations in the high-risk sample between NSSI and self-induced vomiting versus NSSI and the combination of vomiting, laxative misuse, and excessive exercise, providing support for this hypothesis.

Results on the association of SC with NSSI and DE add to the growing body of literature on these relations. Meta-analytic work has documented significant associations of SC with NSSI

and DE examined separately (Zelkowitz & Cole, 2018). Here, we present evidence of significant associations among SC, NSSI, and DE examined concurrently, over discrete time periods, and using multiple measures of SC to enhance reliability and validity of our assessments.<sup>16</sup> The consistency of this association in both the undergraduate and high-risk sample helps build the case for SC as a common psychological substrate of NSSI and DE across differing levels of clinical severity. We further found that SC accounted for roughly a quarter of the (small) correlation between NSSI and DE, with results varying by the specific DE behavior considered. It is notable that, of the DE behaviors considered, SC accounted for the smallest portion of the correlation between NSSI and purging.<sup>17</sup> This is consistent with findings by Turner et al. (2016) that self-hatred predicted acting on binge/purge urges but not on urges to engage in NSSI. Taken together, the findings suggest psychological processes other than self-criticism may contribute more consequentially to co-occurrence of these behaviors (e.g., negative urgency; Peterson & Fischer, 2012).

## **Aim 2**

A key goal of the present work was to test the longitudinal effects of SC on subsequent NSSI and DE, thus testing an important tenet of etiological theories proposed by Svirko and Hawton (2007) and Claes and Muehlenkamp (2014). Results were mixed, in that SC significantly predicted NSSI and several forms of DE in the undergraduate sample (even after controlling for baseline behavior and the closely related constructs of neuroticism and negative

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<sup>16</sup> Only one other study to our knowledge has used both explicit and implicit methods of assessing SC (Fox et al., 2018). The authors report associations of SC and NSSI using both methods of assessment. Our findings partially replicate theirs, in that both explicit and implicit SC were associated with NSSI at Wave 2 only.

<sup>17</sup> Notable given previous data on associations of NSSI and BN/AN purging subtype and the conceptualization of purging as a particularly violent form of DE.

affect). In the high-risk sample, however, SC significantly predicted only the DE composite, purging, and fasting after controlling for baseline behavior. (SC did not significantly predict NSSI even prior to controlling for baseline behavior). Several factors may have contributed to the discrepant results between the two samples. First, we used a composite of the SITBI and DSHI to assess NSSI in the high-risk sample. Although the goal was to achieve a comprehensive assessment of NSSI, the broader set of individual behaviors included in the composite measure may actually have resulted in increased response variability, thus obscuring an effect for SC. This might also explain the discrepancy between our findings and those reported in Fox et al. (2018). There, the authors observed significant effects of SC on subsequent NSSI among adults with a recent history of the behavior. Our factor-score method for assessing SC and efforts to control for baseline DE may also contribute to our divergent results.

A second explanation for the inconsistency in results between Studies 1 and 2 could be the low rate of engagement in NSSI at follow-up in the undergraduate sample. In this case, data from each participant who did engage in NSSI would have particularly high potential to affect the results. It is also possible that the slight difference in follow-up periods (two months in Study 1 versus one month in Study 2) may have influenced the results. Finally, demographic differences between the two samples may have also impacted the results. The high-risk sample was slightly older and thus had more time to have established a history of NSSI. The undergraduate sample also encompassed greater ethnic/racial diversity. Further research is necessary to explore age and race/ethnicity as moderators of SC or other psychological processes in relation to NSSI. This will be an important area of inquiry, given growing research that supports racial/ethnic differences in correlates of the behavior (Gholamrezaei, De Stefano, & Heath, 2017; Polanco-Roman, Tsypes, Soffer, & Miranda, 2014).

Taken together, results of Studies 1 and 2 offer mixed support for the SC-to-NSSI prospective link proposed in both theoretical models of NSSI and DE.<sup>18</sup> Studies of SC and NSSI in the context of DE behaviors are limited. Itzhaky, Shahar, Stein, & Fennig (2016) found a significant cross-sectional relation between SC and past-year NSSI among a sample of adolescents receiving inpatient treatment for DE. Our findings build on this work by analyzing the longitudinal effect of SC on NSSI in two different samples (i.e., undergraduates and high-risk adults versus those receiving inpatient treatment) while also controlling for DE symptomatology. Further research will be required to reconcile the discrepant findings SC as a predictor of NSSI in these two studies. Another important area of inquiry will be assessing whether self-reported function of the behavior moderates the SC-NSSI relation. Affect regulation and self-punishment are the two most commonly reported functions of NSSI (Klonsky, 2007). An intriguing possibility (and intuitively valid) possibility is that SC is a more salient predictor for NSSI among those who engage in the behavior primarily as a means of self-punishment. Further research will be required to test this hypothesis, however.

Within the DE behaviors, we observed significant effects of SC on a composite of DE behaviors and on fasting in both Studies 1 and 2. In Study 1, SC also significantly predicted binge eating, whereas in Study 2, the effect on purging was significant (all after controlling for baseline DE behavior and NSSI). Minimal research has been conducted on SC as a predictor of DE behaviors writ large, and less still on specific DE behaviors. In cross-sectional research, Fennig et al. (2008) noted higher levels of self-criticism among patients with bulimia nervosa compared to those with anorexia nervosa. However, the authors did not examine self-criticism in relation to specific DE behaviors or control for rates of purging in the two subgroups. Earlier

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<sup>18</sup> Similarly, it offers mixed support for the benefits and barriers model of NSSI (Hooley & Franklin, 2017).



work by Williams et al. (1993) found no differences between self-directed hostility in patients with bulimia versus anorexia (although both groups differed from both healthy controls, individuals with obesity, and those who were dieting). Past research on DE and perfectionism, in contrast, has shown stronger associations of that construct with fasting and purging compared to bingeing and longitudinal associations with AN (Bardone-Cone, et al., 2007; Forbush, Heatherton, & Keel, 2006). Our findings on the longitudinal effect of SC on fasting thus extends previous investigation on perfectionism. The consistency of our findings across samples lends support for the role of SC in fasting over time, although an important line of future work will be comparing the incremental utility of perfectionism versus SC in prediction of fasting.

In contrast, findings for the effect of SC on purging were mixed. This is likely due to differing rates of severe purging behaviors (i.e., self-induced vomiting, laxative misuse) across the two samples. This is not to say that excessive exercise cannot be an extreme method of purging; indeed, it can cause substantial physical and psychological harm. However, I could not clarify the intensity and duration of exercise episodes reported by participants in Studies 1 and 2 (i.e., without substantially altering the EDE-Q). It is thus possible that individuals reported on and characterized as “excessive or compulsive” what others might consider normative levels of exercise. Debate exists on how best to characterize excessive exercise. Some have used 20+ episodes a month as a cutoff, based on one definition offered by Mond, Hay, Rogers, and Owen (2006). However, Mond, Hay, Rogers, Owen, and Beumont (2004) noted considerable variability in frequency and intensity of exercise in different populations. They reported that motivation for exercise and/or feelings of guilt following missed exercise might better delineate excessive exercise as a DE behavior. Unfortunately, the present version of the EDE-Q does not offer such nuanced assessment of this behavior. Modifying items about exercise to more

comprehensively assess respondents' "drive" for the behavior will be an important area of future work.

Within the high-risk sample, SC predicted purging at follow-up, even after controlling for baseline behavior and NSSI. This is consistent with results of EMA studies in which criticism triggered thoughts about purging (and bingeing) and self-hatred significantly predicted binge-purge episodes (Shingleton et al., 2013, Turner, Yiu, Claes, Muehlenkamp, & Chapman, 2016). These results support SC as a potential target for purging behaviors, although several questions still remain regarding the mechanism of the effect of SC on purging. For example, does SC about any self-related topic suffice to predict purging? Or does SC operate through affect and cognition specific to one's weight, body, and/or eating behaviors? Does SC equally predict purging episodes in combination with or isolated from binge eating episodes?

We observed significant effects of SC on bingeing in the undergraduate sample after controlling for baseline behavior and prior to controlling for baseline bingeing in the high-risk sample. SC also accounted for the highest proportion of the association between NSSI and any DE behavior (although the association of NSSI and bingeing itself was small and nonsignificant). Our findings on SC and binge eating are consistent with past work documenting cross-sectional relations of SC and binge eating, along with other constructs such as body dissatisfaction and shame (Duarte, Ferreira, & Pinto-Gouveia, 2016; Duarte, Pinto-Gouveia, & Ferreira, 2014). Additionally, pilot intervention work has demonstrated preliminary effectiveness of self-compassion training on reducing binge eating (Kelly & Carter, 2015). Bingeing was originally conceptualized as serving an affect regulation function (Polivy & Herman, 1993); however, later research has cast doubt on whether bingeing actually serves to reduce negative affect (see reviews by Dingemans, Danner, & Parks, 2017 and Haedt-Matt & Keel, 2011; Leehr et al., 2015

for an opposing view). Findings of the present study support the need for further investigation of SC as a driver of (and potential clinical target for) binge eating. As with the investigation of purging, it will be important to discern whether people binge in response to experiencing general SC or SC related to body/weight/eating-specific concerns. Another relevant question is whether bingeing also serves a self-punishment function, especially given findings that negative affect actually *increases* following binge eating episodes (Haedt-Matt & Keel).

### **Aim 3**

I did not find support in either sample for Muehlenkamp and Claes' (2014) hypothesis that life stress moderates the relation of self-criticism and NSSI/DE. Results did support a main effect of life stress in relation to DE controlling for SC. (In contrast, the DE-SC relations were largely nonsignificant after controlling for stress). This is consistent with past research on the role of stress in these behaviors and strengthens the case for the salience of stressful live events in relation to DE (e.g., Ball & Lee, 2000). For NSSI, however, SC showed significant associations with the behavior in both samples after controlling for stress. Conversely, stress showed a negative association with NSSI in the undergraduate sample and was not significantly related to the behavior in the high-risk group. Past research has generally focused more on simple main effects of stress and NSSI (Liu, Cheek, & Nestor, 2016). The present findings indicate the need for a more nuanced investigation of whether or how stressful experiences relate to psychological processes to promote NSSI. It is also important to note that I used an omnibus measure of normative stress in both Studies 1 and 2. Findings may differ if one were to parse effects by type of stressor (e.g., interpersonal versus academic/occupational stressor vs. financial) or focus on traumatic life events.

Although not an explicit aim of the study, I noted significant longitudinal effects of NSSI on subsequent fasting and bingeing in the undergraduate (but not high-risk) sample. In the high-risk group, purging showed a trending effect on subsequent NSSI (controlling for baseline NSSI and SC). These findings, although offering mixed evidence of reciprocal NSSI-DE relations, are consistent with the complexity described in the existing literature. Turner et al. (2015) found that NSSI significantly predicted subsequent DE symptoms among university undergraduates, however their study used a composite measure of DE attitudes, cognitions, and behaviors. More directly comparable to the present work, Peterson and Fischer (2012) showed longitudinal effects of purging on subsequent NSSI among undergraduates. Past work has shown that switching to binge/purging was predictive of later suicide attempt among patients with AN, hinting at a potential link between purging and direct self-harm (Foulon et al., 2007). Others have conceptualized purging as a means of acquired capability for direct self-harm, consistent with Joiner's (2005) theory of suicide (e.g., Riley, Davis, Combs, Jordan, & Smith, 2016). Riley et al. found significant effects of lifetime NSSI on purging onset (and vice versa) over one year in a sample of university students. They focused specifically on *onset* of purging and NSSI, which could partially account for the discrepancy between their findings and results of Study 1. This small but growing body of literature points to reciprocal relations between NSSI and DE, particularly purging. Findings of the present studies add to our knowledge, but further research on this topic is needed. In contrast, there is a relative dearth of research on reciprocal effects of bingeing and fasting with NSSI, so continuing to study relations among these behaviors in university and clinical samples will be important to confirm the present results. It will also be valuable to examine how interpersonal and social context differentially impacts individuals' risk for engaging in both NSSI and various DE behaviors, as in Turner et al., 2016.

The present work adds to our knowledge of SC as both substrate for and predictor of NSSI and DE. Nevertheless, I acknowledge key limitations that signal directions for future research. The first limitation is my use of an undergraduate sample from a private, Tier 1 research university. Although both NSSI and DE represent substantial concerns in this population, findings may not generalize to other populations or even other university samples (e.g., college counseling center samples). Rates of NSSI and severe DE behaviors were low at follow-up. This is not unexpected given the relatively short follow-up period and low base rates of these behaviors in undergraduate samples more broadly. Continued research on this topic using larger samples of both undergraduate and clinical participants will be critical to confirm the present findings.

A second limitation concerns the recruitment strategy for the high-risk sample (i.e., selecting participants based on lifetime NSSI and DE history). I used this sampling strategy in an effort to address the relatively low endorsement of NSSI and DE associated with the undergraduate sample. I readily acknowledge that there are likely differences between the high-risk sample recruited for Study 2 and other clinical samples. The follow-up rate in Study 2 was also moderate, although I again sought to address this limitation through recruiting additional participants at Wave 2 and using full-information estimation methods in the analyses.

A third major limitation of Studies 1 and 2 was that both analyses relied on self-reported NSSI and DE. Although typical for the field, self-report data have known limitations. Self-report of excessive exercising may be especially problematic, for the reasons described above. Future research would benefit from combining self-report of NSSI and DE with clinical interviews about the behaviors.

A fourth limitation was the relatively brief interval between waves in both Studies 1 and 2. Changes in NSSI in particular may have been greater over a longer-term follow-up period, and SC may have differential relations to changes in NSSI and DE over time. This limitation also reflected a strength, however, given that much of the existing literature (especially on undergraduate samples) focuses on longer-term follow-ups. The one-month and two-month time-lags here thus add to our knowledge of longitudinal effects of SC on NSSI and DE. Future studies would ideally integrate both short-term assessment (even micro-assessment, as in EMA or daily diary studies) with long-term follow-up periods to provide a more comprehensive assessment of each behavior over time.

Finally, the assessment of SC itself also reflected both a limitation and strength of the study. I used multiple measures of the construct, thus decreasing concerns about reliability and validity associated with mono-method bias. However, we focused predominantly on assessment of static, traitlike SC, i.e., general predisposition to engage in SC. This precluded any evaluation of within-person changes in SC, particularly in response to stressors or experience of negative affect. It is unclear whether individuals who engage in these behaviors are more apt to experience SC in these contexts or how such reactive SC relates to NSSI or DE. These are important questions given the integral role of negative affect in relation to both NSSI and DE and paved the way for the third and final study of the dissertation.

