

Emotional Reactivity and Emotion Regulation Among Adults With a History of Self-Harm: Laboratory Self-Report and Functional MRI Evidence

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Intentionally hurting one's body (deliberate self-harm; DSH) is theorized to be associated with high negative emotional reactivity and poor emotion regulation ability. However, little research has assessed the relationship between these potential risk factors and DSH using laboratory measures. Therefore, we conducted 2 studies using laboratory measures of negative emotional reactivity and emotion regulation ability. Study 1 assessed self-reported negative emotions during a sad film clip (reactivity) and during a sad film clip for which participants were instructed to use reappraisal (regulation). Those with a history of DSH were compared with 2 control groups without a history of DSH matched on key demographics: 1 healthy group low in depression and anxiety symptoms and 1 group matched to the DSH group on depression and anxiety symptoms. Study 2 extended Study 1 by assessing neural responding to negative images (reactivity) and negative images for which participants were instructed to use reappraisal (regulation). Those with a history of DSH were compared with a control group matched to the DSH group on demographics, depression, and anxiety symptoms. Compared with control groups, participants with a history of DSH did not exhibit greater negative emotional reactivity but did exhibit lower ability to regulate emotion with reappraisal (greater self-reported negative emotions in Study 1 and greater amygdala activation in Study 2 during regulation). These results suggest that poor emotion regulation ability, but not necessarily greater negative emotional reactivity, is a correlate of and may be a risk factor for DSH, even when controlling for mood disorder symptoms.

Keywords: self-harm, emotional reactivity, emotion regulation, reappraisal, fMRI

Deliberate self-harm (DSH) refers to behaviors that involve directly and deliberately injuring oneself. In addition to being common in clinical populations (Claes et al., 2010), a large study found that 17% of college students have engaged in DSH (Whitlock, Eckenrode, & Silverman, 2006), suggesting that enhancing understanding of this behavior is urgent. Unfortunately, it is still unclear which factors are reliably associated with DSH. Therefore, the present investigation examined two factors suggested by the-

oretical models to be key risk factors for DSH: heightened negative emotional reactivity and poor emotion regulation ability.

Many genetic, environmental, and psychological factors may put people at risk for DSH (see Nock, 2010, for a review). However, heightened negative emotional reactivity and poor emotion regulation ability have been highlighted as central risk factors (Chapman, Gratz, & Brown, 2006; Linehan, Bohus, & Lynch, 2007; Nock, 2009). Heightened negative emotional reactivity re-

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fers to an intense negative emotional response to a situation (either in magnitude or duration; Linehan et al., 2007). Poor emotion regulation refers to deficits in the ability to alter an emotional response. Research has shown that negative emotional reactivity and emotion regulation are separable processes that differentially predict psychological health (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Gross & John, 2003; Troy, Wilhelm, Shallcross, & Mauss, 2010). However, Chapman and colleagues' (2006) experiential avoidance model, Nock's (2009) integrated model, and Selby and Joiner's (2009) emotional cascade model all suggest that both high negative emotional reactivity and poor emotion regulation contribute to DSH.

Studies that have assessed the relationship between negative emotional reactivity and DSH have yielded inconsistent findings. Several studies using trait measures of negative emotional reactivity found that a DSH group reported greater negative emotional reactivity than a control group (Crowell et al., 2005; Glenn, Blumenthal, Klonsky, & Hajcak, 2011; Gratz & Roemer, 2008); however, other studies using trait measures found no differences between a DSH group and a control group (Gratz, 2006; Gratz & Chapman, 2007), and two studies found no differences between a DSH group and a control group on self-reported negative emotional reactivity to negative images (Glenn et al., 2011; Niedtfeld et al., 2010). Similarly, several laboratory studies found DSH participants to have greater reactivity than controls when indexed with respiratory sinus arrhythmia (Crowell et al., 2005), skin conductance level (Nock & Mendes, 2008), and activation in the amygdala (Niedtfeld et al., 2010). However, other studies found no differences between a DSH group and a control group when reactivity was indexed with skin conductance responses (Crowell et al., 2005, 2012), pre-ejection period (Crowell et al., 2005), or startle response (Franklin et al., 2010; Glenn et al., 2011). Thus, across studies using different measures, there is no consistent support for differences between DSH and control groups in negative emotional reactivity. Given that these measures assess an unknown combination of emotional reactivity and emotion regulation ability, without taking emotion regulation into consideration, it is unclear whether group differences that were observed reflect heightened negative emotional reactivity or, rather, reflect poor emotion regulation.

Findings from studies assessing the relationship between DSH and emotion regulation have been more consistent. In the present investigation, we focused on reappraisal, which involves cognitively reframing a negative situation to reduce its negative impact (Gross & Thompson, 2007). We did so because this form of emotion regulation (a) is particularly well understood (e.g., Gross, 1998), (b) has been assessed using established and validated experimental manipulations (McRae et al., 2010; Troy et al., 2010), and (c) has been specifically theorized to be impaired among those who engage in DSH (Selby & Joiner, 2009). Across several studies, compared with control groups, DSH groups reported less use of reappraisal (Brown, Williams, & Collins, 2007; Hasking et al., 2010; Slee, Garnefski, Spinhoven, & Arensman, 2008). Although there are mixed findings regarding a number of other processes that may contribute to successful emotion regulation (e.g., emotional awareness, clarity, and acceptance; Crowell et al., 2012; Gratz & Roemer, 2008; Slee et al., 2008), existing research has shown a relatively consistent relationship between poor reappraisal and DSH.

In sum, models of DSH suggest that both negative emotional reactivity and emotion regulation ability are risk factors for DSH. However, empirical support for the association between greater negative emotional reactivity and DSH is surprisingly mixed, whereas empirical support for the association between ability to regulate emotion with reappraisal and DSH is relatively consistent. Beyond mixed evidence, the existing research leaves open a number of questions that we addressed with the present investigation.

Limitations of Prior Research

Limitations of existing research make it difficult to evaluate whether high negative emotional reactivity, poor emotion regulation ability, or both are associated with DSH. First, much of the existing research has focused on the strength of negative emotional responding, which can be influenced by heightened emotional reactivity, poor emotion regulation ability, or both (Gross, Sheppes, & Urry, 2011). By separating emotional reactivity and emotion regulation within the same experimental paradigm and controlling for reactivity in tests of regulation and regulation in tests of reactivity, we can clarify whether one or both of these processes are impaired among those with a history of DSH.

