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## The role of emotion reactivity and gender in the relationship between psychopathology and self-injurious behavior

Evan M. Kleiman<sup>a,\*</sup>, Brooke Ammerman<sup>a</sup>, Amy E. Look<sup>a</sup>, Mitchell E. Berman<sup>b</sup>, Michael S. McCloskey<sup>a</sup><sup>a</sup> Temple University, Philadelphia, PA, United States<sup>b</sup> Mississippi State University, MS, United States

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### ABSTRACT

The goal of the present study was to examine emotion reactivity, a broad construct that consists of an individual's sensitivity, intensity, and persistence of emotional reactions, as a mediator of the relationship between two types of psychopathology (depression symptoms and borderline personality disorder (BPD) symptoms) and history of self-injurious behavior (non-suicidal self-injury (NSSI) and suicide attempts (SA)). We also examined gender as a potential moderator of this relationship. Participants ( $N = 1914$ ) completed measures of emotion reactivity, psychopathology, and self-injurious behavior. Results using a series of mediated path analyses indicated that emotion reactivity mediated the relationship between (1) depressive symptoms and NSSI in females only, (2) depressive symptoms and SA in females only, and (3) probable BPD diagnosis and NSSI in both genders. Emotion reactivity did not mediate the relationship between probable BPD diagnosis and SA in either gender. Our findings suggest that emotion reactivity is a possible pathway through which depression and self-injurious behavior relate, especially in women. We temper these findings, however, within the context of relatively modest observed effects.

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### 1. Introduction

Self-injurious behavior (SIB), a construct consisting of suicide attempts (SA) and non-suicidal self-injury (NSSI), is a problem of widespread concern. Lifetime prevalence rates of SIB in the United States are estimated at 5% for SA (Kessler, Borges, & Walters, 1999) and 6% for NSSI (Klonsky, 2011). NSSI and SA are considered distinct, but related, constructs. There are several factors that differentiate NSSI and SA. For example, emotion regulation is positively associated with NSSI (Gratz & Roemer, 2008), but negatively associated with SA (Anestis, Bagge, Tull, & Joiner, 2011). Despite evidence differentiating NSSI and SA, there are factors that confer risk to both NSSI and SA, including psychopathology such as depression and borderline personality disorder (BPD) (Zisook, Goff, Sledge, & Shuchter, 1994), female gender (Mościcki, 1994), and emotion reactivity. Emotion reactivity consists of sensitivity, intensity, and persistence of emotional reactions (Nock, Wedig, Holmberg, & Hooley, 2008) that has been related to both SA history (Dour, Cha, & Nock, 2011; Najmi, Wegner, & Nock, 2007) and NSSI (Glenn, Blumenthal, Klonsky, & Hajcak, 2011).

\* Corresponding author. Address: Department of Psychology, Temple University, 1701 N 13th Street, Philadelphia, PA 19122, United States. Tel.: +1 215 359 6148.

E-mail address: [evan.kleiman@temple.edu](mailto:evan.kleiman@temple.edu) (E.M. Kleiman).

In addition to its direct relationship with SIB, emotion reactivity represents a mechanism through which psychopathology and SIB relate. For example, emotion reactivity associated with depression may drive individuals to escape aversive emotional states through NSSI. Supporting this idea, Nock et al. (2008) found that emotion reactivity mediated the relationship between a composite of depression, anxiety, and eating disorders, and SIB. Surprisingly, however, there has been no examination of emotion reactivity in the relationship between specific disorders and SIB. Depression is strongly associated with NSSI (Hoff & Muehlenkamp, 2009) and SA (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006). Thus, it is important to replicate the findings that emotion reactivity mediated the relationship between a composite of psychopathology (in which depression was included) and SIB using a measure of only depressive symptoms.

It is also surprising that there has been no exploration of whether these findings apply to borderline personality disorder (BPD). Engagement in SIB is a core diagnostic feature of BPD (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). There is support for the idea that emotion reactivity plays a role in the BPD-SIB relationship. Emotion regulation deficits are a key aspect of BPD (Rosenthal et al., 2008) and NSSI (Gratz & Roemer, 2008) and emotion reactivity is thought to predispose individuals to poor emotion regulation (Gross, 1998; Nock et al., 2008). Thus, we would expect

that, like other psychopathologies, emotion reactivity would also mediate the relationship between BPD and SIB.