## **Chapter VI**

### **STUDY 3**

#### **Summary of Aims**

Study 3 aimed to explore the relation of reactive self-criticism to NSSI and each form of DE (see Study Aims for associated hypotheses).

#### **Participants**

Participants included 80 university students ( $M$  age = 19.33,  $SD$  = 1.31; 76.3% female, 21.3% male, 2.5% transgender) recruited from a mid-sized private university in the southeastern United States. The sample was moderately diverse (57.5% Caucasian, 15% African American, 26.3% Asian or Asian American, 2.5% Hispanic or Latino, 3.8% another race/ethnicity; participants could select more than one option, so percentages do not add to 100). Participants were recruited from a larger longitudinal study on NSSI and DE (see Study 1) and from an introductory psychology class after completing a pre-screening measure of NSSI and DE. Individuals with lifetime histories of these behaviors were oversampled to ensure their adequate representation in the study. Table 16 shows rates of NSSI and DE in the sample. Cutting and self-hitting were the most frequently reported forms of NSSI among those reporting a history of the behavior (endorsed by 60 percent and 62.86 percent of this subgroup, respectively).

#### **Methods**

The study was presented to participants as an effort to learn how “the way that people think about themselves might relate to the kinds of behaviors they may or may not engage in.” Participants were informed that the study involved answering questions about NSSI and DE. After arriving to the lab, participants received a brief orientation to the study procedures and completed baseline measures of self-criticism. They then underwent a three-part negative mood induction. First, they were asked to complete a series of unsolvable anagrams (stimuli adopted from Aspinwall & Richter, 1999). Participants were given two minutes and told that people “typically complete about half” of the word puzzles. The anagram list included four solvable puzzles to enhance the realism of the experience. Participants were then asked to write for five minutes about a recent time in which they felt “particularly distressed or upset.” They were instructed to consider, in particular, the role they played in the event.<sup>19</sup> Somber music (“Russia under the Mongolian Yoke” by Alexander Nevsky) played as the third component of the induction. Participants completed the PANAS before and after the induction as a manipulation check. They completed the SC battery post-mood-induction and participated in clinical interviewing about lifetime NSSI and DE behaviors. Finally, participants were debriefed about the anagram deception and viewed two pleasant animal-related YouTube videos as to ensure mood reversal. The lab sessions were conducted by the primary author or another trained graduate research assistant, who closely monitored the participants for signs of distress. All participants received information about the university counseling center and other campus mental health resources, as well as information about Web-based resources for NSSI or DE. Participants received either course credit or a \$12 Amazon.com gift card in exchange for their

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<sup>19</sup> This instruction was designed to further evoke self-criticism if salient.



time. Procedures were approved by the Vanderbilt University Institutional Review Board.

### **Measures**

**NSSI.** The *Self-Injurious Thoughts and Behavior Interview* (SITBI, Nock et al., 2007) is a structured interview designed to assess the frequency and function of various forms of self-injury. The full measure consists of five modules tapping various aspects of suicidality and NSSI. We administered the NSSI module, which asks about engagement in NSSI over a variety of time frames (i.e., past month, past year, lifetime), specific methods used, perceived functions of the behavior, etc. The measure has shown adequate test-retest reliability and convergent validity with other assessments of NSSI (Nock et al., 2007).

**DE.** *Eating Disorder Examination-Questionnaire-6.0* (EDE-Q, Fairburn, 2008). This 28-question instrument asks about DE behaviors (i.e., binge eating, fasting, and purging via self-induced vomiting, laxative misuse and excessive exercise) and DE-associated cognitions and emotions that an individual may have experienced in the past month. The measure has a well-established factor structure, has been validated among undergraduate men and women, and has acceptable reliability across subscales in both groups (Lavender et al., 2010; Luce et al., 2008). We focused on the DE behavior items only to ensure the most salient comparison to NSSI. Focusing on the DE behavior items (versus the subscales or total score) also safeguarded against artificially inflating the DE-SC correlation due to negative cognitions about one's weight or shape. Test-retest stability of the DE behavior items ranged from moderate to good in a validation study with a follow-up period of approximately two weeks (Luce & Crowther, 1999). As expected, stability of the behavior items was much poorer over a 10-month follow-up period (Bardone-Cone & Boyd, 2007).

*Structured Clinical Interview for DSM Disorders-Research Version, Eating Disorders* module (SCID, First, Spitzer, Gibbon, & Williams, 2002). The SCID was used to assess lifetime DE history<sup>20</sup>. The SCID-IV-R is a widely used, well-validated clinical interview employed in psychopathology assessment. This module version contains detailed questions for all three types of DE of interest (restriction, purging, and bingeing) within the context of assessment for anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED). I omitted skip rules and applied diagnostic criteria as detailed in DSM-5 to ensure comprehensive assessment of DE behaviors (see Keel, Brown, Holm-Denoma, & Bodell, 2011, for details about this approach). The most notable change in DE diagnostic criteria from DSM-IV to DSM-5 was the inclusion of binge eating disorder as a formal diagnosis (versus being subsumed under Eating Disorder-Not Otherwise Specified). Other changes, such as reducing the required frequency of binge/purge episodes in the diagnosis of bulimia nervosa from 3 times a week to 1 time a week, resulted in occasional re-classification of individuals, as noted in Keel et al. Questions about rating specific symptoms were resolved by consensus within the research team.

**SC. *Self-Rating Scale*** (SRS; Hooley et al., 2002). The SRS is an eight-item measure of self-criticism that asks people to rate their agreement with statements such as “others are justified in criticizing me.” The measure has been used in a community sample, where it distinguished between self-injurers and non-self-injurers (St. Germain & Hooley, 2012). In the present sample, Cronbach’s  $\alpha$  was 0.85. For the SRS and all other self-report SC measures, respondents were asked to rate their general agreement with the items during baseline assessment. After the mood induction, they were asked to respond to the item prompts based on how they felt “right now.”

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<sup>20</sup> This facilitated comparisons with lifetime NSSI, although we note the limitations of this comparison, as full DE diagnoses encompass both affective and cognitive components in addition to behaviors.

*Depressive Experiences Questionnaire-Short Form* (DEQ-SF; Rudich, Lerman, Gurevich, Weksler, & Shahar, 2008). The DEQ-SF is based on the Depressive Experiences Questionnaire (Blatt, Quinlan, & D'Afflitti, 1976), a 66-item measure designed to assess depressive experiences stemming from concern about rejection from others (dependency) or one's own self-criticism. In creating the DEQ-SF, Rudich et al. selected items with high face validity for self-criticism and whose wording did not contain references to mood or affect (thus avoiding a potential confound with these domains). An example item is "Often I find that I do not live according to my standards or ideals." The final six-item scale showed excellent convergence with the original scale and acceptable internal consistency (Rudich et al.) The DEQ-SF has been used in both medically complex and undergraduate populations (e.g., Zuroff, Sadikaj, Kelly, & Leybman, 2016). The measure showed acceptable internal consistency in the present sample (Cronbach's  $\alpha = .76$ ).

*Automatic Thoughts Questionnaire* (ATQ; Hollon & Kendall, 1980). The ATQ is a 30-item scale consisting of negative cognitions associated with depressive symptoms. Respondents rate their agreement with each of the cognitions on a 1 ("Not at all") to 5 ("All the time") Likert scale. The scale has shown both adequate internal consistency and convergent validity with negative cognitions associated with depression in both undergraduate and clinical samples (Harrell & Ryon, 1983; Hollon & Kendall). Factor analysis in the original validation sample supported a four-factor solution reflecting (1) "personal maladjustment and desire for change," (2) "negative self-concept/expectations," (3) "low self-esteem," and (4) "giving up/hopelessness." For the present study, we selected nine items from Factors 1-3 with high face validity and relevance to self-criticism (Example: "I hate myself.") Internal consistency of this self-criticism subscale was excellent (Cronbach's  $\alpha = .90$ ).

*Affect Misattribution Procedure* (AMP-Self, AMP-Body; Payne, Cheng, Govorun, & Stewart, 2005). The AMP is a computer-based task designed to tap implicit affect and has been used in assessments of disparate topics including NSSI (Franklin, Lee, Puzia, & Prinstein, 2014; Franklin, Puzia, Lee, & Prinstein, 2014). Individuals briefly view an affective stimulus followed by a blank screen and finally an ambiguous prompt (e.g., a Chinese pictograph). Respondents are asked to rate whether the pictograph is more pleasant or more unpleasant than the average pictograph. Before the task, they are told to ignore any affect provoked by the initial stimuli. Previous research indicates that, despite these admonitions, affective associations with the initial stimuli strongly impact participants' ratings of the neutral prompt. In the proposed study, the affective stimuli will consist of six different self-focused words and short phrases (AMP-Self; e.g., "me," "myself," "I," "my own,") and six different body-focused words and short phrases (AMP-Body; e.g., "my body," "my weight," "my shape"). These will be interspersed with six neutral, six positive, and six negative terms (as characterized by the Affective Norms for English Words; Bradley & Lang, 1999). Scores are based on the proportion of positive evaluations of the target prompt. Lower scores for self-focused stimuli, for example, are thus thought to be reflective of greater SC. In the present sample, AMP-Self scores showed small correlations in the expected direction with the self-report SC measures; Pearson's  $r$ s range from  $-.13$  (n.s.) to  $-.25$  ( $p = .03$ ). This is consistent with past research on the correlation of implicit and explicit assessments (Hoffman, Gawronski, Gschwendner, Le, & Schmitt, 2005.) Notably, however, correlations of the explicit (i.e., self-report) SC measures were larger with the AMP-Body scores (Pearson's  $r$ s =  $-.30$  to  $-.44$ ,  $p$ s  $< .05$ ; a similar pattern was apparent in Study 1.

## Data Preparation and Analytic Approach

Confirmatory factor analysis suggested that all three self-report SC measures loaded strongly onto a single latent SC factor ( $\lambda_s > .70$ ).<sup>21</sup> Therefore, I combined them at pre-test by standardizing each measure and summing the  $z$ -scores. This  $z$ -score reflected explicit measurement of trait-like SC. I elected to retain the AMP-Self and AMP-Body variables in separate analyses given the slight difference in focus of their stimuli. Taken together, pre-test values for the AMP-Self, and AMP-Body variables reflected implicit measures of trait-like SC. I conceptualized reactive SC as the difference from pre-test to post-test divided by the respondent's change in negative affect from the mood induction, thus accounting for individual differences in change in emotional reactivity (see Cole et al., under review). Equation 1 shows the formula used to calculate reactive SC. Note that I added 0.5 to the denominator to avoid any circumstance in which I would have divided the numerator by 0.

$$(\text{PostSC} - \text{PreSC}) / [(\text{PANAS Negative Post-test} - \text{PANAS Negative Pre-test}) + 0.5] \quad (1)$$

I standardized post-test values for all of the explicit measures on their respective pre-test means and *SDs* in order to detect change after the mood induction. Due to item-coding, higher scores based on the difference of explicit measures corresponded to increased reactive SC whereas lower scores on the difference of implicit measures indicated higher reactive SC.

The literature reflects a debate over the optimal way to analyze pre-test/post-test data, specifically whether to evaluate the post-test scores via an ANCOVA model with pre-test scores as a covariate or to analyze change scores. Van Breukelen (2006, 2013) argued that ANCOVA may produce biased results in the absence of random assignment to baseline treatment and/or

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<sup>21</sup> The model was just-identified thus fit indices are non-interpretable.

existence of “true” subgroups at baseline. As I anticipated baseline differences in SC by both NSSI and DE status, I elected to analyze the change scores for the self-report SC composite, AMP-Self, and AMP-Body scores (adjusting for changes in negative affect as described above). I then assessed the relation of these reactive SC scores with (1) NSSI (lifetime history, lifetime frequency, past-year history) and (2) DE (past-month binge eating, purging, fasting and restriction, based on the EDE-Q, and lifetime anorexia nervosa, bulimia nervosa, and binge eating disorder diagnoses, based on the SCID). Owing to the directional nature of the hypotheses regarding reactive SC, I used one-tailed tests with  $\alpha = .05$ .

I conducted several steps to prepare the EDE-Q data for analyses. First, I identified four items that reflected restriction behaviors (see Appendix C for specific items). Response choices for these behaviors refer to a range of days during which the respondent engaged in that particular behavior (e.g., “0 days,” “1-5 days,” etc.). I calculated the mean number of days in each range and coded responses accordingly (Note: Participants reporting “0 days” retained a score of 0). I then summed responses to obtain a count of restrictive behaviors. Because this restriction score encompassed behaviors that could be interpreted as mild (e.g., excluding favored food items, attempting to limit intake) and more severe behaviors (e.g., fasting), we also analyzed responses to the fasting item alone, as the most extreme example of restriction. I created a composite purging measure by summing episode counts of laxative misuse, self-induced vomiting, and excessive exercise.<sup>22</sup> Bingeing was represented by number of days on which the participant reported eating an unusually large amount of food while experiencing a sense of loss of control (corresponding to the DSM-5 definition of an “objective binge episode”

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<sup>22</sup> The choice to combine across purging items was motivated both by low rates of laxative misuse and self-induced vomiting and because all three behaviors are considered forms of “inappropriate compensatory behaviors” in DSM-5.

that encompasses both quantity of food consumed and loss of control). Finally, I summed scores for fasting, purging, and bingeing to create a composite DE score.

## Results

Table 17 shows the association of pre-test and post-test SC with all NSSI and DE variables.<sup>23</sup> The table shows SC based both on explicit assessment (i.e., the sum of *z*-scores for all three self-report measures) and implicit measurement (i.e., self-focused and body-focused stimuli on the AMP). At baseline, lifetime and past-year NSSI status (i.e., whether the participant reported engagement in the behavior in that timeframe) showed large and significant associations with SC based on explicit measures. Associations with either form of implicit assessment were not significant. Lifetime and past-year NSSI frequency showed small but significant associations with SC as assessed by both explicit and implicit methods. The DE composite showed medium-to-large, significant correlations with both explicit and implicit SC measures at baseline. Correlations of specific DE behaviors with any SC measure reflected small to medium effect sizes (all significant). Among lifetime DE diagnoses, only AN and BN showed significant associations with SC at baseline (and only via implicit assessment using body-focused stimuli).