In addition, previous research typically compared DSH groups with control groups that were unmatched on important variables such as depression, anxiety, history of borderline personality disorder (BPD), or demographic factors. By comparing a group with a history of DSH with a group of carefully matched controls, we can rule out the possibility that DSH is confounded with another related disorder and, therefore, can make more internally valid claims about the relationships between DSH, negative emotional reactivity, and emotion regulation ability.

Overview of the Present Investigation

In two laboratory studies, we assessed negative emotional reactivity and emotion regulation ability. We focused on participants' ability to use a particularly adaptive form of emotion regulation: reappraisal. Consistent with past research and theorizing, reactivity was operationalized as one's natural, uninstructed response to emotional stimuli; emotion regulation ability was operationalized as one's emotional response when instructed to use reappraisal (reframe a negative situation to reduce its negative impact; see Ochsner, Silvers, & Buhle, 2012). Study 1 assessed self-reported negative emotional reactivity and ability to regulate emotion with reappraisal in response to sad film clips. Study 2 extended Study 1 by assessing self-reported negative emotion and neural indices of negative emotional reactivity and ability to regulate emotion with reappraisal in response to negative images. Those with a history of DSH were compared with control groups matched to the DSH group on demographics, depression, anxiety symptoms, and history of BPD. Across both studies, we hypothesized that, compared with control groups, the DSH group would show (1) either higher or equal negative emotional reactivity and (2) lower emotion regulation ability.

Study 1

Multiple negative emotions are elevated among those who engage in DSH (Nock, Prinstein, & Sterba, 2009); therefore, we

measured a variety of negative emotions (e.g., sadness, anger, anxiety, guilt) in response to sad film clips. Because sadness is a negative emotion that is salient to individuals who engage in DSH (Nock et al., 2009; Selby, Franklin, Carson-Wong, & Rizvi, 2013) and because we used film clips that elicit sadness (Troy et al., 2010), we examined whether DSH would be associated with negative emotions in general as well as sadness in particular. Emotion experience was measured immediately after an emotionally neutral baseline film clip (“negative baseline” and “sadness baseline”), a sad film clip (“negative reactivity” and “sadness reactivity”), a neutral film clip that immediately followed the sad film clip (“negative recovery” and “sadness recovery”), and a sad film clip for which participants were asked to regulate their emotions with reappraisal (“negative regulation” and “sadness regulation”).

Method

Participants. Participants ($N = 148$; 57.4% male) between the ages of 23 and 60 years ($M = 43.7$ years, $SD = 10.4$) were recruited from the Denver metropolitan area through postings on online bulletins for a larger study on emotion.¹ Potential participants were not included in the study if they (a) were hospitalized for emotional reasons, (b) had attempted suicide within the past 6 months, or (c) were below age 20 or above age 60. Twenty-five participants (16.9%) indicated having a history of DSH (henceforth referred to as the DSH group). No one in the study reported having been diagnosed with BPD. Initial group comparisons revealed that participants in the DSH group were younger, had fewer years of education, and had more symptoms of depression and anxiety than those without a history of DSH. Because these factors presented potential confounds, we created two control groups with no history of DSH that were matched to the DSH group by mean level (not by participant) on age, sex, race, education, income, and intelligence. The first control group (“healthy control group”; $n = 37$) included only participants with low depression and anxiety symptoms; the second control group (“depression control group”; $n = 49$) was matched to the DSH group on this group’s mean depression and anxiety symptoms (see Table 1 for descriptive statistics).

Measures.

Demographics. Participants responded to questions about their age, sex, race, education, income, and diagnosis history.

History of DSH. One item (“I have hurt myself on purpose several times”) from the Schedule for Nonadaptive and Adaptive Personality (Simms & Clark, 2006) was used to measure history of DSH. We removed “several times” from this item so that it could be rated on a Likert scale: 1 (*never*), 2 (*occasionally*), 3 (*sometimes*), 4 (*frequently*). Past research has used this item to identify DSH in nonclinical samples (Klonsky, Oltmanns, & Turkheimer, 2003). To ensure that this item was comparable to established measures of DSH, we ran a second study with this single item measure and with the Deliberate Self Harm Inventory (DSHI), a validated and commonly used measure of DSH (see also Study 2). Participants ($N = 87$) were college students (75.9% female) who completed surveys online in exchange for credit. The data revealed good agreement in identification of those with a history of DSH using the two measures ($\kappa = .65$).

Verbal intelligence. Verbal intelligence was measured with the 40-item Vocabulary subscale of the Shipley Institute of Living Scale (Zachary, Crumpton, & Spiegel, 1985).

Depression symptoms. Depression symptoms were measured with the 21-item Beck Depression Inventory (Beck, Steer, & Garbin, 1988). One item regarding suicidality was removed because of institutional review board concerns. The inventory was shown to have good internal consistency ($\alpha = .92$ in the present sample).

Anxiety symptoms. Anxiety symptoms were measured with the 46-item Anxiety Screening Questionnaire (Wittchen & Boyer, 1998). The questionnaire has been shown to have good internal consistency ($\alpha = .95$ in the present sample).

Negative emotional reactivity and emotion regulation task. A standardized laboratory procedure was used to measure negative emotional reactivity and ability to regulate emotion with reappraisal (see also Troy et al., 2010). Participants began by watching a neutral film clip, a video about how to build sandcastles (“sadness baseline” and “negative baseline”). Participants then were instructed to *just watch* Sad Film Clip 1, a scene from the film *Fatal Attraction* (“sadness reactivity” and “negative reactivity”). Participants then watched a second neutral film clip, a video about how to make pottery (“sadness recovery” and “negative recovery”). Participants then watched two more sad film clips, a scene from the film *I Am Sam* and a scene from the film *Kramer Versus Kramer*. To ensure that decreases in negative emotion during the regulation clip were due to regulation instruction rather than habituation, half of the participants were chosen by random assignment to receive instructions to regulate their emotions during Sad Film Clip 2 (Order 1), and the other half during Sad Film Clip 3 (Order 2). Participants in each of the three groups (DSH group, healthy control group, depression control group) were equally likely to be randomized into one of the two orders of instructions, $\chi^2(2) = 0.17, p = .92$, and to have previously seen any of the film clips, $ps > .05$.² For the regulated film clip (“sadness regulation” and “negative regulation”), prerecorded instructions directed the participants to cognitively reappraise by thinking about “unexpected good outcomes.” For the film clip for which participants were not given regulation instructions, they were instructed to “just watch.” After each clip, participants used a 1 (*none at all*) to 9 (*extreme*) scale to indicate the greatest amount of 11 negative emotions (i.e., sadness, anger, anxiety, contempt, frustration, fear, hopelessness, loneliness, guilt, embarrassment, and shame; $\alpha_{\text{baseline}} = .85, \alpha_{\text{reactivity}} = .91, \alpha_{\text{recovery}} = .72, \alpha_{\text{regulation}} = .91$) they felt during each clip.