Another variable of interest is gender. We expected that women will have higher levels of psychopathology, emotion reactivity, and SIB than men and that the relationships between the three variables are stronger for women. We expected these differences for several reasons. First, women experience more severe and debilitating symptoms of depression (Hankin & Abramson, 2001; Piccinelli & Wilkinson, 2000) and BPD (Grant et al., 2008). Second, women have higher rates of SIB (Mościcki, 1994). Research in adolescents suggests girls experience stronger emotion reactivity to stress than boys (Hankin, Mermelstein, & Roesch, 2007; Rudolph, 2002). In contrast, in the study by Nock et al. (2008) no gender differences in emotion reactivity emerged. These analyses, however, were at the bivariate (i.e., correlational) level. We posited that gender differences are more relevant when considering the overall mediational framework involving both psychopathology and SIB. Thus, a secondary goal of this study is to examine gender as a moderator of the mediational relationship between psychopathology and SIB. Specifically, we expected that the effect of emotion reactivity on the relationship between psychopathology and SIB will be more pronounced for women than men.

### 1.1. The present study

The present study has several goals. First, we aim to replicate (Nock et al., 2008) findings that emotion reactivity mediates the relationship between depression and SIB. While Nock et al. (2008) used a composite of anxiety, depression, and eating disorders, we used depression symptoms due to its higher relevance to SIB compared to anxiety and eating disorders. Second, we examine emotion reactivity as a mediator in the relationship between BPD and SIB. Finally, we also examine female gender as a moderator of mediated effect of depressive symptoms and BPD on NSSI and SA through emotion reactivity, expecting women to experience a more profound effect than men. While some factors such as distress tolerance have opposite effects on NSSI and SA (i.e., distress tolerance is negatively associated with NSSI but positively associated with SA; Anestis, Knorr, Tull, Lavender, & Gratz, 2013), previous research (Nock et al., 2008) suggests that emotion reactivity is a factor that is positively associated with both NSSI and SA. Thus, given the conflicting evidence as to which factors do or do not differentially predict NSSI and SA, we are not making any specific hypothesis with respect to how emotion reactivity may be differentially related to NSSI and SA.

## 2. Method

### 2.1. Participants and procedure

Participants were 1914 undergraduates (61.4% female,  $M$  age = 21.02,  $SD$  = 3.66, range 17–72) from a large urban university who completed a series of self-report measures as part of a larger, IRB-approved study on a secure website for course credit. Approximately 61% of the sample identified as Caucasian, 14% Asian, 13% African American, and 4% mixed race. Approximately 2% of the sample indicated they preferred not to give their race; the remaining 8% self-identified as “other”.

### 2.2. Materials

#### 2.2.1. Probable BPD diagnoses

The Mclean Screening Instrument for Borderline Personality Disorder (MSI-BPD; Zanarini et al., 2003) is a ten item self-report measure of BPD symptoms. Higher scores equal greater severity

of symptoms. The MSI-BPD demonstrates strong internal consistency and convergent validity with other measures of BPD (Gardner & Qualter, 2009). In the present study, the MSI-BPD demonstrated acceptable internal consistency ( $\alpha$  = .82). Zanarini et al. (2003) find that a cutoff score of seven best distinguishes probable from non-probable diagnoses of BPD. Thus, consistent with Zanarini et al. (2003), we dichotomized the total score on the MSI-BPD so that scores of 7 and above were identified as having clinically elevated BPD symptoms and a probable BPD diagnosis.

#### 2.2.2. Depressive symptoms

The Quick Inventory of Depressive Symptomology (QIDS; Rush et al., 2003) is a sixteen item self-report measure of depressive symptomology. Higher scores equal higher levels of depressive symptoms. The QIDS has strong convergent validity with other measures of depressive symptoms (Rush et al., 2006). In the present study, the QIDS demonstrated high internal consistency ( $\alpha$  = .96). Although scores can be converted to severity level-scores (e.g., mild, moderate, severe), we used the measure as a continuous variable. This is because QIDS severity level scores do not correspond to likelihood of depression diagnosis, unlike the MSI-BPD's cutoff scores that correspond to likelihood of BPD diagnosis.

#### 2.2.3. Emotion reactivity

The Emotion Reactivity Scale (ERS; Nock et al., 2008) is a 21 item self-report measure of emotion reactivity. It includes items that measure the sensitivity, intensity, and duration of emotions. All items are summed to a single scale where higher scores equal higher levels of emotion reactivity. The ERS is reported to have strong internal consistency and convergent validity (Nock et al., 2008). In the present study, we found the ERS to have high internal consistency ( $\alpha$  = .96).