Pre-test/post-test comparisons confirmed significant increases in negative affect (and decreases in positive affect) following the mood induction (Negative affect:  $t(79) = 6.45, p < .001, 95\% \text{ CI } [3.07, 5.81]$ ; Positive affect:  $t(78) = -7.81, p < .001, 95\% \text{ CI } [-7.80, -4.63]$ ). Interestingly, negative affect reactivity was negatively correlated with lifetime and past-year NSSI history ( $r = -.05$  &  $-.10$ , for lifetime and past-year frequency, respectively,  $ps < .05$ ). SC

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<sup>23</sup> See Appendix A for correlations based on Spearman's  $\rho$ .

scores increased across the full sample following the mood induction on both explicit and implicit assessments (Explicit SC:  $t(79) = 4.04, p < .001, 95\% \text{ CI } [0.31, 0.91]$ ; AMP-Self:  $t(76) = -2.36, p = .02, 95\% \text{ CI } [-0.14, -.01]$ ; AMP-Body:  $t(76) = -2.98, p < .01, 95\% \text{ CI } [-0.15, -0.03]$ ). Again, scores on the AMP are reverse-coded, thus negative values for the difference indicated increased SC following the mood induction.

Associations between post-test SC and NSSI largely mirrored the pre-test relations (although the correlations dropped slightly, see Table 17). There were a few noteworthy exceptions, all concerning the implicit assessment methods. For example, lifetime NSSI frequency was significantly related to scores on the self-focused AMP stimuli at baseline but not following the mood induction. Lifetime and past-year NSSI frequency were also positively and significantly associated with scores on the body-focused stimuli after the mood induction (i.e., more frequent NSSI was related to more positive implicit associations with the body).

Among the DE measures, purging and restriction no longer showed significant associations with the explicit SC measure post-mood induction. Associations with the self-focused AMP stimuli were also largely nonsignificant after the induction (although see findings for bingeing, Table 17). Bingeing and lifetime diagnoses of AN and BN were each no longer significantly associated with body-focused AMP scores following the mood induction.

Table 18 shows the relation of reactive SC (as measured by explicit assessment and implicit assessment) with each NSSI and DE variable. Lifetime BN diagnosis was associated with increased reactivity to the body-focused stimuli (the negative value reflects the scoring of the instrument); all other associations between NSSI and DE variables and reactive SC were nonsignificant based on one-tailed tests ( $\alpha = .05$ ).



## Discussion

The aim of this study was to explore the association of NSSI and DE to SC experienced in response to negative affect (i.e., reactive SC). Two main findings emerged. First, contrary to hypothesis, neither lifetime nor past-year NSSI was significantly associated with increased reactive SC. Second, increased reactive SC was not significantly related to any recent DE behavior. Discussion of these findings continues below.

The first main finding is noteworthy given the conceptualization of NSSI as a (maladaptive) means of affect regulation. To my knowledge, this is the first study to examine the relation of NSSI to changes in SC in response to experimentally manipulated affect. Results of the present study suggest that changes in negative affect may not differentially potentiate self-critical cognitions among those with histories of NSSI.<sup>24</sup> This is not to say there is no association of SC with NSSI. On the contrary, I saw strong, significant associations of each SC assessment method with NSSI history (lifetime and past-year). These findings, coupled with evidence of an overall increase in SC across the participants, suggests a scenario in which “the poor get [even] poorer.”

Findings were generally consistent using explicit (i.e., self-report) and implicit measures of SC, along with an implicit measure of SC focused specifically on the body. This last measure offered one notable exception to the pattern of results: Prior to the mood induction, NSSI frequency was related to more negative views of the body. After the mood induction, NSSI was related to more positive (implicit) associations with the body. One possible explanation for this is that individuals who engage in NSSI to reduce negative affect may reflect on their bodies as a

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<sup>24</sup> Or that any such effects were small. I was sufficiently powered (>.80) to detect medium-sized effects for the point-biserial correlations and Pearson correlation between reactive SC scores and any lifetime NSSI history and any past-year NSSI history (respectively). I thus draw no conclusions regarding the possibility of a small effect. See discussion of limitations.

potential source of emotional release during periods of distress. Gratz, Tull, Dixon-Gordon, Turner, and Chapman (2018) found no difference between self-harm related expectancies in neutral versus negative emotional states for participants with lifetime NSSI histories. This work did not examine thoughts about the body, however. It will be important to explore further how individuals who engage in NSSI view their bodies across different mood states.

Previous work has found significant associations of NSSI with baseline SC and negative affect reactivity (Fox, Toole, Franklin, & Hooley, 2017). Differences in their sample and methods preclude direct comparisons to my own results (e.g., participants included only those with recent NSSI histories, SC was assessed using a single measure, the procedure was in the context of an investigation of mood and pain in NSSI).

Although negative emotion reactivity was not the main focus of this study, I nevertheless observed an association between NSSI history and reduced affective change. This is in contrast to results by Arbuthnott, Lewis, and Bailey (2015), who used similar induction procedures as the one used in the present study. The discrepancy calls to mind questions in the literature regarding emotion reactivity in depression (i.e., enhanced reactivity to negative stimuli versus blunted emotional responses to both positive and negative prompts; Rottenberg, Gross & Gotlib, 2005). Future research should continue to explore factors related to emotion reactivity and NSSI, particularly given conflicting findings of self-report and physiological emotion reactivity among those reporting the behavior (Glenn, Blumenthal, Klonsky, & Hajcak, 2011). Also important is to investigate how other psychological processes (e.g., depressive symptoms, deficits in emotion regulation) and the self-reported function of the behavior interact with SC in relation to NSSI.

The second major finding concerned the role of reactive SC and DE. Despite theoretical emphasis on the role of negative beliefs about the self in DE, laboratory-based protocols in the

DE literature have largely focused on attentional bias for and reactivity to food- and weight-related stimuli (e.g., Brooks, Prince, Stahl, Campbell, & Treasure, 2011; Dobson & Dozois, 2004; Fairburn, Cooper, & Shafran, 2003; Johansson, Ghaderi, & Andersson, 2005; Vitousek & Hollon, 1990). More recent efforts have investigated general beliefs about the self and DE via laboratory-based methods or as a therapeutic target (e.g., Pinto-Gouveia et al., 2016; Pringle, Harmer, & Cooper, 2010; Yiend, Parnes, Shepherd, Roche, & Cooper, 2014). The current study extends this work by examining SC in the context of experimentally-induced affect. I observed significant relations between multiple forms of DE and SC at baseline. Using implicit measures, I saw significant relations between DE and negative views toward both the self and the body. This supports arguments that both negative beliefs about the self and specific criticism of one's body play a role in DE (Fairburn, Cooper, & Shafran, 2003). These findings also reflect the often-reported SC-DE association in the literature (e.g., Yiend et al., 2014; Zelkowitz & Cole, 2018).

Following the mood induction, however, only the DE composite, bingeing and fasting remained associated with self-reported SC. Bingeing was also notable as the only DE behavior that showed an association with negative implicit views toward the self (considered more broadly) following the mood induction. Unlike the other DE behaviors (i.e., purging, restriction, fasting), bingeing was not associated with negative implicit views toward the body following the induction. This suggests a potentially unique role for SC in binge eating, at least in the context of heightened negative affect. Further research is required to confirm this effect, however, and to investigate its possible mechanisms.

Relations of reactive SC to DE behaviors were largely nonsignificant. This null result does not suggest that SC is unrelated to DE. Rather, it suggests that DE behaviors were not

associated with increased *change* in SC following a negative mood induction. One notable exception, however, was the association of lifetime BN diagnosis and increased reactive SC based on implicit assessment using body-focused stimuli. This may be due to the fact that the diagnostic criteria for BN explicitly includes a tendency for self-evaluation based on shape and weight. The nonsignificant association of actual purging with implicit SC based on body-focused stimuli provides further evidence that this cognitive aspect of the BN diagnosis may have driven the result. Additional research using clinical samples will be required to confirm the finding and this interpretation, however.

Although the present work offers a unique contribution to the literature on SC, NSSI, and DE, certain limitations deserve mention. The first concerned the size and nature of the present sample. The sample size ensured sufficient power to detect medium-sized effects. A larger sample size would have enabled me to detect small effects regarding reactive SC. The sample consisted of undergraduates, potentially compromising generalization to more clinical samples. Although I endeavored to oversample individuals who reported lifetime experience with NSSI or DE, rates of recent engagement in these behaviors were still relatively low. Future research on reactive SC (and SC more generally) should include participants with a range of clinical severity for both NSSI and DE. It would also be beneficial to gather data on depressive symptoms and other psychopathology to control for their effects in relation to reactive SC.

A second major limitation concerned the affect induction itself. The three-part induction procedure produced large changes in both negative and positive affect. The strength of the induction may have inadvertently obscured differences in reactive SC to weaker stimuli. In other words, the induction may have been so strong that all individuals responded with increased SC, thus masking any variability associated with NSSI or DE. Lending support to this argument,

Cole et al. (2014) found stronger associations between depressive symptoms and cognitive reactivity for stimuli of medium intensity (versus especially strong negative stimuli). Further research should explore reactive SC in response to mildly and moderately intense affect induction procedures. I note as well that while I strove to enhance the ecological validity of the induction by having participants write about a personal experience, the procedure undoubtedly retained some element of artificiality. It would be valuable to administer both lab-based paradigms such as this one and ecological momentary assessments to the same participants to examine reactive SC in both controlled settings and their real-world context.

Finally, my assessment of reactive SC itself carried limitations. Participants were instructed to distinguish between their tendencies to self-criticize in general (at the baseline assessment) versus “right now” (post-induction). Responses to the post-induction measures may have been influenced by answers provided at baseline. Ideally, participants would complete multiple forms of the same instrument to mitigate such influence or complete the baseline measures on another day prior to the mood induction to prevent these carryover effects. Despite these limitations, this study offers new insights into the role of SC in both NSSI and DE. If confirmed through future research, findings suggest a more salient role for global (trait-like) SC in both behaviors than reactive SC.

## Chapter VII

### GENERAL DISCUSSION

Individuals who engage in one form of self-harm are more likely to perform other such behaviors, either concurrently or at other points in their lives. This pattern of comorbidity among self-harm behaviors has lead researchers and clinicians to seek transdiagnostic processes in the etiology and maintenance of multiple forms of self-harm so as to guide prevention and intervention efforts. In this dissertation, I aimed to investigate one such factor, self-criticism, in an effort to elucidate its role in two pernicious and prevalent behaviors: nonsuicidal self-injury (NSSI) and disordered eating (DE).

Results from three studies showed correlations among SC, NSSI, and DE, although the proportion of the correlation between NSSI and DE accounted for by SC varied by specific DE behavior. Two studies specifically tested the longitudinal effects of SC, providing one of the first direct tests of etiological theories of NSSI and DE as set forth by Claes and Muehlenkamp (2014) and Svirko and Hawton (2007). Findings consistently revealed longitudinal effects of SC on DE behaviors (although the exact relations varied by behavior) and mixed support for the longitudinal effect of SC on NSSI. Results of the third study clarified that although the general tendency to engage in SC is strongly associated with NSSI and DE, the behaviors are not associated with increased *change* in SC in response to negative affect. Taken together, these findings largely support a role for global SC in both NSSI and DE, although further research is necessary to confirm its utility as a predictor of these behaviors over the short- and long-term.

Currently available treatments for both behaviors show mixed effectiveness, although support for the effectiveness of cognitive behavior therapy in bulimia nervosa and binge eating is slightly more pronounced (e.g., Franklin et al., 2016; Hawton et al., 2016; Hay, Bacaltchuk,

Stefano, & Kashyap, 2009; Hay, Claudino, Touyz, & Elbaky, 2015; Turner, Austin, & Chapman, 2014). If confirmed through future research, the present findings suggest SC may be a fruitful target for interventions for NSSI and DE.

There is an intuitive connection between an increased tendency to attack oneself mentally (or verbally) and to engage in physical acts of self-harm. The present set of results are important in that they provide initial empirical support for this notion. Findings also show that this process may play a role in both direct and indirect methods of self-harm. Substantial work remains to understand potential mechanisms underlying this effect and whether population characteristics moderate the relation of SC to direct or indirect forms of self-harm.

Two questions are particularly crucial. First, does targeting SC produce meaningful decreases in both NSSI and DE engagement? Previous work has suggested that increasing self-worth or self-compassion may reduce willingness to endure pain among participants with NSSI histories and may lead to decreases in bingeing when presented as part of an overall intervention program (Hooley & St. Germain, 2014; Pinto-Gouveia, et al., 2018). Although these findings are promising, substantial more research is required to establish SC as an effective therapeutic target in treatment of NSSI and DE behaviors more broadly.

A second critical question is whether and how premorbid SC produces vulnerability to NSSI and DE. Average age of onset for both sets of behaviors is in mid- to late-adolescence (e.g., Heath, Toste, Nedcheva, & Charlebois, 2008; Hudson, Hirpi, Pope, & Kessler, 2007; Nock, 2010). As the current studies did not to test SC as a predictor of NSSI and DE onset, future research should assess SC in early adolescence (prior to typical age of onset for NSSI and DE) to track its utility as a predictor of these devastating behaviors. Increasing our capacity to

identify those at risk of NSSI and DE, prevent their onset or at least intervene at an early stage will be crucial to alleviating the morbidity and mortality associated with these behaviors.

Acts of self-harm are increasingly prevalent. If left untreated, these behaviors can be deadly. Efforts to understand SC and other psychological processes that underlie both direct and indirect self-harm behaviors will be critical to reduce suffering associated with these acts and, ultimately, save lives.



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## TABLES

Table 1.

*Schematic of Aims By Study*

Study Name	Sample Type (n)	Aims Addressed
Study 1	University students (251)	Aims 1, 2, 3
Study 2	High-risk young adults (517)	Aims 1, 2, 3
Study 3	University students (80)	Aim 4

Table 2.

*Self-Reported Baseline NSSI, DE Among Study 1 Participants*

Behavior	% Sample Endorsing Behavior	M (SD) <sup>a</sup> /Range	Skewness	Kurtosis
Past Month				
NSSI	2.5	0.04 (0.26)/0-3	8.58	82.48
Restriction	72.8	32.45 (29.58)/0-112	0.71	-0.39
Fasting	22.8	3.34 (6.43)/0-28	1.69	1.81
Bingeing	38.5	1.79 (3.95)/0-28	3.95	19.27
Purging	30.6	2.45 (5.39)/0-28	2.83	8.21
Past Year				
NSSI	8.2	0.44 (2.34)/0-30	9.37	108.03
Fasting	23.4	3.71 (11.64)/0-81 <sup>b</sup>	4.68	24.42
Bingeing	27.4	4.08 (15.21)/0-170	7.61	70.86
Purging	36.8	18.21 (53.45)/0-361 <sup>b</sup>	4.54	22.52
Lifetime				



NSSI	15.9	1.91 (7.44)/0-60	5.01 (0.16)	27.33 (0.31)
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*Note.* NSSI = Nonsuicidal self-injury, DE = Disordered eating.

<sup>a</sup> Values based on full sample

<sup>b</sup> Reflects outlier adjustment

Table 3.