Procedure. Participants first completed an initial phone screening interview. Next, participants completed questionnaires online to

¹ The data for the present study were derived from the same larger study as those reported in other research (Gruber, Kogan, Quoidbach, & Mauss, 2013; Hopp et al., 2013; Hopp, Troy, & Mauss, 2011; Kogan, Gruber, Shallcross, Ford, & Mauss, 2013; Mauss et al., 2012; Mauss, Troy, & LeBourgeois, 2013; Shallcross, Ford, Floerke, & Mauss, 2013; Troy, Shallcross, Davis, & Mauss, 2012; Troy, Shallcross, & Mauss, 2013). These articles are concerned with variables and questions different from the ones addressed in the present article; therefore, there is no overlap with the present article.

² Within-group analyses showed that there were no differences in emotional responses between people who had seen the film clip and those who had not, $ps > .05$.

Table 1
 Study 1: Participant Characteristics by Group

Variable	DSH (<i>n</i> = 25)	Depression controls (<i>n</i> = 49)	Healthy controls (<i>n</i> = 37)	Statistic	<i>p</i>
Mean (<i>SD</i>) age (years)	39.5 _a (9.3)	43.5 _a (10.2)	42.2 _a (10.9)	$F(2, 108) = 1.27$.29
Sex, (% male)	64.0	57.1	54.1	$\chi^2(2) = 0.61$.74
Race (%)				$\chi^2(10) = 11.34$.33
White	92.0	83.7	77.8		
American Indian/Alaskan Native	0	2.0	0		
Asian	0	0	8.3		
Black	0	2.0	0		
Pacific Islander	0	0	2.8		
Multiple ethnicities	8.0	12.2	11.1		
Mean (<i>SD</i>) education	5.2 _a (1.3)	5.5 _a (1.0)	5.6 _a (0.7)	$F(2, 108) = 1.19$.31
Mean (<i>SD</i>) income	5.0 _a (2.3)	5.4 _a (2.3)	5.8 _a (2.4)	$F(2, 105) = 0.83$.44
Mean (<i>SD</i>) verbal intelligence	33.3 _a (3.9)	33.4 _a (3.6)	33.1 _a (3.9)	$F(2, 108) = 0.07$.94
Mean (<i>SD</i>) depression	15.7 _a (9.9)	15.0 _a (6.1)	3.1 _b (2.8)	$F(2, 108) = 44.30$	<.01
Mean (<i>SD</i>) anxiety	12.4 _a (10.8)	11.8 _a (9.8)	4.0 _b (5.2)	$F(2, 108) = 10.18$	<.01

Note. DSH = deliberate self-harm. Education was measured on a scale from 1 (<7th grade) to 7 (graduate professional training; graduate degree). Income earned per year was measured on a scale from 1 (\leq \$10,000) to 8 (\geq \$100,000). Intelligence was measured with the Vocabulary subscale of the Shipley Institute of Living Scale (0–40; Zachary et al., 1985). Depression was measured with the Beck Depression Inventory (0–60; Beck et al., 1988). Anxiety was measured with the Anxiety Screening Questionnaire (0–46; Wittchen & Boyer, 1998). Means that do not share subscripts differ at $p < .05$.

assess demographics, BPD diagnosis, depression, and anxiety. An average of 8 days after completing the questionnaires ($M = 8.2$ days, $SD = 5.6$), participants came to a laboratory session. Upon completion of the laboratory session, participants were debriefed and paid \$35. Six months after completing the laboratory session ($M = 189.4$ days, $SD = 10.2$), participants completed follow-up questionnaires online. One of these questionnaires assessed history of DSH. Upon completion of these questionnaires, participants were mailed a check for \$20. All procedures were in compliance with the local institutional review board.

Results

Preliminary analyses. To ensure that the emotion inductions reliably increased negative emotions, we used within-group *t* tests to compare emotion during the baseline neutral condition with emotion during each sad film clip that participants “just watched.” Negative emotion and sadness ratings were greater during Sad Film Clip 1 ($M = 4.3$, $SD = 2.0$; $M = 5.9$, $SD = 2.7$, respectively), Sad Film Clip 2 ($M = 3.7$, $SD = 2.0$; $M = 6.0$, $SD = 2.4$, respectively), and Sad Film Clip 3 ($M = 3.2$, $SD = 1.7$; $M = 6.2$, $SD = 2.3$, respectively), than during the baseline neutral condition ($M = 1.5$, $SD = 1.0$; $M = 1.3$, $SD = 0.9$, respectively), $ps < .01$.

To ensure that the emotion regulation instructions reliably reduced negative emotions, we used within-group *t* tests to compare emotion during the regulation condition with emotion during the reactivity condition (Sad Film Clip 1). Negative emotion and sadness ratings were lower during the regulation condition ($M = 3.1$, $SD = 1.5$; $M = 5.3$, $SD = 2.5$, respectively) than the reactivity condition ($M = 4.3$, $SD = 2.0$; $M = 5.9$, $SD = 2.7$, respectively), $ps \leq .03$. Negative reactivity and negative regulation, $r = .62$, $p < .01$, and sadness reactivity and sadness regulation, $r = .40$, $p < .01$, were correlated with one another.

To confirm that participants in each of the three groups were equally likely to follow instructions, participants described what they did to change their emotions, and three reappraisal experts assessed how well each participant followed instructions (1–7

scale; $\alpha = .84$; $M = 6.2$, $SD = 1.2$). The three groups did not differ on the extent to which they followed instructions, $F(2, 99) = 0.05$, $p = .95$. Moreover, self-reported effort (indexed with an item assessing effort exerted during regulation) rated on a scale of 1 (*none*) to 9 (*extreme*) did not differ between groups, $F(2, 107) = 0.08$, $p = .93$: DSH group, $M = 7.0$, $SD = 1.2$; depression control group, $M = 7.1$, $SD = 1.5$; and healthy control group, $M = 6.9$, $SD = 1.9$.