#### 2.2.4. Non-suicidal self-injury history

We used an item from the Forms and Functions of Self Injury Interview (FAFSI; Jenkins, Conner, & Alloy, 2011) for this study that assessed whether or not an individual had ever engaged in at least one incident of NSSI<sup>1</sup> in their lifetime. We coded this item such that 0 = no past NSSI and 1 = past NSSI.

#### 2.2.5. Suicide attempt history

We used the item assessing whether or not an individual had attempted suicide in their lifetime from the Suicidal Behaviors Questionnaire-Revised (SBQ-R; Osman et al., 2001). We coded the item such that 0 = no past attempts and 1 = past suicide attempt.

### 2.3. Analytic strategy

#### 2.3.1. Mediation

We tested four mediated path analyses. The first two models tested emotion reactivity as a mediator of the relationships between (1) depressive symptoms and SA and (2) depressive symptoms and NSSI. The second two models tested emotion reactivity as a mediator of the relationships between (3) BPD symptoms and SA and (4) BPD symptoms and NSSI. Thus the difference between models 1/2 and 3/4 was the use of depressive or BPD symptoms as the predictor variable. The difference between models 1/3 and 2/4 was the use of SA history or NSSI history as the outcome variable. All models used emotion reactivity as the medi-

<sup>1</sup> We also conducted analyses using repeated NSSI (i.e., five or more times in life) vs. non-repeated NSSI (i.e., 0–4 times in life) and analyses using number of times engaging in NSSI. Both sets of analyses had the same interpretation as the analyses reported.

ator variable. We tested all models using Mplus 7.0 (Muthén & Muthén, 2012) with weighted least squares means and variance adjusted (WLSMV) estimation. WLSMV estimation is recommended over traditional maximum likelihood approaches when using a dichotomous or categorical dependent variable because standard errors are found to be calculated more accurately (Beauducel & Herzberg, 2006).

2.3.2. Moderated mediation

We used the DIFFTEST option in Mplus to examine gender as a moderator of these mediated relationships (i.e., moderated mediation). We compared the fit of a model where paths from independent variable to mediator and mediator to dependent variable were constrained to equality between men and women to an unconstrained model where all paths were estimated freely for men and women. These two models are nested within each other, making a differential test of the respective fits possible. DIFFTEST option is a directional chi-square difference test that tested whether the constrained model had poorer fit than the unconstrained model. Thus, a significant chi-square difference test would indicate that the constrained model where both genders were equal had poorer fit than the unconstrained model where both genders were not equal, indicating that the models for males and females differed in either the path from independent variable to mediator or mediator to dependent variable if the direct path from the mediating variable to the outcome variable was significant for one gender but not the other (MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002; Muller, Judd, & Yzerbyt, 2005). If the models were significantly different for males and females, but there was no difference in the path from mediator to outcome, then there would likely be another difference between models (e.g., in the relationship between independent variable and mediator).

2.3.3. Special issues with the MSI-BPD and QIDS

The MSI-BPD and QIDS both contain items that assess SIB. These items could contaminate the relationship between the independent and dependent variables. Thus, we conducted analyses with these items removed. We kept the MSI-BPD cutoff score of 7 because changing the cutoff score to 6 did not change the interpretation of the results.

3. Results

3.1. Preliminary analyses

Means, standard deviations, and intercorrelations for all study variables by gender are displayed in Table 1. To examine differences in study variables by gender, we used *t*-tests for continuous variables and chi-square tests for dichotomous variables. Females had significantly higher levels of depression symptoms ( $t(1912) = 3.12, p < .001$ ) and emotion reactivity ( $t(1912) = 6.73,$

$p < .001$ ) than males. Moreover, females had significantly greater likelihood of having elevated BPD symptoms ( $(\chi^2(1) = 4.18, p < .05)$ , engaged in NSSI ( $\chi^2(1) = 4.39, p < .05$ ), and attempted suicide ( $\chi^2(1) = 4.51, p < .05$ ). The effect sizes for all of these differences were small. When examining the intercorrelations, for females, all study variables were positively correlated among each other. For males, all study variables were positively correlated among each other with the exception of (1) BPD symptom group and NSSI history and (2) emotion reactivity and SA history.