*Correlations of SC with Each NSSI and DE, Pearson's r with robust standard errors (Waves 1 and 2)*

Behavior	W1 Explicit SC	W1 AMP-Self	W1 AMP-Body
1. W1 NSSI	.21**	-.05	-.05
2. W1 DE Composite	.22**	-.07	-.06
3. W1 Binge	.04	.02	.04
4. W1 Purge	.07	-.03	-.02
5. W1 Restrict	.18**	-.07	-.11
6. W1 Fast	.28**	-.10	-.10
Wave 2 Correlations			
Outcome	W2 Explicit SC	W2 AMP-Self	W2 AMP-Body
1. W2 NSSI	.39**	-.09	-.18**
2. W2 DE Composite	.33**	-.18*	-.37**
3. W2 Binge	.16*	-.09	-.15
4. W2 Purge	.14*	-.14*	-.24**
5. W2 Restrict	.20**	-.03	-.27**
6. W2 Fast	.36**	-.14	-.37**

*Note.* AMP = Affect Misattribution Procedure (self-focused and body-focused stimuli); NSSI = Nonsuicidal self-injury; DE Composite = Disordered eating, composite of bingeing, purging, and fasting; SC = Self-criticism.

\*\*  $p < .01$

Table 4.

*Zero-order and Partial Correlations Among Wave 1 NSSI and DE Variables (controlling for self-criticism), Study 1 (Pearson's  $r$ , robust standard error)*

DE Variable	Zero-order		Partial Correlation (controlling for Wave 1 SC)		% of Zero-order Correlation Accounted for by SC
	$r$	p-value	$r$	p-value	
2. W1 DE Composite	.15	.03	.11	ns	26.67
3. W1 Binge Wave 1	-.07	<.01	-.09	<.01	n/a <sup>1</sup>
4. W1 Purge Wave 1	.13	<.05	.12	<.05	7.69
5. W1 Restriction Wave 1	.062	ns	.057	ns	8.06
6. W1 Fasting Wave 1	.17	.09	.124	.08	29.41

*Note.* NSSI = Nonsuicidal self-injury; DE Composite = Disordered eating, composite of bingeing, purging, and fasting; SC = Self-criticism.

<sup>1</sup>Not calculated due to increase in correlation after controlling for SC.

Table 5.

*Study 1: Longitudinal Effect of Self-Reported Self-Criticism on NSSI, DE*

Dependent Variable	Unstandardized Path ( <i>SE</i> )	Standardized Path	<i>z</i>	95% CI
Model 1: Not Controlling for Baseline Behavior				
NSSI	0.12 (0.04)	0.33	2.79*	[0.05, ∞]
DE Composite	3.10 (0.71)	0.31	4.38**	[1.94, ∞]
Binge	0.55 (0.24)	0.17	2.25*	[0.15, ∞]
Purge	0.62 (0.34)	0.14	1.83*	[0.06, ∞]
Restriction	4.71 (2.13)	0.16	2.20*	[1.19, ∞]
Fasting	1.96 (0.40)	0.33	4.89**	[1.30, ∞]
Model 2: Controlling for Baseline NSSI or DE				
NSSI	0.10 (0.04)	0.27	2.38**	[0.03, ∞]
DE Composite	1.66 (0.62)	0.17	2.69**	[0.64, ∞]
Binge	0.56 (0.22)	0.17	2.58**	[0.20, ∞]
Purge	0.29 (0.29)	0.07	1.00	[-0.19, ∞]

Restriction	0.79 (1.87)	0.03	0.42	[-2.29, ∞]
Fasting	1.08 (0.38)	0.18	2.82**	[0.45, ∞]

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Model 3: Controlling for Baseline NSSI and DE

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NSSI	[0.10 – 0.11] [0.04 – 0.05]	[.27 - 0.28]	2.27 – 2.38*	[0.03, ∞]
DE Composite	1.66 (0.64)	0.17	2.61*	[0.61, ∞]
Binge	0.62 (0.23)	0.18	2.73*	[0.25, ∞]
Purge	0.35 (0.29)	0.08	1.19	[-0.13, ∞]
Restriction	0.64 (1.91)	0.02	0.34	[-2.49, ∞]
Fasting	0.97 (0.39)	0.16	2.52*	[0.34, ∞]

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*Note.* NSSI = Nonsuicidal self-injury, DE Composite = Disordered eating, composite of bingeing, purging, and fasting

<sup>a</sup>Model controls for SC and baseline value of dependent variable

\*  $p < .05$ , one-tailed, \*\*  $p < .01$ , one-tailed

Table 6.

*Study 1: Longitudinal Effects of NSSI on DE (Controlling for Baseline DE Behavior and SC)*

DE Variable (Dependent)	Unstandardized Path (SE)	Standardized Path	<i>z</i> (Unstd. Path)	95% CI
DE Composite	0.07 (2.17)	0.00	0.03	[-4.18, 4.32]
Binge	-1.30 (0.45)	-0.08	-2.28*	[-2.17, -0.42]
Purge	-1.35 (1.47)	-0.06	-0.92	[-4.24, 1.54]
Restriction	3.75 (7.05)	0.00	0.53	[-10.07, 17.56]
Fasting	3.21 (1.21)	0.11	2.65**	[0.84, 5.58]

*Note.* NSSI = Nonsuicidal self-injury, DE Composite = Disordered eating, composite of bingeing, purging, and fasting

\*  $p < .05$ , two-tailed, \*\*  $p < .01$ , two-tailed

Table 7.

*Study 1: Longitudinal Effects of DE on NSSI (Controlling for Baseline NSSI and SC)*

Predictor: DE Variable	Unstandardized Path (SE)	Standardized Path	<i>z</i> (Unstd. Path)	95% CI
DE Composite	-0.001 (0.002)	-0.04	-0.72	[-0.01, 0.00]
Binge	-0.003 (0.003)	-0.03	-0.78	[-0.01, 0.00]
Purge	-0.00 (0.04)	-0.00	-0.33	[-0.01, 0.01]
Restriction	0.00 (0.00)	0.00	0.05	[-0.00, 0.00]
Fasting	-0.003 (0.004)	-0.05	-0.66	[-0.01, 0.01]

*Note.* NSSI = Nonsuicidal self-injury, DE Composite = Disordered eating, composite of bingeing, purging, and fasting

Table 8.

*Study 1: Longitudinal Effect of Self-Criticism on NSSI, DE, Controlling for Baseline Behavior, Negative Affect, Neuroticism*

Dependent Variable	Unstandardized Path ( <i>SE</i> )	Standardized Path	<i>z</i>	95% CI
Model 4: Controlling for Baseline NSSI and DE and Negative Affect				
NSSI	[0.06 – 0.07] ([0.03 – 0.04])	[0.17 - 0.19]	[1.75 – 1.91]*	[0.01, ∞]
DE Composite	1.60 (0.67)	0.16	2.39**	[0.50, ∞]
Binge	0.67 (0.30)	0.20	2.27*	[0.19, ∞]
Purge	0.29 (0.37)	0.07	0.79	[-0.31, ∞]
Restriction	-0.33 (1.98)	-0.01	-0.17	[-3.60, ∞]
Fasting	0.87 (0.45)	0.15	1.92*	[0.12, ∞]
Model 5: Controlling for Baseline NSSI and DE and Neuroticism				
NSSI	.10 -0.11 (.05)	.27- 0.30	2.04-2.18	[0.02, ∞]
DE Composite	1.62 (0.72)	0.16	2.26*	[0.44, ∞]
Binge	0.52 (0.28)	0.15	1.87*	[0.06, ∞]
Purge	0.33 (0.34)	0.07	0.97	[-0.05, ∞]



Restriction	0.91 (2.61)	0.03	0.35	[-3.38, ∞]
Fasting	1.10 (0.51)	0.18	2.17*	[0.27, ∞]

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*Note.* DE = Disordered eating (composite is the sum of binge eating days, purging episodes, and fasting days); NSSI = Nonsuicidal self-injury  
 \* $p < .05$ , one-tailed.

Table 9.

*Study 2: Self-Reported Baseline NSSI, DE*

Behavior	% Sample Endorsing Behavior	M ( <i>SD</i> )/Range	Skewness	Kurtosis
Past Month (Initial Sample)				
NSSI <sup>a</sup>	40.72	9.15 (61.40)/0-1010	14.68	234.19
DE Composite	83.06	15.72 (18.80)/0-94	1.80	3.32
Bingeing	65.80	3.97 (5.67)/0-30	2.32	6.08
BULIT Binge	n/a	2.44(0.99)/1-4.88	0.23	-1.15
Purging	55.70	6.68 (11.73)/0-66	2.72	8.22
BULIT Purge	n/a	1.96 (0.84)/1-4.5	0.77	-0.13
Restriction	85.67	37.53 (31.18)/0-112	0.46	-0.97
DEBQ Restriction	n/a	3.09 (1.00)/1-5	-0.20	-0.69
Fasting	56.68	5.10 (7.21)/0-28	1.60	1.69
Past Month (Replacement Participants)				
NSSI <sup>†</sup>	36.19	7.00 (35.54)/0-351	8.85	83.14

DE Composite	80.48	14.61 (18.27)/0-119	2.30	7.11
EDE-Q Bingeing	64.29	4.09 (5.81)/0-28	2.23	5.41
BULIT Binge	n/a	2.54 (1.09)/1-5	0.22	-1.08
EDE-Q Purging	50.00	6.67 (12.97)/0-98	3.32	14.80
BULIT Purge	n/a	1.94 (0.87)/1-5	0.87	0.17
EDE-Q Restriction	85.24	30.22 (28.64)/0-112	0.82	-0.35
DEBQ Restriction	n/a	2.90 (1.00)/1-5	-0.01	-0.43
Fasting	53.81	3.93 (5.91)/0-28	2.02	3.83

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*Note.* Bingeing, restriction, and fasting counts reflect number of days on which the behavior occurred. NSSI and purging reflect number of episodes. DE composite reflects composite of bingeing, all purging behaviors (“compulsive” exercise), and fasting.

<sup>a</sup> Presented as raw data. See text for *M* (*SD*) and skewness for binned NSSI values. All analyses were conducted using binned NSSI data.

Table 10.

*Study 2: Correlations of SC with NSSI, DE, By Wave*

Behavior	W1 Explicit SC
1. W1 NSSI	.20**
2. W1 DE Composite	.25**
3. W1 Binge	.23*
4. W1 Purge	.13*
5. W1 Restrict	.21**
6. W1 Fast	.25**
Behavior	W2 Explicit SC
1. W2 NSSI	.17**
2. W2 DE Composite	.27**
3. W2 Binge	.20**
4. W2 Purge	.19**
5. W2 Restrict	.21**
6. W2 Fast	.23**

*Note.* All estimates calculated as Pearson's  $r$  with robust standard errors.

\*  $p < .05$ , one-tailed, \*\*  $p < .01$ , one-tailed

Table 11.

*Study 2. Zero-order and partial correlation, parametric, robust standard error estimation*

DE Variable	Zero-order		Partial Correlation (controlling for Wave 1 SC)		% of Zero-order Correlation Accounted for by SC
	<i>r</i>	p-value	<i>r</i>	p-value	
DE All	.13	.04	.08	.17	38.46
W1 Binge	.06	.24	.02	.76	66.67
W1 Purge	.09	.17	.07	.31	22.22
W1 Restriction	.10	.08	.07	.25	30.00
W1 Fasting	.14	.02	.09	.12	35.71

*Note.* Based on  $n = 307$ . P-values based on  $\alpha = .05$ , two-tailed.

Table 12.

*Study 2: Longitudinal Effect of Self-Criticism on NSSI, DE (n = 517)*

Dependent Variable	Unstandardized Path (SE)	Standardized Path	<i>z</i> (Unstd. Path)	95% CI
Model 1: Not Controlling for Baseline Behavior				
NSSI	[0.19 – 0.29] ([0.25 – 0.27])	[0.06 – 0.09]	[0.81 – 1.12]	[-0.21, ∞]
DE Composite	6.40 (1.38)	0.34	4.62**	[4.12, ∞]
Binge	1.25 (0.56)	0.20	2.25*	[0.33, ∞]
Purge	3.38 (0.95)	0.26	3.57**	[0.16, ∞]
Restriction	6.25 (2.32)	0.20	2.69**	[2.43, ∞]
Fasting	1.58 (0.46)	0.23	3.45**	[0.82, ∞]
Model 2: Controlling for Baseline NSSI or DE <sup>a</sup>				
NSSI	0.12 (0.27)	0.04	0.45	[-0.32, ∞]
DE Composite	3.85 (1.29)	0.20	2.99**	[1.73, ∞]
Binge	0.46 (0.50)	0.07	0.92	[-0.36, ∞]
Purge	2.17 (0.77)	0.16	2.81**	[0.90, ∞]

Restriction	1.56 (1.76)	0.05	0.89	[-1.34, ∞]
Fasting	1.03 (0.43)	0.15	2.38**	[0.32, ∞]

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Model 3: Controlling for Baseline NSSI and DE

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NSSI	[-0.04 – 0.15] (0.25 – 0.27)	-0.01 – 0.03	[-0.08 – 0.56]	[-0.46, ∞]
DE Composite	3.89 (1.28)	0.20	3.05**	[1.79, ∞]
Binge	0.40 (0.49)	0.06	0.811	[-0.41, ∞]
Purge	2.25 (0.78)	0.77	2.92**	[0.98, ∞]
Restriction	1.62 (1.81)	0.05	0.89	[-1.37, ∞]
Fasting	1.08 (0.43)	0.16	2.54*	[0.38, ∞]

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*Note.* NSSI = Nonsuicidal self-injury, DE Composite = Disordered eating, composite of bingeing, purging, and fasting

<sup>a</sup> Model controls for SC and baseline value of dependent variable only.

\*  $p < .05$ , one-tailed, \*\*  $p < .01$ , one-tailed

Table 13.

*Study 2: Longitudinal Effects of DE on NSSI (Controlling for Baseline NSSI and SC)*

Predictor	Unstandardized Path (SE)	Standardized Path	<i>z</i> (Unstd. Path)	95% CI
DE Composite	0.02 (0.02)	0.13	1.19	[-0.01, 0.06]
Binge	-0.00 (0.04)	-0.00	0.98	[-0.09, 0.08]
Purge	0.05 (0.03)	0.18	1.79 <sup>†</sup>	[-0.01, 0.10]
Restriction	-0.01 (0.01)	-0.05	-0.64	[-0.02, 0.01]
Fasting	0.03 (0.04)	0.06	0.66	[-0.05, 0.10]

*Note.* DE = Disordered eating (composite of bingeing, purging, and fasting); NSSI = Nonsuicidal self-injury; SC = Self-criticism

<sup>†</sup>*p* < .10, two-tailed



Table 14.