Primary analyses. To test our hypotheses regarding negative emotional reactivity and reappraisal ability, we conducted a series of analyses of covariance (ANCOVAs) with group (DSH group, healthy control group, depression control group) as a between-subjects factor and baseline, reactivity, recovery, and regulation as dependent variables. Given that elevated negative emotion in the regulation condition could be due to either having more negative emotion to regulate (i.e., reactivity) or difficulty regulating the emotions one has (i.e., poor emotion regulation), we entered baseline responding as a covariate in tests of reactivity; baseline and reactivity as covariates in tests of recovery; and baseline, reactivity, and recovery as covariates in tests of regulation for all subsequent tests. Analyses revealed that there was no effect of group on negative baseline or sadness baseline, $F(2, 108) = 0.27$, $p = .77$, $\eta_p^2 < .01$; $F(2, 108) = 0.77$, $p = .47$, $\eta_p^2 = .01$, respectively. There was an effect of group on negative reactivity, $F(2, 107) = 4.44$, $p = .01$, $\eta_p^2 = .08$, but not sadness reactivity, $F(2, 107) = 1.10$, $p = .34$, $\eta_p^2 = .02$. There was no effect of group on negative recovery or sadness recovery, $F(2, 106) = 1.73$, $p = .18$, $\eta_p^2 = .03$; $F(2, 106) = 1.75$, $p = .18$, $\eta_p^2 = .03$, respectively. There was, however, an effect of group on both negative regulation and sadness regulation, $F(2, 105) = 6.17$, $p < .01$, $\eta_p^2 = .11$; $F(2, 105) = 5.39$, $p = .01$, $\eta_p^2 = .09$ (see Table 2 for means).

Follow-up planned comparisons for negative reactivity revealed that the DSH group did not differ from the healthy control group, $F(1, 107) = 1.15$, $p = .29$, $\eta_p^2 = .01$, or the depression control group, $F(1, 107) = 2.21$, $p = .14$, $\eta_p^2 = .02$. The healthy control group exhibited lower negative reactivity than the depression

Table 2
Study 1: Group Means for Responding at Baseline, Reactivity, Recovery, and Regulation

Variable	DSH		Depression control		Healthy control	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Negative baseline	1.4 _a	0.5	1.5 _a	1.0	1.4 _a	1.2
Sadness baseline	1.1 _a	0.4	1.2 _a	0.7	1.4 _a	1.4
Negative reactivity	4.2 _{ab}	1.9	4.9 _a	2.1	3.6 _b	1.7
Sadness reactivity	5.9 _a	2.7	6.2 _a	2.7	5.4 _a	2.8
Negative recovery	1.1 _a	0.3	1.2 _a	0.4	1.1 _a	0.3
Sadness recovery	1.0 _a	0.2	1.2 _a	0.7	1.1 _a	0.3
Negative regulation	3.6 _a	1.7	3.3 _b	1.5	2.3 _b	1.2
Sadness regulation	6.4 _a	2.7	5.4 _b	2.3	4.3 _b	2.4

Note. DSH = deliberate self-harm; Negative = composite of rating of 11 negative emotions. Table means reflect raw scores. Means that do not share subscripts differ at $p < .05$ according to planned comparisons after entering baseline responding as a covariate in tests of reactivity; baseline and reactivity as covariates in tests of recovery; and baseline, reactivity, and recovery as covariates in tests of regulation. The DSH group no longer differs from the depression control group on negative regulation and sadness regulation when covariates are not included.

control group, $F(1, 107) = 8.73, p < .01, \eta_p^2 = .08$. Because regulation was assessed after reactivity and we therefore did not control for regulation in tests of reactivity in primary analyses, we additionally tested whether results regarding reactivity held when controlling for regulation. The same pattern emerged.

Follow-up planned comparisons for negative regulation and sadness regulation revealed that the DSH group exhibited lower regulation ability than the healthy control group, $F(1, 105) = 12.32, p < .01, \eta_p^2 = .11$; $F(1, 105) = 10.75, p < .01, \eta_p^2 = .09$, respectively, and the depression control group, $F(1, 105) = 4.71, p = .03, \eta_p^2 = .04$; $F(1, 105) = 4.60, p = .03, \eta_p^2 = .04$, respectively. The healthy control group did not differ from the depression control group, $F(1, 105) = 2.72, p = .10, \eta_p^2 = .03$; $F(1, 105) = 2.10, p = .15, \eta_p^2 = .02$, respectively. The same pattern emerged when covarying out the effect of age or gender in all analyses. When baseline, reactivity, and recovery were not included as covariates, the DSH group did not differ from the depression control group on negative regulation or sadness regulation, $p = .34$ and $p = .07$, respectively. All other group differences were unchanged.

Additional within-group t tests were used to assess whether each group was successful at regulation. Analyses revealed that the healthy control group exhibited less negative emotion and sadness during regulation compared with reactivity, $t(36) = 7.47, p < .01$; $t(36) = 3.56, p < .01$, respectively, and the depression control group tended to exhibit less negative emotion and sadness during regulation compared with reactivity, $t(48) = 6.16, p < .01$; $t(48) = 1.82, p = .08$, respectively. However, the DSH group did not exhibit less negative emotion or sadness during regulation than reactivity, $t(24) = 1.66, p = .11$; $t(24) = -0.83, p = .42$, respectively. These results suggest that although the control groups reduced negative emotion with reappraisal, the DSH group did not.

Discussion

Compared with individuals in a healthy control group and a depression control group, individuals with a history of DSH did

not show greater negative emotional reactivity during or shortly after a negative emotion induction. However, compared with individuals in both control groups, individuals with a history of DSH tended to exhibit lower ability to regulate emotions with reappraisal. A similar effect emerged when assessing group differences in sadness reactivity and sadness regulation specifically. These findings suggest that poor ability to regulate emotion with reappraisal, but not higher negative emotional reactivity, is related to DSH.