Approximately 14% ( $n = 268$ ) of the sample scored above 7 on the MSI-BPD, indicating clinically elevated BPD symptoms. Approximately 18% of the sample ( $n = 339$ ) had ever engaged in NSSI, and 10% of the sample ( $n = 189$ ) had attempted suicide in their lifetimes. These prevalence rates are in line with other prevalence rates reported in the literature. Whitlock, Eckenrode, and Silverman (2006) report a lifetime NSSI prevalence rate of 17% among college students. Meehan, Lamb, Saltzman, and O'Carroll (1992) report a lifetime SA prevalence rate of 10% among college students. Participants who had clinically elevated levels of BPD symptoms were more likely to have engaged in NSSI ( $\chi^2(1) = 253.47, p < .001$ ) and SA ( $\chi^2(1) = 191.80, p < .001$ ). Among those with elevated levels of BPD symptoms, 63.8% had engaged in NSSI and 32.4% had made a suicide attempt. Among those without elevated levels of BPD symptoms, 23% had engaged in NSSI and 9.6% had ever attempted suicide.

3.2. Model 1: emotion reactivity mediates the depression symptoms–NSSI history relationship

The left side of Fig. 1 shows the model (Model 1) testing the hypothesis that emotion reactivity mediates the depression symptom–NSSI relationship. The constrained model fit the data significantly worse than the unconstrained model suggesting that the models differed by gender. As can be seen in the figure, all direct paths were significant for females. Additionally, all direct paths were significant for males, with the exception of the path from emotion reactivity to NSSI history. The direct path from mediator (emotion reactivity) to outcome (NSSI) was only significant in one group, suggesting a moderation effect. Examination of indirect effects confirmed our hypothesis. The standardized indirect effect of depression symptoms on NSSI through emotion reactivity was significant for females ( $\beta = 0.12, 95\% \text{ CI} = 0.05\text{--}0.21$ ), but not males ( $\beta = 0.10, 95\% \text{ CI} = -0.01\text{--}0.18$ ).

3.3. Model 2: emotion reactivity mediates the depressive symptom–SA history relationship

The right side of Fig. 1 shows the model (Model 2) testing the hypothesis that emotion reactivity mediates the relationship between depression symptoms and SA history. The constrained model fit the data significantly worse than the unconstrained

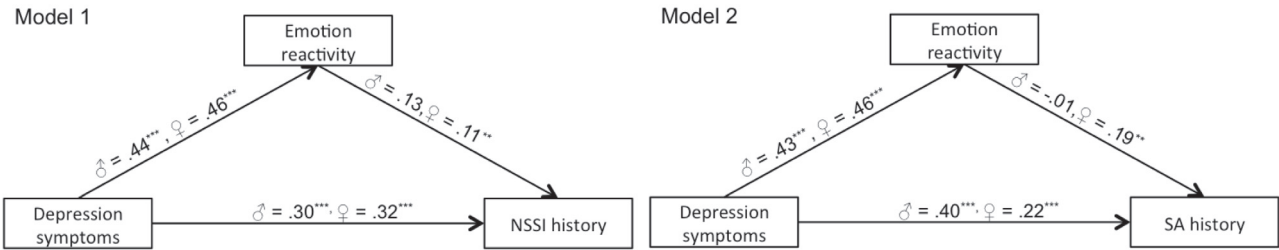
**Table 1**  
Means, standard deviations, and intercorrelations for all study variables by gender.<sup>a</sup>

	1	2	3	4	Male M (SD) n (%)	Female M (SD) n (%)	Effect size
1. BPD symptom group (1 = MSI-BPD ≥ 7)	–	.32***	.35***	.29***	94 (12.7%)	192 (16.4%)	$r = .04$
2. Depression symptoms (QIDS)	.30***	–	.41***	.30***	5.41 (4.41)	6.02 (4.40)	$d = .14$
3. Emotion reactivity (ERS)	.29***	.41***	–	.23***	24.05 (17.07)	29.87 (18.68)	$d = .33$
4. NSSI history	.07	.22***	.18***	–	104 (14.1%)	235 (20.0%)	$r = .05$
5. SA history	.13**	.27***	.08	.19***	60 (8.0%)	129 (11.0%)	$r = .05$

BPD = Borderline Personality Disorder, MSI-BPD = Mclean Screening Instrument for BPD, QIDS = Quick Inventory of Depressive Symptoms, ERS = Emotion Reactivity Scale. Df for *t*-test (female is reference group) = 1912, for chi-square = 1.

<sup>a</sup> Correlations for females are above the diagonal and correlations for males are below the diagonal.

\*\*  $p < .01$ .  
\*\*\*  $p < .001$ .