*Study 2: Longitudinal Effects of NSSI on DE (Controlling for Baseline DE and SC)*

Dependent Variable	Unstandardized Path (SE)	Standardized Path	<i>z</i> (Unstd. Path)	95% CI
DE Composite	-0.13 (0.31)	-0.03	-0.42	[-0.73, 0.48]
Binge	0.11 (0.16)	0.07	0.73	[-0.19, 0.42]
Purge	-0.23 (0.16)	-0.07	-1.39	[-0.57, 0.08]
Restriction	0.42 (0.46)	0.05	0.91	[-0.48, 1.32]
Fasting	-0.07 (0.12)	-0.04	-0.54	[-0.30, 0.17]

*Note.* NSSI = Nonsuicidal self-injury; DE composite = Disordered eating composite (sum of bingeing, purging, and fasting); SC = Self-criticism.

Table 15.

*Study 2: Main effects of SC and Stress on NSSI and DE*

	Unstandardized Path ( <i>SE</i> )	Standardized Path	<i>z</i>	95% CI
Main effect of Stress (controlling for SC)				
NSSI	[0.59-0.60] (0.45)	[0.09-0.10]	[1.32-1.34]	[-0.14, ∞]
DE composite	8.18 (2.28)	0.25	3.59**	[4.43, ∞]
Binge	2.29 (0.69)	0.23	3.25**	[0.12, ∞]
Purge	2.95 (1.50)	0.15	1.97*	[0.48, ∞]
Restriction	6.66 (4.01)	0.12	1.66*	[0.07, ∞]
Fasting	3.00 (0.87)	0.24	3.47**	[1.58, ∞]
Main Effect of SC (controlling for Stress)				
NSSI	[0.55-0.56] (0.28)	0.14	[1.99-2.01]*	[0.03, ∞]
DE composite	2.05 (1.32)	0.10	1.55	[-0.12, ∞]
Binge	0.59 (0.38)	0.10	1.58	[-0.01, ∞]
Purge	0.62 (0.89)	0.05	0.69	[-0.85, ∞]

Restriction	4.62 (2.37)	0.14	1.95*	[0.72, ∞]
Fasting	0.83 (0.48)	0.11	1.75*	[0.05, ∞]

---

*Note.* NSSI = Nonsuicidal self-injury; DE composite = composite of bingeing, purging, and fasting.

\* $p < .05$ , one-tailed, \*\*  $p < .01$ , one-tailed.

Table 16.

*Study 3: Self-Reported Baseline NSSI, DE*

Behavior	% Sample Endorsing Behavior	M ( <i>SD</i> )/Range	Skewness	Kurtosis
EDE-Q DE Composite <sup>†</sup>	67.50	6.90 (9.68)	1.88	3.71
EDE-Q Binge	43.75	1.71 (3.07)/0-20	3.31	15.51
EDE-Q Purge	42.50	4.08 (7.42)	2.23	4.69
EDE-Q Restriction	85.00	26.12 (27.75)/0-89	0.98	-0.44
EDE-Q Fasting	26.25	1.11 (2.33)/0-14	3.24	13.32
SCID AN (Lifetime diagnosis)	5.00	n/a	n/a	n/a
SCID BN (Lifetime diagnosis)	10.00	n/a	n/a	n/a
SCID BED (Lifetime diagnosis)	2.50	n/a	n/a	n/a
Past Year NSSI	25.00	2.39 (10.21)/0-88	7.73	64.50
Lifetime NSSI	42.50	27.73 (195.32)/0-1750	8.90	79.44

*Note.* Bingeing, restriction, and fasting counts reflect number of days on which the behavior occurred. NSSI and purging reflect number of episodes. EDE-Q = Eating Disorder Examination-Questionnaire, SCID = Structured Clinical Interview for DSM-IV, AN = Anorexia nervosa, BN = Bulimia nervosa, BED = Binge eating disorder.

<sup>†</sup>Reflects past-month behavior

Table 17.

*Study 3: Correlations of NSSI, DE Variables with Pre- and Post-test SC (Pearson's  $r$  with robust standard error)*

Outcome	Explicit SC Pre-Test	AMP-Self Pre-Test	AMP-Body Pre-Test	Explicit SC Post-Test	AMP-Self Post-Test	AMP-Body Post-Test
4. NSSI Any Lifetime Hx <sup>a</sup>	.68**	-.21	-.15	.60**	-.34**	.01
5. NSSI Lifetime Frequency	.11**	.09*	-.12**	.05**	.04	.19*
6. NSSI Any Past Year Hx <sup>a</sup>	.73**	-.13	-.17	.56**	-.14	-.02
7. NSSI Past Year Frequency	.20**	.11*	-.13	.10**	.02	.19*
8. EDE-Q DE All	.40**	-.50*	-.31**	.28*	-.10	-.27**
9. EDE-Q Binge	.37**	-.22**	-.22*	.32**	-.20*	-.12
10. EDE-Q Purge	.24*	-.16*	-.22*	.14	-.01	-.22*
11. EDE-Q Restriction	.23*	-.28**	-.31**	.13	-.17	-.38**
12. EDE-Q Fasting	.42**	-.16	-.29*	.29**	-.10	-.26**
13. Lifetime AN <sup>a</sup>	-.21	-.20	-.42*	-.16	-.31	.06
14. Lifetime BN <sup>a</sup>	.24	-.31	-.39*	.13	-.22	-.06
15. Lifetime BED <sup>a</sup>	.05	-.12	.11	-.09	.20	-.02

*Note.* Point estimates are Pearson's  $r$ . Significance levels determined using robust standard errors (except where indicated).

<sup>a</sup> Denotes dichotomous variable. Estimates are point-biserial correlations without use of robust standard errors.

\*  $p < .05$ , one-tailed, \*\*  $p < .01$ , one-tailed.

Table 18.

*Associations of Reactive SC with NSSI, DE (Pearson's r with Robust Standard Errors)*

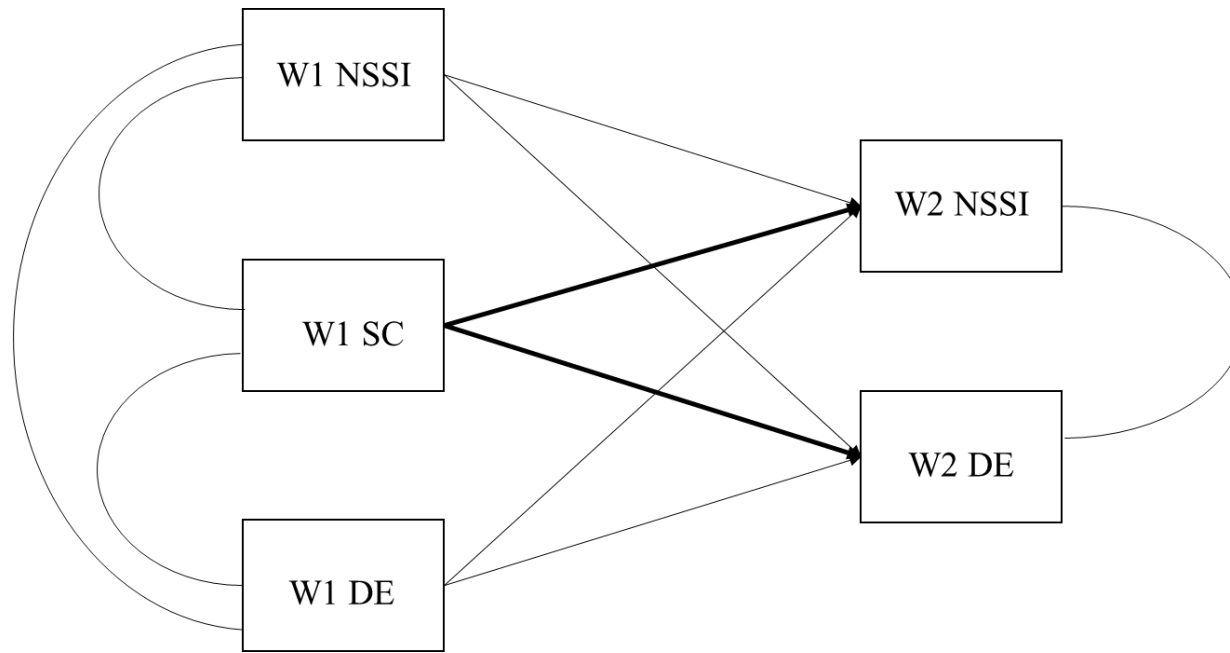
Outcome	Explicit SC	AMP-Self <sup>b</sup>	AMP-Body <sup>b</sup>
4. NSSI Any Lifetime Hx <sup>a</sup>	-.13	.16	.07
5. NSSI Lifetime Frequency	-.01	-.02	.08
6. NSSI Any Past Year Hx <sup>a</sup>	-.09	.08	-.07
7. NSSI Past Year Frequency	.08	-.07	.12
8. EDE-Q DE All	-.06	.11	.04
9. EDE-Q Binge	-.01	.09	.03
10. EDE-Q Purge	-.03	.07	.05
11. EDE-Q Restriction	-.13	.07	.06
12. EDE-Q Fasting	-.13	.11	-.05
13. Lifetime AN <sup>a</sup>	-.00	-.07	.23
14. Lifetime BN <sup>a</sup>	-.32	.22 <sup>†</sup>	-.19*
15. Lifetime BED <sup>a</sup>	.00	.46 <sup>†</sup>	n/a

*Note.* SC = self-criticism, AMP-Self = Affect Misattribution Procedure, self-focused stimuli, AMP-Body = Affect Misattribution Procedure, body-focused stimuli, NSSI = Nonsuicidal self-injury, EDE-Q = Eating Disorders Examination-Questionnaire, DE All = Disordered eating composite (bingeing, purging, fasting), AN = Anorexia nervosa, BN = Bulimia nervosa, BED = Binge eating disorder.

<sup>a</sup> Dichotomous variables. Correlations reflect point-biserial values without the use of robust standard errors. The model for reactive SC based on body-focused stimuli could not be estimated for BED due to low numbers of participants meeting criteria for the diagnosis.

<sup>b</sup> Scales are reverse-coded.

\* $p < .05$ , one-tailed.



*Figure 1.* Heuristic of full path diagram. Wave 2 NSSI and DE are regressed on baseline behaviors and SC; DE behaviors are tested in separate models.

## APPENDIX A – SUPPLEMENTARY TABLES

Table A1.

*Zero-order correlations between SC, NSSI, and DE (Spearman's  $\rho$ ), Study 1*

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
1. W1 Self-Criticism	–														
2. W1 AMP Self-focused stimuli	-.24	–													
3. W1 Body-focused stimuli	-.36	.45	–												
4. W1 NSSI Wave 1	.22	-.06	-.07	–											
5. W1 DE Composite	.25	-.05	-.05	.11	–										
6. W1 Binge Wave 1	.17	-.00	-.01	-.08	.66	–									
7. W1 Purge Wave 1	.11	-.02	.02	.15	.69	.31	–								
8. W1 Restriction	.16	-.03	-.09	.09	.68	.32	.51	–							
9. W1 Fasting	.27	-.09	-.09	.17	.70	.18	.36	.61	–						
10. W2 NSSI	.33	-.06	-.10	.43	.18	.03	.08	.18	.23	–					
11. W2 DE Composite	.29	-.10	-.16	.15	.58	.43	.50	.48	.39	.26	–				
12. W2 Binge Wave 1	.14	-.01	-.09	-.11	.29	.49	.22	.25	.04	.06	.64	–			
13. W2 Purge Wave 1	.14	-.10	-.06	.13	.43	.22	.55	.29	.25	.09	.66	.21	–		
14. W2 Restriction	.13	-.09	-.09	.12	.50	.28	.41	.60	.42	.24	.63	.28	.46	–	
15. W2 Fasting	.33	-.12	-.17	.26	.46	.23	.31	.42	.52	.33	.70	.17	.42	.57	–
<i>M</i>	.00	0.68	0.61	0.03	7.49	1.79	2.45	8.14	3.34	.06	5.46	1.36	1.55	7.07	2.61
<i>SD</i>	0.94	0.25	0.27	.19	10.72	3.95	5.39	7.41	6.43	0.35	9.37	3.08	4.21	6.78	5.63
Skewness	.08	-.42	-.34	7.34	1.58	3.95	2.83	0.70	1.69	6.05	2.20	3.67	3.56	0.80	1.88

*Note.* *Ns* range from 222-245 for Wave 1 correlations. *Ns* range from 196-201 for Wave 2 correlations. AMP = Affect Misattribution Procedure, NSSI = Nonsuicidal self-injury; DE = Disordered eating; SC = Self-criticism. Correlations  $\geq .11$  are significant at  $p < .10$ ; correlations  $\geq .14$  are significant at  $p < .05$ .



Table A2.

*Comparison of Zero-Order NSSI-DE Correlations and Partial Correlations Controlling for SC, Study 1 (Spearman's rho)*

DE Variable	Zero-order		Partial Correlation (controlling for Wave 1 SC)		Zero-order Correlation Accounted for by SC %
	<i>r</i>	p-value	<i>r</i>	p-value	
2. W1 DE Composite	.11	.09	.06	ns	45.45
3. W1 Binge Wave 1	-.08	ns	-.12	.07	58.67
4. W1 Purge Wave 1	.15	.02	.13	.05	13.33
5. W1 Restriction Wave 1	.09	ns	.08	ns	2.33
6. W1 Fasting Wave 1	.17	.01	.12	.05	27.06

*Note.* NSSI = Nonsuicidal self-injury; DE composite = composite of bingeing, purging, and fasting; SC = Self-criticism

Table A3.