At the same time, a number of questions about the relationships between DSH, negative emotional reactivity, and emotion regulation ability remained unanswered by this study. First, although we used laboratory emotion inductions and assessed current emotion experience, this approach still allowed some self-report biases. Physiological measures could help confirm that those with a history of DSH do indeed differ from controls in emotion regulation ability and that this effect is not due to self-report biases. Second, the measure used to assess DSH consisted of only one item. Using a more comprehensive measure that assesses multiple forms of DSH would ensure that any measurement error inherent in the single-item measure was not responsible for the relationships between DSH, negative emotional reactivity, and emotion regulation ability. Third, Study 1 used film clips that induced primarily sadness. Although these film clips also induced a range of other negative emotions (as evidenced by the ratings), assessing negative emotional reactivity and emotion regulation ability in response to negative emotional stimuli more broadly would ensure that Study 1's findings generalize across negative emotional contexts. To address these limitations and to further clarify whether DSH is associated with poor emotion regulation but not heightened negative emotional reactivity, we conducted a second study.

Study 2

To further test our hypotheses, reduce self-report biases, and assess responding in a different emotional context, we conducted a second study. In Study 2, we measured ratings of negative emotion and neural activation with a focus on a region of the brain (i.e., amygdala) that is involved in emotional reactivity and whose activation can be reduced by successful reappraisal (Ochsner et al., 2012). Responding was measured while participants looked at neutral images ("negative baseline" and "amygdala baseline"), negative images ("negative reactivity" and "amygdala reactivity"), and negative images for which they were instructed to regulate their emotions with reappraisal ("negative regulation" and "amygdala regulation"). We supplemented amygdala region of interest (ROI) analyses with whole-brain analyses. Finally, we assessed ratings of perceived success at regulating emotions with reappraisal after participants completed the task.

Method

Participants. Female participants ($N = 48$) between the ages of 19 and 35 years ($M = 28.0$ years, $SD = 4.2$) were recruited in the Denver metropolitan area through postings on online bulletins for a larger study on stress. Potential participants were not included in the study if they (a) were hospitalized for emotional reasons, (b) had recently attempted suicide, (c) had drug dependency, (d) had past diagnosis of BPD, (e) were below age 18 or above age 35, or

(f) were disqualified for any non-MRI compatible conditions (e.g., metal in body, left-handed). Twenty-one participants (43.8%) indicated a history of DSH. Initial group comparisons revealed that participants in the DSH group did not differ from participants without a history of DSH on demographics (i.e., age, race, ethnicity, education, income, and intelligence) or psychopathology (depression and anxiety; see Table 3 for descriptive statistics). Given that Study 1 supported our strictest hypothesis (that a DSH group would exhibit lower emotion regulation ability compared with a depression control group), we wanted to include the most strict control group possible. Therefore, the remaining 27 participants without a history of DSH served as our strict control group (henceforth referred to as the depression control group).

Measures.

Demographics. Participants responded to questions about their age, race, ethnicity, education, and income.

History of DSH. History of DSH was measured with the DSHI, a validated and commonly used measure consisting of 17 items assessing whether participants have ever engaged in a variety of DSH behaviors. The DSHI has been shown to have good reliability and validity (Gratz, 2001). In the present sample, average number of DSH acts was 69.5 ($SD = 134.2$; range = 1–506), average age of first DSH was 15.9 years ($SD = 3.4$; range 8–23), average duration of engaging in DSH was 4.3 years ($SD = 5.0$; range < 1–20), and average number of hospitalizations for DSH was 0.3 ($SD = 0.5$; range = 0–1).

Verbal intelligence. Verbal intelligence was measured with the Shipley Institute of Living Scale (Zachary et al., 1985).

Depression symptoms. Depression symptoms were measured with the 21-item Beck Depression Inventory ($\alpha = .88$ in the present sample).

Anxiety symptoms. Anxiety symptoms were measured with the 21-item Beck Anxiety Inventory (Fydrich, Dowdall, & Chambless, 1992). The inventory has been shown to have good reliability, validity, and internal consistency ($\alpha = .88$ in the present sample).

Negative emotional reactivity and emotion regulation task. A standardized laboratory paradigm was used to measure negative emotional reactivity and ability to regulate emotion with reappraisal while a blood oxygen level-dependent signal was being acquired (McRae et al., 2010). This procedure used standardized negative images from the International Affective Picture System (Lang, Bradley, & Cuthbert, 2005) to induce negative emotion and standardized neutral images for an emotional baseline. Before beginning, participants were trained to (a) look at, react naturally, and not reappraise to images that followed the instruction to “LOOK” and (b) decrease their negative emotions to images that followed the instruction to “CHANGE.” Examples of reappraisals were provided (e.g., “the situation is not as bad as it first seemed” or “he or she is feeling better now”).³ To ensure that they understood the instructions and that learning effects did not influence results, all participants were required to generate at least two correct reappraisals before advancing.

During the task, neutral and negative images were presented. Neutral images always followed the instruction to “LOOK” (“negative baseline” and “amygdala baseline”). Half of the negative images followed the instruction to “LOOK” (“negative reactivity” and “amygdala reactivity”), and the other half of the negative images followed the instruction to “CHANGE” (“negative regulation” and “amygdala regulation”). Assignment of negative im-

ages to the “LOOK” and “CHANGE” conditions was counterbalanced across participants. For each trial, a fixation cross was presented for a variable duration with an average of 2 s, instruction to “LOOK” or “CHANGE” was presented for 2 s, an image was presented for 7 s, the question “How negative do you feel right now?” and a 4-point emotion rating scale were presented for 3.5 s, and two other unrelated questions were presented for 3.5 s each. Between trials, a slide directing participants to “RELAX” was presented for 4 s. Eighty-four trials (28 trials each of neutral, reactivity, and regulation) were presented for each participant. During the baseline, reactivity, and regulation trials, whole-brain echo-planar images were obtained. To measure perceived regulation success, following completion of the task (outside of the scanner), participants responded to the question “How successful do you think you were at [decreasing your negative emotion]?”

Procedure. Participants first completed an initial phone screening interview to assess eligibility. Next, eligible participants completed questionnaires online. Approximately 20 days after completing questionnaires ($M = 19.5$ days, $SD = 9.8$), participants came to the functional MRI (fMRI) session, which took place at The University of Colorado Health Sciences Center’s Brain Imaging Center. Upon completion of the session, participants were debriefed and paid \$40. All procedures were in compliance with the local institutional review board.

Scan parameters. Data were collected with a 3.0 T GE MR System scanner (General Electric Healthcare, Waukesha, WI) using a standard quadrature head coil. Functional images were acquired by using gradient-echo T2* blood oxygenation level-dependent contrast technique (TR = 2,000 ms, TE = 28 ms, matrix = 64 × 64, field of view = 22 × 22 cm²). Each volume included 32 slices (4-mm thick, no gap). High-resolution spoiled gradient recalled scans were acquired for anatomical normalization. We used a Talairach template in Analysis of Functional Neuroimages for normalization.

fMRI analyses. Standard preprocessing steps were completed using Analysis of Functional Neuroimages. Functional images were corrected for motion across scans using the last functional scan and then automatically coregistered to each subject’s high-resolution anatomical image. Anatomical images were then normalized to a structural template image, and normalization parameters were applied to the functional images. Finally, images were resampled to a resolution of 2 mm × 2 mm × 2 mm and smoothed spatially with a 4-mm filter. We then used a general linear model at the individual level to predict the condition-specific hemodynamic response, modeled with the gamma function, during the picture viewing period (7 s) in each experimental condition. We used a structurally defined ROI approach to examine between-groups differences in average parameter estimates from bilateral amygdala (defined using a normalized template at the group level) while participants looked at neutral (amygdala baseline), negative (amygdala reactivity), and negative images while using reappraisal (amygdala regulation). The average parameter estimates for the amygdala voxels in the anatomical ROI were extracted for each

³ Half of the baseline neutral and reactivity trials were assessed in blocks with the type of regulation discussed here; the other half were distributed throughout blocks for a second type of regulation unrelated to the present investigation.

Table 3
 Study 2: Participant Characteristics by Group

Variable	DSH (<i>n</i> = 21)	Depression controls (<i>n</i> = 27)	Statistic	<i>p</i>
Mean (<i>SD</i>) age (years)	27.5 (4.4)	28.3 (4.1)	$F(1, 46) = 0.43$.52
Ethnicity (%)			$\chi^2(3) = 1.45$.69
Hispanic/Latino	14.3	18.5		
Not Hispanic/Latino	76.2	77.8		
Multiple ethnicities	4.8	3.7		
Decline to answer	4.8	0		
Mean (<i>SD</i>) education	5.5 (0.9)	5.6 (1.0)	$F(1, 46) = 0.18$.68
Mean (<i>SD</i>) income	5.6 (3.2)	5.6 (3.0)	$F(1, 38) < 0.01$.98
Mean (<i>SD</i>) verbal intelligence	32.3 (3.6)	29.7 (4.8)	$F(1, 34) = 3.28$.08
Mean (<i>SD</i>) depression	17.6 (10.0)	14.8 (7.9)	$F(1, 46) = 1.23$.27
Mean (<i>SD</i>) anxiety	16.9 (10.4)	13.5 (7.6)	$F(1, 46) = 1.66$.20

Note. DSH = deliberate self-harm. Education was measured on a scale from 1 (<7th grade) to 7 (graduate professional training; graduate degree). Income was measured with a scale (1–12) representing <\$10,000/year (1) to >\$200,000/year (12). Intelligence was measured with the Vocabulary subscale of the Shipley Institute of Living Scale (0–40; Zachary et al., 1985). Depression was measured with the Beck Depression Inventory (0–63; Beck et al., 1988). Anxiety was measured with the Beck Anxiety Inventory (0–63; Fydrich et al., 1992).

individual using an amygdala mask in MarsBaR (Brett, Anton, Valabregue, & Poline, 2002). To explore further group differences in cognitive control regions (e.g., the prefrontal cortex) during regulation, we supplemented amygdala ROI analyses with whole-brain analyses examining group differences for the look negative > look neutral and decrease negative > look negative contrasts using a two-sample *t* test at the group level.

Results

Preliminary analyses. To ensure that the emotion inductions reliably increased negative emotion and amygdala activation, we used within-group *t* tests to compare responding during the baseline neutral condition with responding during the reactivity condition. Participants exhibited greater negative emotion ($M = 2.6$, $SD = 0.5$) and activation in the left ($M = 0.1$, $SD = 0.1$) and right amygdala ($M = 0.1$, $SD = 0.2$) during the reactivity condition than negative emotion ($M = 1.2$, $SD = 0.2$) and activation in the left ($M = 0.0$, $SD = 0.2$) and right amygdala ($M = 0.0$, $SD = 0.2$) during the neutral condition, $ps < .01$.

To ensure that the emotion regulation instructions reliably reduced negative emotion and amygdala activation, we used within-group *t* tests to compare responding during the regulation condition with responding during the reactivity condition. Participants exhibited less negative emotion ($M = 2.1$, $SD = 0.5$) and activation in the left ($M = 0.0$, $SD = 0.2$) and right amygdala ($M = 0.0$, $SD = 0.2$) during the regulation condition than negative emotion and activation in the left and right amygdala during the reactivity condition $ps < .02$. Negative emotion and left and right amygdala reactivity were correlated to varying degrees with negative emotion and left and right amygdala regulation, $r = .76$, $p < .01$; $r = .22$, $p = .13$; and $r = .33$, $p = .02$, respectively.

To confirm that participants in both groups were equally likely to follow instructions, we examined a qualitative item asking participants to describe what they did to change their emotions. Two experts in reappraisal rated each description based on how well the participant followed instructions (1–3 scale; $\alpha = .71$; $M = 2.9$, $SD = 0.4$). There was no effect of group on likelihood to follow instructions, $F(1, 35) = 0.04$, $p = .56$.

Primary analyses. Following Study 1, we hypothesized that compared with the control group, the DSH group would exhibit lower ability to regulate emotion with reappraisal but not heightened negative emotional reactivity. To test these hypotheses, we ran a series of ANCOVAs with group (DSH group, depression control group) as a between-subjects factor and baseline, reactivity, and regulation as dependent variables. We entered baseline responding as a covariate in tests of reactivity and baseline and reactivity as covariates in tests of regulation.

Analyses of negative emotion showed no effect of group on baseline responding, $F(1, 45) = 0.02$, $p = .88$, $\eta_p^2 < .01$, reactivity, $F(1, 44) = 3.31$, $p = .08$, $\eta_p^2 = .07$, or regulation, $F(1, 43) = 0.08$, $p = .78$, $\eta_p^2 < .01$. Because we did not control for negative regulation in tests of negative reactivity, we additionally tested whether results regarding negative reactivity held when controlling for negative regulation. The same pattern emerged. The same pattern also emerged when covarying out the effect of age and when not including any covariates.