*Correlations among W1 and W2 Variables, Study 2 (Spearman's  $\rho$ )*

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1. W1 Self-Criticism	–																			
2. W1 NSSI	.24	–																		
3. W1 DE Composite	.25	.17	–																	
4. W1 Binge Count	.23	.14	.71	–																
5. W1 BULIT – Binge	.33	.13	.53	.67	–															
6. W1 Purge Count	.14	.13	.76	.36	.27	–														
7. W1 BULIT – Purge	.25	.12	.69	.45	.53	.63	–													
8. W1 Restriction	.18	.08	.62	.41	.37	.43	.52	–												
9. W1 DEBQ	.26	.13	.55	.31	.31	.45	.63	.71	–											
10. W1 Fasting	.27	.17	.78	.46	.39	.44	.59	.59	.51	–										
11. W2 NSSI	.13	.41	.21	.21	.18	.18	.17	–	.07	.14	–									
12. W2 DE Composite	.29	.17	.63	.52	.53	.50	.66	.43	.46	.53	.12	–								
13. W2 Binge Count	.30	.19	.43	.49	.56	.27	.42	.25	.24	.29	.07	.72	–							
14. W2 BULIT – Binge	.40	.24	.49	.56	.74	.29	.48	.29	.23	.35	.12	.60	.71	–						
15. W2 Purge Count	.17	.07	.48	.29	.33	.59	.56	.38	.43	.36	.07	.73	.38	.36	–					
16. W2 BULIT – Purge	.32	.18	.56	.42	.49	.41	.67	.51	.57	.55	.13	.65	.39	.51	.65	–				
17. W2 Restriction	.18	.14	.48	.27	.30	.45	.55	.68	.68	.45	.10	.59	.29	.30	.47	.57	–			
18. W2 DEBQ	.20	.19	.48	.33	.37	.44	.71	.62	.79	.49	.07	.48	.23	.34	.43	.60	.71	–		
19. W2 Fasting	.26	.07	.47	.36	.32	.32	.56	.39	.44	.53	.15	.72	.29	.29	.42	.53	.59	.47	–	
20. W2 Self-Criticism	.75	.25	.20	.17	.33	.15	.31	.17	.19	.22	.26	.27	.25	.40	.15	.28	.20	.30	.25	–

*Note.* *Ns* among Wave 1 variables = 307, *Ns* among Wave 2 variables = 315, *Ns* from Wave 1 to Wave 2 variables = 139. DEBQ = Dutch Eating Behaviors Questionnaire, Restriction Subscale; BULIT = Bulimia Test-Revised (binge eating items or purging items as designated); NSSI = Nonsuicidal self-injury; DE composite = composite of bingeing, purging, and fasting. Correlations > .17 are significant at  $p < .05$ .

Table A4.

*Comparison of Zero-order and Partial Correlations of Wave 1 NSSI and DE Variables (Study 2, Spearman's  $\rho$ )*

DE Variable	Zero-order		Partial Correlation (controlling for Wave 1 SC)		Zero-order Correlation Accounted for by SC
	<i>r</i>	p-value	<i>r</i>	p-value	%
1. W1 DE Composite	.17	.00	.12	.04	30.18
2. W1 Binge Wave 1	.14	.02	.09	.11	35.51
3. W1 BULIT-Binge	.13	.03	.06	.34	57.03
4. W1 Purge Wave 1	.13	.02	.10	.08	22.14
5. W1 BULIT-Purge	.12	.04	.06	.27	46.15
6. W1 Restriction	.08	.19	.04	.54	53.33
7. W1 DEBQ	.13	.03	.07	.23	45.24
8. W1 Fasting Wave 1	.17	.00	.12	.04	32.18

*Note.* DE = Disordered eating (composite consists of bingeing, purging, and fasting); BULIT = Bulimia Test-Revised (binge or purge items, where indicated); DEBQ = Dutch Eating Behaviors Questionnaire; NSSI = Nonsuicidal self-injury; SC = Self-criticism.

Table A5.

*Comparison of Zero-Order and Partial Correlations of Wave 1 NSSI, DEBQ, and BULIT (Study 2, Parametric Statistics, Robust Standard Errors)*

DE Variable	Zero-order		Partial Correlation (controlling for Wave 1 SC)		Zero-order Correlation Accounted for by SC
	<i>r</i>	p-value	<i>r</i>	p-value	%
1. W1 BULIT-Binge	0.15	0.01	0.08	0.15	46.67
2. W1 BULIT-Purge	.12	.05	.07	.24	41.66
3. W1 DEBQ	0.12	0.03	0.06	0.23	50.00

*Note.* BULIT = Bulimia Test-Revised (binge or purge items, where indicated); DEBQ = Dutch Eating Behaviors Questionnaire; NSSI = Nonsuicidal self-injury; SC = Self-criticism.

Table A6.

*Study 2: Longitudinal Effect of Self-Criticism on NSSI, DE (BULIT & DEBQ) (n = 517)*

Dependent Variable	Unstandardized Path (SE)	Standardized Path	z (Unstd. Path)
Model 1: Not Controlling for Baseline Behavior			
NSSI	[0.19 – 0.29] ([0.25 – 0.27])	[0.06 – 0.09]	[0.81 – 1.12]
BULIT - Binge	0.43 (0.10)	0.36	4.51**
BULIT - Purge	0.26 (0.06)	0.27	4.20**
DEBQ	0.20 (0.08)	0.19	2.48*
Model 2: Controlling for Baseline Behavior			
NSSI	[-0.04 – 0.15] (0.25 – 0.27)	-0.01 – 0.03	[-0.08 – 0.56]
BULIT - Binge	0.14 (0.09)	0.12	1.48
BULIT - Purge	0.07 (0.04)	0.07	1.79*
DEBQ	-0.01 (0.06)	-0.01	0.25

*Note.* DEBQ = Dutch Eating Behaviors Questionnaire, Restriction Subscale; BULIT = Bulimia Test-Revised (binge eating items or purging items).

\*  $p < .05$ , one-tailed; \*\*  $p < .01$ , one-tailed.

Table A7.

*Study 2: Main effects of SC and Stress on NSSI and DE (BULIT and DEBQ)*

	Unstandardized Path ( <i>SE</i> )	Standardized Path	<i>z</i>
Main effect of Stress (controlling for SC)			
BULIT-Binge	0.48 (0.11)	0.28	4.43**
BULIT-Purge	0.21 (0.07)	0.20	3.03**
DEBQ	0.20 (0.13)	0.12	1.59
Main Effect of SC (controlling for Stress)			
BULIT-Binge	0.21 (0.07)	0.20	3.00**
BULIT-Purge	0.14 (0.04)	0.22	3.20**
DEBQ Restriction	0.24 (0.07)	0.22	3.18**

*Note.* BULIT = Bulimia Test-Revised, DEBQ = Dutch Eating Behaviors Questionnaire; NSSI = Nonsuicidal self-injury; DE = Disordered eating; SC = Self-criticism.

Table A8.

*Reciprocal Longitudinal Effects of NSSI and DE (Controlling for Baseline Behavior and SC)*

DE Variable	Unstandardized Path (SE)	Standardized Path	Z (Unstd. Path)
DE As Predictor of NSSI			
BULIT - Binge	-0.05 (0.22)	-0.02	-0.23
BULIT - Purge	0.45 (0.49)	0.09	0.92
DEBQ Restriction	-0.23 (0.24)	-0.07	-0.98
NSSI as Predictor of DE			
BULIT - Binge	0.01 (0.02)	0.03	0.62
BULIT - Purge	-0.00 (0.01)	-0.01	-0.08
DEBQ Restriction	0.01 (0.02)	0.03	0.41

*Note.* BULIT = Bulimia Test-Revised (binge item subscale and purge item subscale where indicated), DEBQ = Dutch Eating Behaviors Questionnaire, Restriction subscale. Each row represents a separate model in which NSSI or DE was regressed onto the other behavior, controlling for baseline NSSI or DE and SC.

Table A9.

*Pre-test and Post-test SC in Relation to NSSI, DE (Spearman's rho)*

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.
1. Pre SC (explicit measures)	–																	
2. Pre AMP-Self	-.16	–																
3. Pre AMP-Body	-.43	.22	–															
4. NSSI Any Lifetime Hx	.55	-.17	-.14	–														
5. NSSI Lifetime Frequency	.56	-.12	-.19	.93	–													
6. NSSI Any Past Year Hx	.56	-.07	-.14	.66	.74	–												
7. NSSI Past Year Frequency	.55	-.03	-.16	.65	.75	.99	–											
8. EDE-Q DE All	.41	-.33	-.29	.24	.17	.12	.09	–										
9. EDE-Q Binge	.28	-.24	-.25	.27	.20	-.06	-.06	.57	–									
10. EDE-Q Purge	.28	-.25	-.21	.12	.05	.12	.08	.80	.19	–								
11. EDE-Q Restriction	.20	-.30	-.33	-.07	-.07	-.02	-.03	.61	.22	.61	–							
12. EDE-Q Fasting	.38	-.20	-.25	.09	.07	.18	.15	.64	.22	.42	.53	–						
13. Lifetime AN	-.09	-.08	-.18	-.08	-.05	-.13	-.13	-.02	.02	-.10	-.07	-.01	–					
14. Lifetime BN	.16	-.18	-.26	.21	.31	.19	.18	.21	.18	.11	.18	.24	.31	–				
15. Lifetime BED	.03	-.06	.04	.02	.06	.10	.06	.10	.10	.15	.11	.08	-.04	.21	–			
16. Post SC (explicit measures)	.90	-.07	-.44	.49	.47	.43	.42	.29	.25	.19	.13	.25	-.06	.10	-.03	–		
17. Post AMP-Self	-.23	.33	.35	-.28	-.21	-.09	-.10	-.12	-.15	-.15	-.10	-.11	-.16	-.14	.07	-.25	–	
18. Post AMP-Body	-.23	.37	.49	.01	.01	-.03	-.03	-.27	-.15	-.27	-.35	-.18	.04	-.02	-.00	-.33	.42	–

*Note.* Correlations greater than  $|.21|$  are significant at  $p < .05$ . SC = self-criticism, AMP-Self = Affect Misattribution Procedure, self-focused stimuli, AMP-Body = Affect Misattribution Procedure, body-focused stimuli, NSSI = Nonsuicidal self-injury, EDE-Q = Eating Disorders Examination-Questionnaire, DE All = Disordered eating composite (bingeing, purging, fasting), AN = Anorexia nervosa, BN = Bulimia nervosa, BED = Binge eating disorder.



Table A10.

*Associations of Reactive SC with NSSI, DE (Spearman's  $\rho$ )*

Outcome	Explicit SC	AMP-Self	AMP-Body
4. NSSI Any Lifetime Hx	-.04	-.05	.22
5. NSSI Lifetime Frequency	-.07	-.04	.24*
6. NSSI Any Past Year Hx	-.15	.04	.15
7. NSSI Past Year Frequency	-.13	.01	.17
8. EDE-Q DE All	-.09	.08	.11
9. EDE-Q Binge	-.03	.09	.03
10. EDE-Q Purge	-.01	-.00	.05
11. EDE-Q Restriction	-.32**	.14	.11
12. EDE-Q Fasting	-.29**	.13	.06
13. Lifetime AN	-.07	-.03	.26*
14. Lifetime BN	-.14	.01	.14
15. Lifetime BED	-.17	.17	-.17

*Note.* SC = self-criticism, AMP-Self = Affect Misattribution Procedure, self-focused stimuli, AMP-Body = Affect Misattribution Procedure, body-focused stimuli, NSSI = Nonsuicidal self-injury, EDE-Q = Eating Disorders Examination-Questionnaire, DE All = Disordered eating composite (bingeing, purging, fasting), AN = Anorexia nervosa, BN = Bulimia nervosa, BED = Binge eating disorder.

\*  $p < .05$ , \*\*  $p < .01$ .

**APPENDIX B**  
**STUDY 2 ANALYSES BASED ON SELF-INJURIOUS THOUGHTS AND BEHAVIOR INTERVIEW, SELF-REPORT**  
**VERSION**

**Methods and Results of Study 2 SITBI Assessment**

To facilitate comparisons with Study 1, I conducted the set of analyses described in Study 2 using past-month NSSI as assessed by the self-report version of the Self-Injurious Thoughts and Behavior Interview (SITBI; the same NSSI assessment method used in Study 1). As in Study 1, participants estimated the number of times they engaged in any form of self-harm in the past month after reading a list of example behaviors (e.g., cutting, burning, hitting the self). Based on the SITBI, 16.29% of respondents reported past-month NSSI at Wave 1 ( $M = 1.68$ ,  $SD = 12.97$ , Skewness = 13.06,  $SE$  of skewness = 0.14); 16.05% of respondents reported past-month NSSI at Wave 2 ( $M = 0.77$ ,  $SD = 3.60$ , Skewness = 9.40,  $SE = 0.13$ ). I then binned the values for NSSI at Waves 1 and 2 in a manner similar to that described in Study 2. Table B1 shows the correlations of SC with the binned NSSI value and DE at both waves. (Values for DE variables are identical to the main text). Table B2 shows both the zero-order correlations of NSSI (as assessed by the SITBI) and each DE variable and the partial correlations of the same, controlling for SC. Correlations of NSSI with the DE composite, purging, and fasting dropped after controlling for SC (correlations of NSSI with bingeing and restriction actually increased after controlling for SC).

Aim 2 was to explore SC as a transdiagnostic predictor of NSSI and DE. I first explored Wave 1 SC as a predictor of each behavior at Wave 2, absent any controls for baseline behavior. The first portion of Table B3 shows these results; Wave 1 SC significantly predicted both NSSI and all forms of DE at Wave 2. I then extended this analysis by testing SC as a predictor of NSSI or

DE, controlling for baseline levels of the focal behavior (see Table B3, Model 2). SC significantly predicted NSSI, the DE composite, purging, and fasting. Finally, I repeated the analysis using a multivariate model that included both NSSI and DE at Wave 1 and Wave 2 (see Figure 1; DE behaviors were rotated through the models). In this expanded model, SC remained a significant predictor of NSSI when controlling for baseline NSSI and bingeing; however, the SC → NSSI path was nonsignificant when each of the other DE behaviors was rotated through the model. SC remained a significant predictor of the DE composite, purging, and fasting when controlling for baseline levels of the focal DE variable and NSSI. I also noted reciprocal effects of NSSI and DE (see Tables B4 and B5). The DE composite at baseline significantly predicted NSSI at Wave 2; NSSI did not predict any DE behavior at follow-up.

I did not find evidence of a Stress x SC interaction (Aim 3) using the SITBI assessment of NSSI. I proceeded to explore main effects of stress and SC in relation to NSSI. SC was significantly related to NSSI after controlling for stress (unstandardized path = 0.19,  $SE = 0.11$ , standardized path = 0.12,  $z = 1.71$ ,  $p = .04$ , one-tailed). Stress was not significantly related to NSSI after controlling for SC.

## **Discussion**

When I used the SITBI to assess NSSI (i.e., a method comparable to that used in Study 1), SC emerged as a significant predictor of both NSSI and multiple forms of DE prior to and after controlling for baseline behavior. These results are generally consistent with those of Study 1 and provide further support for SC as a transdiagnostic predictor of both NSSI and DE. Findings are also consistent with work by Fox et al. (2018), which demonstrated that SC significantly predicted NSSI at follow-up among a sample of adults with recent histories of the behavior. The discrepancy between findings based on the SITBI versus those based on a

composite of the SITBI and the Deliberate Self-Harm Inventory (DSHI; presented in the main text) is notable. This may be due to the fact that the DSHI contains some highly severe forms of NSSI (e.g., breaking bones, rubbing glass into the skin) that are less frequently endorsed than behaviors on the SITBI (Latimer, Covic, & Tennant, 2012). To my knowledge, no research has directly compared NSSI frequency as assessed by the DSHI versus the SITBI (although see Latimer, Meade, & Tennant, 2013, for efforts to integrate multiple NSSI scales including the SITBI and DSHI into a single Rasch measurement model). Further research comparing these two measures will be required to better understand their performance across a variety of samples.

In addition to testing SC as a sole predictor of subsequent behavior and controlling for baseline behavior, I expanded the model to control for baseline DE in addition to NSSI (i.e., a more stringent test of transdiagnostic prediction). SC was no longer a significant predictor of Wave 2 NSSI in this expanded model (except when controlling for baseline bingeing). This was likely due to the baseline correlation of SC and DE rather than the effect of DE itself on NSSI (i.e., the DE-SC correlation diminished the contribution of SC to subsequent NSSI). Lower path estimates for the SC-NSSI relation in Study 2 (versus Study 1) prior to controlling for any form of DE support this explanation.

Analyzing the data from Study 2 using the SITBI to assess NSSI allows for direct comparison of the results from Studies 1 and 2. The findings generally confirm longitudinal effects of SC on both NSSI and DE. A key limitation of this approach, however, is its use of a single item to assess NSSI at both waves. Stability of this item was low (as in Study 1), and it will be important to confirm the findings using other composite assessments of NSSI (e.g., a composite of SITBI behavior items only).

Table B1

*Correlations of SC with NSSI, DE, By Wave (Study 2)*

Behavior	W1 SC
Wave 1	
1. W1 NSSI	.15**
2. W1 DE Composite	.25**
3. W1 Binge	.23*
4. W1 Purge	.13**
5. W1 Restrict	.21**
6. W1 Fast	.25**
Wave 2	
1. W2 NSSI	.21**
2. W2 DE Composite	.27**
3. W2 Binge	.20**
4. W2 Purge	.19**
5. W2 Restrict	.21**
6. W2 Fast	.23**

*Note.* All estimates calculated as Pearson's  $r$  with robust standard errors.

\*\*  $p < .01$ .

Table B2.

*Zero-order and Partial Correlations among Wave 1 NSSI and DE Variables, Study 2 (Pearson's  $r$  with robust standard error)*

DE Variable	Zero-order		Partial Correlation (controlling for Wave 1 SC)		% of Zero-order Correlation Accounted for by SC
	$r$	p-value	$r$	p-value	
2. W1 DE All	.06	ns	.02	ns	66.67
3. W1 Binge Wave 1	-.03	ns	-.07	ns	n/a
5. W1 Purge Wave 1	.09	ns	.07	ns	22.22
7. W1 Restriction	-.03	ns	-.06	ns	n/a
9. W1 Fasting Wave 1	.02	ns	-.01	ns	n/a

*Note.* Based on  $n = 307$ .

Table B3.

*Longitudinal Effect of Self-Criticism on NSSI, DE (n = 517)*

Dependent Variable	Unstandardized Path (SE)	Standardized Path	z (Unstd. Path)
Model 1: No Control for Baseline Behavior			
NSSI	.26 (.11)	.17	2.35**
DE Composite	6.40 (1.38)	0.34	4.62**
Binge	1.25 (0.56)	0.20	2.25*
Purge	3.38 (0.95)	0.26	3.57**
Restriction	1.57 (0.58)	0.20	2.69*
Fasting	1.58 (0.46)	0.23	3.45**
Model 2: Controlling for Baseline NSSI or DE			
NSSI	0.22 (0.11)	0.15	2.06*
DE Composite	3.85 (1.29)	0.20	2.99**
Binge	0.46 (0.50)	0.07	0.92
Purge	2.17 (0.77)	0.16	2.81**
Restriction	1.56 (1.76)	0.05	0.89
Fasting	1.03 (0.43)	0.15	2.38**
Model 3: Controlling for Baseline NSSI and DE			
NSSI	0.09 - .23 (.11-.12)	.08-.15*	1.14-2.05*
DE Composite	3.89 (1.28)	0.20	3.05**
Binge	0.40 (0.49)	0.06	0.811
Purge	2.11 (0.74)	0.16	2.86**
Restriction	0.15 (0.26)	0.05	0.56
Fasting	1.08 (0.43)	0.16	2.54**

*Note.* NSSI = Nonsuicidal self-injury; DE = Disordered eating (composite reflects sum of bingeing, purging, and fasting).

\*  $p < .05$ , one-tailed, \*\*  $p < .01$ , one-tailed.

Table B4.

*Longitudinal Effects of DE on NSSI (Controlling for Baseline NSSI and SC)*

Predictor	Unstandardized Path (SE)	Standardized Path	<i>z</i> (Unstd. Path)
DE Composite	.02 (.009)	.23	1.96*
Binge	-0.00 (0.04)	-0.00	0.98
Purge	.03 (0.02)	.23	1.87
Restriction	.03 (.02)	.14	1.63
EDE-Q Fasting	.04 (.02)	.20	1.79

*Note.* NSSI = Nonsuicidal self-injury; DE = Disordered eating (composite reflects sum of bingeing, purging, and fasting), SC = Self-criticism.

\*  $p \leq .05$ , two-tailed.



Table B5.

*Longitudinal Effects of NSSI on DE (Controlling for Baseline DE and SC)*

Dependent Variable	Unstandardized Path (SE)	Standardized Path	<i>z</i> (Unstd. Path)
DE Composite	1.07 (.72)	.09	1.48
Binge	0.11 (0.16)	0.07	0.73
Purge	0.22 (.47)	.03	0.47
Restriction	-.20 (.74)	-.01	-.27
Fasting	-.01 (.26)	-.003	-.04

*Note.* NSSI = Nonsuicidal self-injury; DE = Disordered eating (composite reflects sum of bingeing, purging, and fasting); SC = Self-criticism.

Table B6

*Study 2: Main effects of SC and Stress on NSSI and DE*

Dependent Variable	Unstandardized Path ( <i>SE</i> )	Standardized Path	<i>z</i>
Main effect of Stress (controlling for SC)			
NSSI	.13 (0.19)	0.05	0.69
DE composite	8.17 (2.28)	0.25	3.59**
Binge	2.22 (0.69)	0.23	3.25**
Purge	2.95 (1.50)	0.15	1.97*
Restriction	6.65 (4.01)	0.12	1.66*
Fasting	3.00 (0.87)	0.24	3.47**
Main Effect of SC (controlling for Stress)			
NSSI	0.19 (0.11)	0.12	1.71*
DE composite	2.05 (1.32)	0.10	1.55
Binge	0.59 (0.38)	0.10	1.58
Purge	0.62 (0.89)	0.05	0.69
Restriction	4.63 (2.37)	0.14	1.95*
Fasting	0.83 (0.48)	0.11	1.75*

*Note.* NSSI = Nonsuicidal self-injury; SC = Self-criticism; DE = Disordered eating (composite refers to sum of bingeing, purging, and fasting)

\*  $p < .05$ , one-tailed, \*\*  $p < .01$ , one-tailed.

## Appendix C

### COMPENDIUM OF MEASURES

#### Qualtrics Panel Screener (Study 2)

What is your age (in years)?

Q7

Do you speak English fluently?

- Yes
- No

Q3

Below are examples of thoughts and behaviors people sometimes report. Please check all that have applied to you over your LIFETIME.

- Felt upset because of something that happened unexpectedly.
- Engaged in severely restricting food, purging food (e.g., by vomiting, laxatives, intense exercise), or bingeing on food.
- Had thoughts of severely restricting food, purging food (e.g., by vomiting, laxatives, intense exercise), or bingeing on food.
- Felt overwhelmed by health concerns.
- Hurt myself without intending to die (e.g., by cutting, carving, burning, banging, biting, picking, giving self a tattoo, scraping skin, pulling out hair, picking at skin to draw blood, etc.)
- Had thoughts of hurting myself without intending to die (e.g., by cutting, carving, burning, etc.)
- Often felt down, depressed, and hopeless.
- × None of the above

Below are examples of thoughts and behaviors people sometimes report. Please check all that have applied to you over the PAST YEAR.

- Felt upset because of something that happened unexpectedly.
- Engaged in severely restricting food, purging food (e.g., by vomiting, laxatives, intense exercise), or bingeing on food.
- Had thoughts of severely restricting food, purging food (e.g., by vomiting, laxatives, intense exercise), or bingeing on food.
- Felt overwhelmed by health concerns.
- Hurt myself without intending to die (e.g., by cutting, carving, burning, banging, biting, picking, giving self a tattoo, scraping skin, pulling out hair, picking at skin to draw blood, etc.)
- Had thoughts of hurting myself without intending to die (e.g., by cutting, carving, burning, etc.)
- Often felt down, depressed, and hopeless.
- × None of the above



137) When you have had these thoughts, how long have they usually lasted?

- 0) 0 seconds
- 1) 1-60 seconds
- 2) 2-15 minutes
- 3) 16-60 minutes
- 4) less than one day
- 5) 1-2 days
- 6) more than 2 days
- 7) wide range (spans > 2 responses)
- 8) not applicable
- 9) unknown

138) Before you ever thought about engaging in NSSI, how many of your friends, to your knowledge, thought about engaging in NSSI?

139) Since the first time you thought about engaging in NSSI, how many of your friends have thought about engaging in NSSI?

140) Before you ever thought about engaging in NSSI, how much did your friends thinking about engaging in NSSI influence your thinking about engaging in NSSI, on a scale of 0 to 4?

41) Since you ever thought about engaging in NSSI, how much have your friends thinking about engaging in NSSI influenced your thinking about engaging in NSSI on a scale of 0 to 4?

142) On a scale of 0 to 4, what do you think the likelihood is that you will have thoughts about engaging in NSSI in the future?

### Non-Suicidal Self-Injury

143) Have you ever actually engaged in NSSI?

- 0) no
- 1) yes

144) How old were you the first time? (*age*)

145) How old were you the last time? (*age*)

146) How many times in your life have you engaged in NSSI?

147) How many times in the past year?

148) How many times in the past month?

149) How many times in the past week?

150) Now I'm going to go through a list of things that people have done to harm themselves. Please note how many times you have done each of these behaviors:

- 1) cut or carved skin \_\_\_\_\_
- 2) hit yourself on purpose \_\_\_\_\_
- 3) pulled your hair out \_\_\_\_\_
- 4) gave yourself a tattoo \_\_\_\_\_
- 5) picked at a wound \_\_\_\_\_
- 6) burned your skin (i.e., with a cigarette, match or other hot object) \_\_\_\_\_



4) less than one day

99) unknown

165) Before you ever engaged in NSSI, how many of your friends, to your knowledge, engaged in NSSI?

166) Since the first time you engaged in NSSI, how many of your friends have engaged in NSSI?

167) Before you ever engaged in NSSI, how much did your friends engaging in NSSI influence your engaging in NSSI, on a scale of 0 to 4?

168) Since the first time you engaged in NSSI, how much have your friends engaging of NSSI influenced your engaging in NSSI, on a scale of 0 to 4?

169) On a scale of 0 to 4, what do you think the likelihood is that you will engage in NSSI in the future?



DELIBERATE SELF-HARM INVENTORY (Gratz, 2001)

For measure, see:

Gratz, K. L. (2001). Measurement of Deliberate Self-Harm: Preliminary Data on the Deliberate Self-Harm Inventory. *Journal of Psychopathology and Behavioral Assessment*, 23(4), 253-263.

## EATING QUESTIONNAIRE

**Instructions:** The following questions are concerned with the past four weeks (28 days) only. Please read each question carefully. Please answer all the questions. Thank you.

**Questions 1 to 12:** Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days) only.

On how many of the past 28 days .....	No days	1-5 days	6-12 days	13-15 days	16-22 days	23-27 days	Every day
1 Have you been deliberately <u>trying</u> to limit the amount of food you eat to influence your shape or weight (whether or not you have succeeded)?	0	1	2	3	4	5	6
2 Have you gone for long periods of time (8 waking hours or more) without eating anything at all in order to influence your shape or weight?	0	1	2	3	4	5	6
3 Have you <u>tried</u> to exclude from your diet any foods that you like in order to influence your shape or weight (whether or not you have succeeded)?	0	1	2	3	4	5	6
4 Have you <u>tried</u> to follow definite rules regarding your eating (for example, a calorie limit) in order to influence your shape or weight (whether or not you have succeeded)?	0	1	2	3	4	5	6
5 Have you had a definite desire to have an <u>empty</u> stomach with the aim of influencing your shape or weight?	0	1	2	3	4	5	6
6 Have you had a definite desire to have a <u>totally flat</u> stomach?	0	1	2	3	4	5	6
7 Has thinking about <u>food, eating or calories</u> made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)?	0	1	2	3	4	5	6
8 Has thinking about <u>shape or weight</u> made it very difficult to concentrate on things you are interested in (for example, working, following a conversation, or reading)?	0	1	2	3	4	5	6
9 Have you had a definite fear of losing control over eating?	0	1	2	3	4	5	6
10 Have you had a definite fear that you might gain weight?	0	1	2	3	4	5	6
11 Have you felt fat?	0	1	2	3	4	5	6
12 Have you had a strong desire to lose weight?	0	1	2	3	4	5	6

Questions 13-18: Please fill in the appropriate number in the boxes on the right. Remember that the questions only refer to the past four weeks (28 days).

Over the past four weeks (28 days) .....

- 
- 13 Over the past 28 days, how many times have you eaten what other people would regard as an unusually large amount of food (given the circumstances)?
- 
- 14 ..... On how many of these times did you have a sense of having lost control over your eating (at the time that you were eating)?
- 
- 15 Over the past 28 days, on how many **DAYS** have such episodes of overeating occurred (i.e., you have eaten an unusually large amount of food and have had a sense of loss of control at the time)?
- 
- 16 Over the past 28 days, how many times have you made yourself sick (vomit) as a means of controlling your shape or weight?
- 
- 17 Over the past 28 days, how many times have you taken laxatives as a means of controlling your shape or weight?
- 
- 18 Over the past 28 days, how many times have you exercised in a "driven" or "compulsive" way as a means of controlling your weight, shape or amount of fat, or to burn off calories?
- 

Questions 19 to 21: Please circle the appropriate number. Please note that for these questions the term "binge eating" means eating what others would regard as an unusually large amount of food for the circumstances, accompanied by a sense of having lost control over eating.