Analyses of left and right amygdala activation revealed no effect of group on baseline responding, $F(1, 46) = 0.03$, $p = .86$, $\eta_p^2 < .01$; $F(1, 46) = 3.38$, $p = .07$, $\eta_p^2 = .07$; or reactivity, $F(1, 45) = 0.48$, $p = .49$, $\eta_p^2 = .01$; $F(1, 45) = 0.07$, $p = .79$, $\eta_p^2 < .01$, respectively. There was an effect of group on left amygdala regulation, $F(1, 44) = 4.74$, $p = .04$, $\eta_p^2 = .10$, but not right amygdala regulation, $F(1, 44) = 1.10$, $p = .30$, $\eta_p^2 = .02$ (see Table 4 for means).⁴ Because we did not control for amygdala regulation in tests of amygdala reactivity, we additionally tested whether results regarding amygdala reactivity held when controlling for amygdala regulation. The same pattern emerged. The same pattern also emerged when covarying out the effect of age and when not including any covariates.

Additional within-group *t* tests were used to assess whether each group was successful at amygdala regulation. Analyses revealed that the depression control group participants exhibited less activation in the left and right amygdala in the regulation condition

⁴ Neither depression nor anxiety symptoms were associated with left or right amygdala regulation or perceived regulation success, $ps > .05$.

Table 4
Study 2: Group Means for Responding at Baseline, Reactivity, and Regulation

Variable	DSH		Depression control	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Negative baseline	1.2 _a	0.2	1.2 _a	0.2
Left amygdala baseline	0.0 _a	0.2	0.0 _a	0.2
Right amygdala baseline	0.0 _a	0.2	-0.1 _a	0.1
Negative reactivity	2.5 _a	0.5	2.7 _a	0.5
Left amygdala reactivity	0.1 _a	0.2	0.1 _a	0.1
Right amygdala reactivity	0.1 _a	0.2	0.1 _a	0.2
Negative regulation	2.0 _a	0.5	2.2 _a	0.5
Left amygdala regulation	0.1 _a	0.2	0.0 _b	0.2
Right amygdala regulation	0.1 _a	0.2	0.0 _a	0.3

Note. Negative = responding to the item “How negative do you feel right now?” Table means reflect raw scores. Means that do not share subscripts differ at $p < .05$ according to planned comparisons after entering baseline responding as a covariate in tests of reactivity; baseline and reactivity as covariates in tests of recovery; and baseline, reactivity, and recovery as covariates in tests of regulation. The same pattern also emerged when not including any covariates. Amygdala values reflect average parameter estimates (beta weights) from the amygdala for each experimental condition.

than they did in the reactivity condition, $t(26) = 3.20$, $p < .01$; $t(26) = 2.11$, $p = .05$, respectively. However, the DSH group participants did not exhibit less activation in the left or right amygdala in the regulation condition than they did in the reactivity condition, $t(20) = 0.80$, $p = .43$; $t(20) = 1.25$, $p = .22$, respectively. This suggests that although the depression control group successfully reduced both left and right amygdala activation with reappraisal, the DSH group did not.

To follow up ROI tests, whole-brain contrasts tested for group differences in the reactivity (look negative > look neutral) and regulation (decrease negative > look negative) comparisons with a voxel-level threshold of $p < .005$ and an extent threshold of 31. The reactivity comparison revealed no group differences. However, several regions including the lateral and midline prefrontal regions were more strongly activated for the regulation contrast (decrease negative > look negative) in the DSH group compared with the control group (see Table 5).

Table 5
Study 2: Group Differences in Whole-Brain Activation (DSH > Depression Controls) for the Reactivity (Look Negative > Look Neutral) and Regulation (Decrease Negative > Look Negative) Comparisons

Region (BA)	Extent	T	X	Y	Z	Hemisphere
Reactivity	–	–	–	–	–	–
Regulation						
Posterior cingulate (31)	106	4.300	3	-45	30	Right
Medial frontal gyrus (8)	101	4.403	-3	27	42	Left
Posterior cingulate (30)	50	3.797	3	-51	16	Right
Midcingulate gyrus (23 _a)	41	4.536	9	-15	30	Right
Middle frontal gyrus (6 _a)	41	3.896	-39	7	44	Left
Uvula	32	4.210	29	-65	-26	Right

Note. DSH = deliberate self-harm; BA = Brodmann’s area; – = no significant findings. Whole-brain voxel threshold of $p < .005$, minimum of 31 voxel clustering. XYZ are in Talairach coordinates. Subscript _a indicates BA > 1 mm from peak.

Analyses of perceived regulation success following completion of the task (outside of the scanner) revealed that there was an effect of group on perceived regulation success, $F(1, 34) = 5.74$, $p = .02$, $\eta_p^2 = .14$, with the DSH group reporting less successful regulation.

Discussion

Compared with the depression control group, the DSH group did not show greater negative emotional reactivity (indexed with negative emotion, amygdala activation, or full brain activation) or lesser decrease in negative emotions using reappraisal; however, the DSH group did report greater activation in the amygdala, medial prefrontal cortex (Brodmann’s areas: 6_a and 8), and cingulate regions (Brodmann’s areas: 23_a, 30, and 31), and less perceived success while regulating emotions with reappraisal. Amygdala activation is thought to index emotional experience (Ochsner et al., 2012), suggesting poor ability to down-regulate negative emotion. Medial prefrontal cortex activation is thought to index use of self-relevant information (Denny, Kober, Wager, & Ochsner, 2012; Northoff et al., 2006), and posterior cingulate activation is thought to index autobiographical memory recall (Maddock, Garrett, & Buonocore, 2003). Heightened activation in these regions suggests that use of self-relevant information may be crucially involved in emotion regulation deficits among those with a history of DSH.

General Discussion

The present studies examined two emotional processes that theories suggest are key risk factors for DSH: high negative emotional reactivity and poor emotion regulation ability. In previous research, conflicting empirical results regarding the relationship between negative emotional reactivity and DSH, reliance on questionnaire and self-report measures, confounding of DSH with elevated depression and anxiety symptoms, and failure to separate negative emotional reactivity from emotion regulation ability made it difficult to evaluate the involvement of negative emotional reactivity and emotion regulation ability in DSH. We addressed these limitations by concurrently examining measures of negative emotional reactivity and emotion regulation ability, using laboratory procedures, using physiological measures, and by comparing

DSH groups with control groups matched on depression and anxiety symptoms. Findings revealed that poor ability to regulate emotion with reappraisal, but not greater negative emotional reactivity, was associated with DSH, even when controlling for depression and anxiety symptoms.

DSH and Negative Emotional Reactivity

Across both studies, DSH was not associated with negative emotional reactivity (and effect sizes were small) regardless of which measures were used (i.e., self-reported emotion experience or neural activation) and regardless of what stimuli were used (i.e., a sad film clip or negative pictures). Despite the difficulty of interpreting null effects, the consistency of these findings and the small effect sizes give some confidence to the conclusion that negative emotional reactivity is not reliably related to DSH.

These findings diverge from some earlier studies that did find greater negative emotional reactivity in DSH groups. One explanation for this divergence is that most measures of reactivity allow responses that reflect an unknown combination of heightened reactivity and poor emotion regulation. For example, some studies that found a relationship between DSH and reactivity measured responding with relatively general questions (e.g., "I experience emotions very strongly"; Nock, Wedig, Holmberg, & Hooley, 2008). Generally feeling strong emotions can result from heightened emotional reactivity, poor emotion regulation ability, or both. Thus, this approach leaves it somewhat unclear whether emotional reactivity or emotion regulation is impaired. In addition, one study that assessed negative emotional reactivity in response to a laboratory frustration induction found that group differences in negative emotional reactivity only emerged after a delay (i.e., well into a 14-min emotion induction; Nock & Mendes, 2008). Because emotion regulation may act on the emotion with some delay (Gross & Thompson, 2007), such group differences may be due to poor emotion regulation rather than elevated negative emotional reactivity. Therefore, it appears to be more likely that negative emotional reactivity is not involved in DSH, is involved only in severe cases of DSH, or is involved only in specific emotional contexts. Future research would benefit from assessing whether DSH is associated with reactivity in specific contexts or for specific cases. However, models of DSH may need to be modified to account for the fact that elevated negative emotional reactivity is not reliably observed in DSH.

DSH and Emotion Regulation Ability

Across two studies, we provided some of the first laboratory evidence to suggest that DSH is associated with reduced emotion regulation ability. Group differences were found for self-reported emotion regulation when assessed with a negative emotion composite as well as for sadness specifically after viewing a sad film clip (Study 1), left amygdala responding after viewing negative images (Study 2), and perceived regulation success after viewing negative images (Study 2).

Notably, no group differences were found for emotion regulation when assessed with self-reported negative emotion after viewing negative images (Study 2). There are a number of possible explanations for the difference in findings across studies. First, Study 1 used 11 questions assessing specific emotions, whereas Study 2 used only one question ("How negative do you feel right

now?"). Single-item measures may be less reliable, or they may measure some emotions more than others (Larsen & Fredrickson, 1999). Second, participants responded on a scale from 1 to 9 in Study 1, whereas they responded on a scale from 1 to 4 in Study 2. The approach used in Study 2 may have limited variability in participants' responses and affected our ability to detect group differences in emotion regulation. These factors may explain why it is not uncommon in tasks like these to find null results for self-reported negative emotion but not for neural measures (e.g., McRae, Ochsner, Mauss, Gabrieli, & Gross, 2008). With these considerations in mind, it appears that the lack of group differences in Study 2 may be a result of the measure we used.

Overall, the present investigation provides crucial empirical support for theories suggesting that poor emotion regulation ability is a correlate of DSH (Chapman et al., 2006; Linehan et al., 2007; Nock, 2009; Selby & Joiner, 2009). Reduced ability to regulate emotions with adaptive types of emotion regulation (such as cognitive reappraisal) may contribute to the intense negative emotions characteristic of those who engage in DSH. Thus, the present investigation is also consistent with theories suggesting that deficits in emotion regulation ability lead people to engage in DSH (Selby & Joiner, 2009). Given that DSH is often used to regulate emotions (see Klonsky, 2007, for a review), diminished ability to regulate emotion with adaptive strategies such as reappraisal may lead these individuals to regulate emotions "by any means necessary," including by deliberately harming themselves.

Implications for Clinical Science

Given that clinical disorders are characterized by groups of symptoms that may not share biological correlates, the National Institute of Mental Health is encouraging researchers to investigate the biological and psychological correlates of single constructs (e.g., DSH) that span multiple disorders (Insel et al., 2010). By taking this approach in the present investigation, we discovered a specific relationship between neural/psychological processes and DSH, even when controlling for mood disorder symptoms. Thus, the present investigation provides support for the suggestion that exploring single constructs that span disorders may more effectively increase understanding of psychopathological constructs. Moreover, with regard to DSH specifically, these results support the inclusion of DSH as a distinct syndrome in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; American Psychiatric Association, 2013).

Limitations and Future Directions

The present investigation addressed multiple limitations of existing research; however, it is not without its own limitations. First, although our investigation took an important first step toward assessing a causal model of DSH, it cannot speak to whether there is a causal relationship between poor emotion regulation ability and DSH. Longitudinal and intervention designs are needed to draw causal conclusions about the role of emotion regulation ability in DSH. Second, we had a smaller sample size than some previous studies on DSH, particularly in Study 2. However, methodological analyses have shown that between 12 and 27 participants per cell is sufficient for fMRI studies (e.g., Desmond & Glover, 2002). Thus, the sample size of 48 (for two cells) in Study 2 meets the requirements and expectations for neuro-

imaging studies. Third, the only emotion regulation strategy we assessed was reappraisal. Although reappraisal is particularly relevant in the present context, assessing other strategies would help clarify whether deficits are specific to reappraisal or extend to other strategies. Fourth, we did not assess mechanisms that may contribute to poor emotion regulation. Exploring these mechanisms, for example, by using connectivity analyses to explore whether prefrontal activation is associated with heightened activity in the amygdala, can further clarify why those with a history of DSH show poor emotion regulation. Fifth, we assessed DSH with a single-item measure in Study 1. However, our research (see Study 1 Measures section) as well as past research has shown that single-item measures of DSH are comparable to the DSHI (the measure we used in Study 2) and yield comparable convergent and discriminant validity (Gratz, 2001). Moreover, by using two different measures, we can be more certain that our results are not an artifact of the particular DSH measure we used.

Concluding Comment

Heightened negative emotional reactivity and poor emotion regulation ability are thought to be important correlates of, and potential risk factors for, DSH. In two laboratory studies, individuals with a history of DSH, compared with control groups, showed diminished ability to regulate emotion with reappraisal, but no evidence was found for heightened negative emotional reactivity. These findings provide some of the first laboratory evidence to suggest that reduced emotion regulation ability is associated with DSH and characterizes DSH more robustly than negative emotional reactivity.

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