19 Over the past 28 days, on how many days have you eaten in secret (ie, furtively)? ..... Do not count episodes of binge eating	No days	1-5 days	6-12 days	13-15 days	16-22 days	23-27 days	Every day
	0	1	2	3	4	5	6
20 On what proportion of the times that you have eaten have you felt guilty (felt that you've done wrong) because of its effect on your shape or weight? ..... Do not count episodes of binge eating	None of the times	A few of the times	Less than half	Half of the times	More than half	Most of the time	Every time
	0	1	2	3	4	5	6
21 Over the past 28 days, how concerned have you been about other people seeing you eat? ..... Do not count episodes of binge eating	Not at all	Slightly	Moderately	Markedly			
	0	1	2	3	4	5	6

Questions 22 to 28: Please circle the appropriate number on the right. Remember that the questions only refer to the past four weeks (28 days).

Over the past 28 days .....	Not at all		Slightly		Moderate-ly		Markedly
22 Has your <u>weight</u> influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
23 Has your <u>shape</u> influenced how you think about (judge) yourself as a person?	0	1	2	3	4	5	6
24 How much would it have upset you if you had been asked to weigh yourself once a week (no more, or less, often) for the next four weeks?	0	1	2	3	4	5	6
25 How dissatisfied have you been with your <u>weight</u> ?	0	1	2	3	4	5	6
26 How dissatisfied have you been with your <u>shape</u> ?	0	1	2	3	4	5	6
27 How uncomfortable have you felt seeing your body (for example, seeing your shape in the mirror, in a shop window reflection, while undressing or taking a bath or shower)?	0	1	2	3	4	5	6
28 How uncomfortable have you felt about <u>others</u> seeing your shape or figure (for example, in communal changing rooms, when swimming, or wearing tight clothes)?	0	1	2	3	4	5	6

### Supplementary Disordered Eating Questions (Past Year Behaviors)

(Note: Questions 1a-1d are adapted from the EDE-Q; Fairburn & Beglin, 1994. Question 1e is adapted from the Eating Attitudes Test-26; Garner, 1982)

1a) In the past year, have you ever gone for long periods of time (8 waking hours or more) without eating anything at all in order to influence your shape or weight?

- Yes
- No
- (If yes) Please estimate how many times:
  - a. 1-2
  - b. 3-10
  - c. More than 10 times

1b) In the past year, have you ever made yourself sick (vomit) as a means of controlling your shape or weight?

- Yes
- No
- (If yes) Please estimate how many times:
  - a. 1-2
  - b. 3-10
  - c. More than 10 times

1c) In the past year, have you ever taken laxatives as a means of controlling your shape or weight?

- Yes
- No
- (If yes) Please estimate how many times:
  - a. 1-2
  - b. 3-10
  - c. More than 10 times

1d) In the past year, have you ever exercised in a “driven” or “compulsive” way as a means of controlling your weight, shape or amount of fat, or to burn off calories?

- Yes
- No
- (If yes) Please estimate how many times:
  - a. 1-2
  - b. 3-10
  - c. More than 10 times

1e) In the past year, have you ever gone on eating binges where you feel that you may not be able to stop? (defined as eating much more than most people would under the same circumstances and feeling that eating is out of control)

- Yes
- No
- (If yes) Please estimate how many times:

- a. 1-2
- b. 3-10
- c. More than 10 times

Dutch Eating Behaviors Questionnaire, Restraint Subscale (DEBQ, Van Strien, et al., 1986 – Study 2)

For measure, see:

- van Strien, T. van, Frijters, J. E. R., Bergers, G. P. A., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders*, 5(2), 295–315.  
[https://doi.org/10.1002/1098-108X\(198602\)5:2<295::AID-EAT2260050209>3.0.CO;2-T](https://doi.org/10.1002/1098-108X(198602)5:2<295::AID-EAT2260050209>3.0.CO;2-T)

Bulimia Test-Revised, Purging Items (BULIT-R, Thelen et al., 1991 – Study 2)

For measure, see:

Thelen, M. H., Farmer, J., Wonderlich, S., & Smith, M. (1991). A revision of the Bulimia Test: The BULIT—R. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, 3(1), 119–124. <https://doi.org/10.1037/1040-3590.3.1.119>

Items:

5.

18.

20.

25.

27.

31.



Bulimia Test-Revised, Bingeing Items (BULIT-R, Thelen et al., 1991 – Study 2)

Thelen, M. H., Farmer, J., Wonderlich, S., & Smith, M. (1991). A revision of the Bulimia Test: The BULIT—R. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, 3(1), 119–124. <https://doi.org/10.1037/1040-3590.3.1.119>

Items:

2.

8.

9.

16.

21.

23.

30.

34.

Self-Rating Scale (SRS; Hooley et al., 2002 – Studies 1, 2)

For measure, see:

Hooley JM, Ho DT, Slater JA, Lockshin A. Pain insensitivity and self-harming behavior. Paper presented at the annual meeting of the Society for Research in Psychopathology. 2002.

Depressive Experiences Questionnaire – Self-Criticism Subscale (Bagby, Parker, Joffe, & Buis, 1994 scoring – Studies 1, 2; Blatt, 1976)

For measure, see:

Bagby, R.M., Parker, J.D.A., Joffe, R.T., & Buis, T. (1994). Reconstruction and Validation of the Depressive Experiences Questionnaire. *Assessment*, 1(1), 59–68.

<https://doi.org/10.1177/1073191194001001009>

Blatt, S. J. (1974). Levels of object representation in anaclitic and introjective depression. *The Psychoanalytic Study of the Child*, 29, 107–157.

Forms of Self-Criticism/Self-Reassurance Scale (FSCSRS; Gilbert, Clarke, Hempel, Miles, & Irons, 2004 – Studies 1, 2)

For measure, see:

Gilbert, P., Clarke, M., Hempel, S., Miles, J. N. V., & Irons, C. (2004). Criticizing and reassuring oneself: An exploration of forms, styles and reasons in female students. *The British Journal of Clinical Psychology*, 43(Pt 1), 31–50. <https://doi.org/10.1348/014466504772812959>

Depressive Experiences Questionnaire – Short-Form (Rudich, Lerman, Gurevich, Weksler, & Shahar, 2008 – Study 3)

For measure, see:

Rudich, Z., Lerman, S. F., Gurevich, B., Weksler, N., & Shahar, G. (2008). Patients' self-criticism is a stronger predictor of physician's evaluation of prognosis than pain diagnosis or severity in chronic pain patients. *The Journal of Pain: Official Journal of the American Pain Society*, 9(3), 210–216. <https://doi.org/10.1016/j.jpain.2007.10.013>

Automatic Thoughts Questionnaire, Self-Criticism Subscale (ATQ, Hollon & Kendall, 1980)

Please rate how much you experience the following thoughts (in general).

(1 = "not at all," 2 = "sometimes," 3 = "moderately often," 4 = "often," and 5 = "all the time")

- 1) I wish I were a better person.
- 2) What's the matter with me?
- 3) I'm so disappointed in myself?
- 4) I'm a failure.
- 5) I'm a loser.
- 6) Why can't I ever succeed?
- 7) I'm no good.
- 8) I hate myself.
- 9) I'm worthless.

Anagram List (Study 3; Adapted from Aspinwall & Richter, 1999)

Oneci

Amoos

Acelo

Rtean

\*Uoseh

Filru

Pecit

\*Kridn

Lelmo

Afnac

\*Utpni

HaacI

Ebnir

Rmeyc

Iudmo

Hwnec

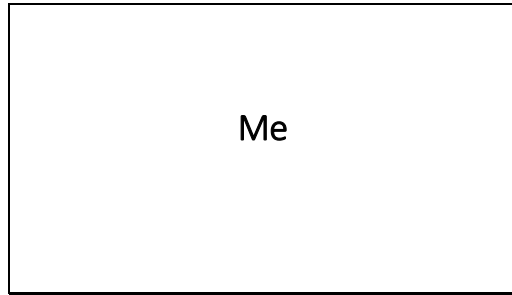
\*Rpwna

\*denotes solvable anagram

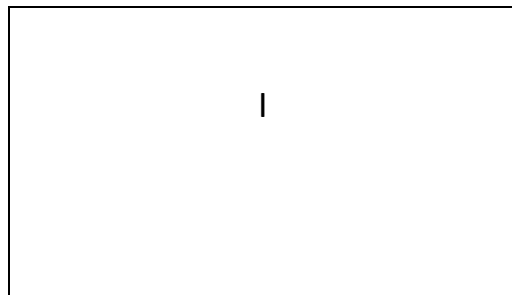
## Affect Misattribution Procedure Stimuli

Affect Misattribution Procedure (AMP) stimuli set

Slide 1



Slide 2





Slide 3

My

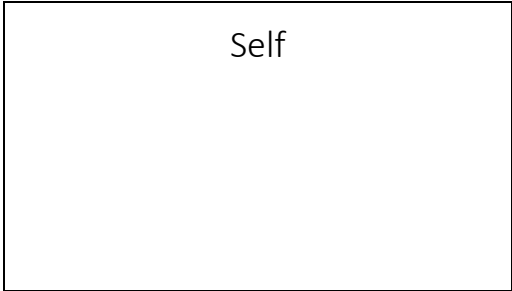
Slide 4

Myself

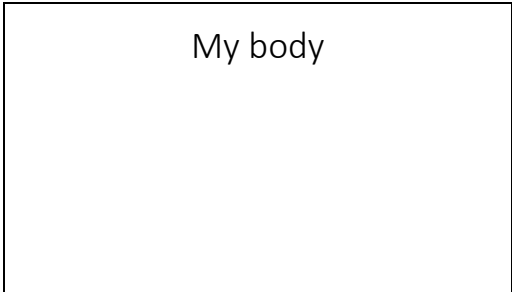
Slide 5

Mine

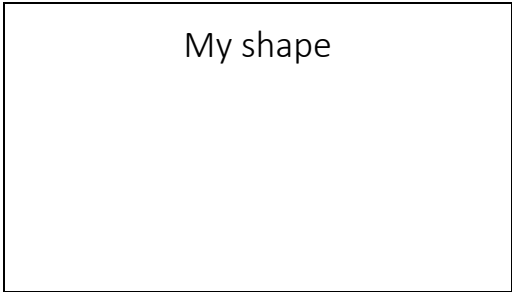
Slide 6



Slide 7



Slide 8



Slide 9

My looks

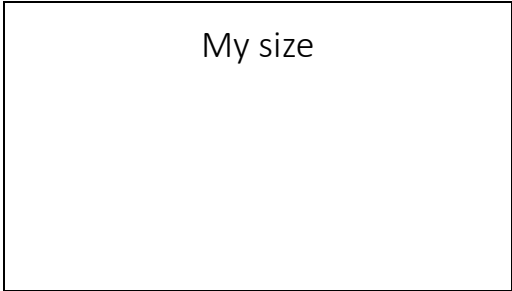
Slide 10

How I look

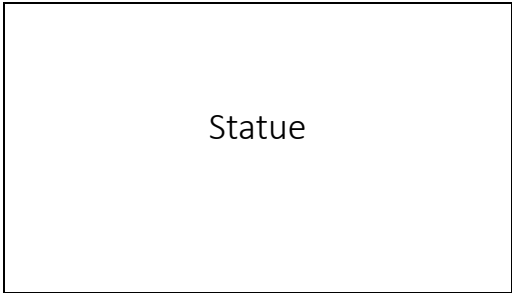
Slide 11

My appearance

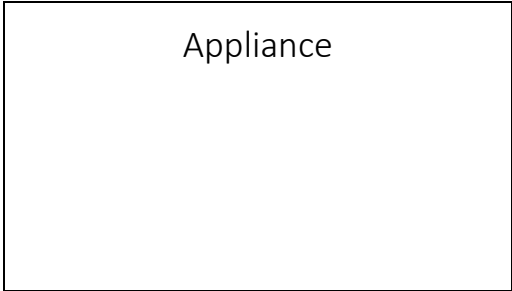
Slide 12



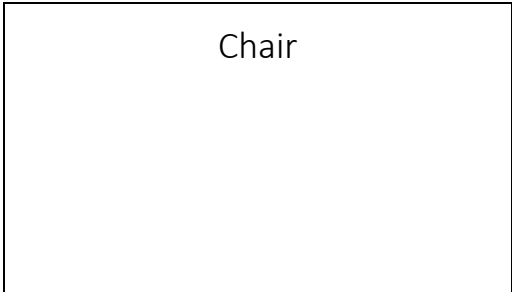
Slide 1



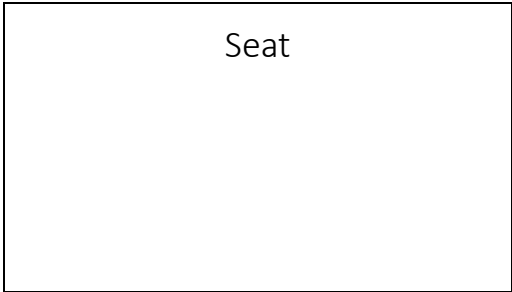
Slide 2



Slide 3



Slide 4



Slide 5

Elbow

Slide 6

Locker

Slide 1

Loneliness

Slide 2

Misery

Slide 3

Jail

Slide 4

Poverty

Slide 5

Unhappy

Slide 6

Betray

Slide 1

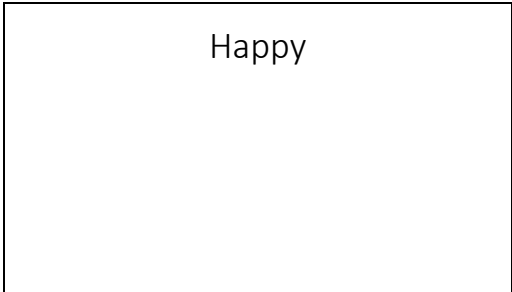
Pleasure



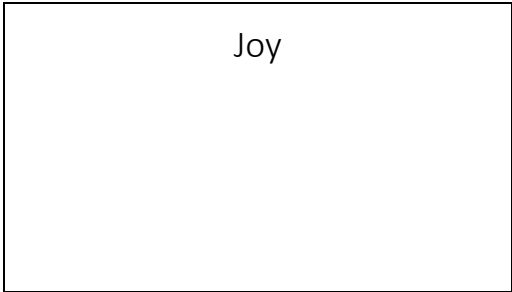
Slide 2



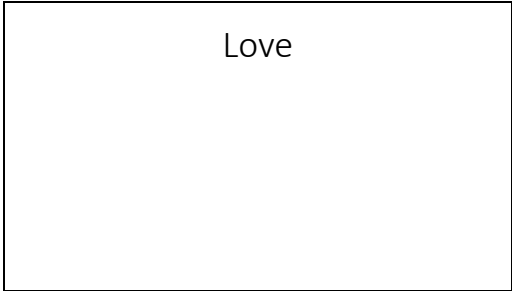
Slide 3



Slide 4



Slide 5



Slide 6