**Fig. 1.** Emotion reactivity as a mediator of the relationship between depression symptoms and NSSI history (model 1) and SA history (model 2). Note: Standardized regression weights are shown, \*\*\* $p < .001$ , \*\* $p < .01$ , \* $p < .05$ , ♂ = weight for males, ♀ = weight for females.

model ( $\chi^2(3) = 535.37, p < .001$ ). As can be seen in the figure, all direct paths were significant for females. All direct paths were significant for males, with the exception of the path from emotion reactivity to SA. When the direct path from mediator (emotion reactivity) to outcome (SA) is only significant in one group, a moderation effect is present (Muller et al., 2005). This is consistent with the observed correlations, where the correlation between emotion reactivity and SA history was significant for females but not males. Examination of indirect effects confirmed our hypothesis. The standardized indirect effect of depression symptoms on SA through emotion reactivity was significant for females ( $\beta = 0.18$ , 95% CI: 0.08–0.27) but not males ( $\beta = -0.01$ , 95% CI = -0.13–0.11).

#### 3.4. Model 3: emotion reactivity mediates the probable BPD–NSSI history relationship

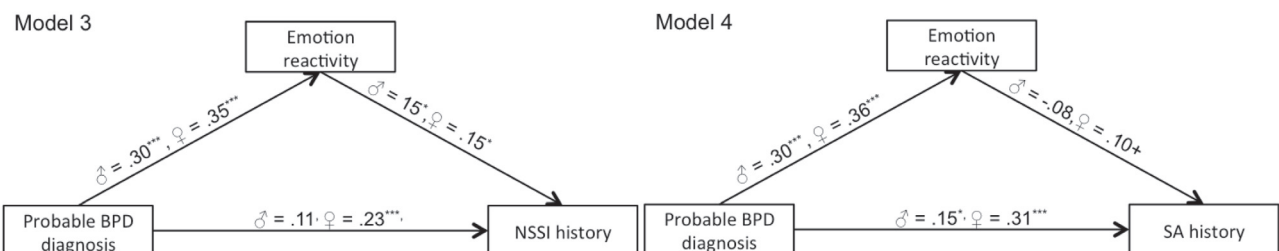
The left side of Fig. 2 shows the model (Model 3) testing the hypothesis that emotion reactivity mediates the relationship between probable BPD diagnosis and NSSI. The constrained model fit the data significantly worse than the unconstrained model ( $\chi^2(3) = 193.14, p < .001$ ). As can be seen in the figure, all direct paths were significant for females. All direct paths were significant for males, with the exception of the path from emotion probable BPD diagnosis to NSSI history. This suggests that emotion reactivity mediated the probable BPD–NSSI relationship in both genders, thus the mediated effect was not moderated. Rather, because the difference in models came from differences significance from independent variable to dependent variable, males and females differed in the type of mediation. The relationship between probable BPD–NSSI relationship was partially mediated by emotion reactivity for females and fully mediated for males. Examination of indirect effects indicated that the standardized indirect effect of probable BPD diagnosis on NSSI through emotion reactivity was significant for both females ( $\beta = 0.15$ , 95% CI = 0.07–0.23) and males ( $\beta = 0.14$ , 95% CI = 0.02–0.24). Thus, in this case, our hypothesis was not fully supported.

#### 3.5. Model 4: emotion reactivity mediates the probable BPD–SA history relationship

The right side of Fig. 2 shows the model (Model 4) testing the hypothesis that emotion reactivity mediates the probable BPD–SA relationship. The constrained model fit the data significantly worse than the unconstrained model ( $\chi^2(3) = 195.27, p < .001$ ). The effects of probable BPD diagnosis on emotion reactivity and SA history were significant for both genders. The effect of emotion reactivity on SA history was not significant for either gender. The standardized indirect effect of probable BPD diagnosis on NSSI through emotion reactivity was significant for neither females ( $\beta = .10$ , 95% CI -0.02–0.22) nor males ( $\beta = 0.07$ , 95% CI = -0.08–0.24). However, there was a near significant trend in the emotion reactivity–SA relationship for females ( $\beta = 0.10, p = .071$ ) and a near significant indirect effect ( $p$  value associated with 95% CI = .081). Thus, in this case, it appears likely that the difference in model fit is due to a nearly significant effect in females.

## 4. Discussion

The goal of this study was to examine emotion reactivity as a mediator between psychopathology (depressive symptoms and borderline personality disorder [BPD] symptoms) and history of self-injurious behavior (SIB; non-suicidal self-injury [NSSI] and suicide attempts [SA]). We intended to replicate previous findings (Nock et al., 2008) with depressive symptoms, and then extend these findings to the study of BPD, as well as examining gender as a moderator of these effects. The results concerning the relationship between depression symptoms and SIB were consistent with our hypotheses. Emotion reactivity mediated the depression symptom–NSSI and depression symptom–SA relationship for females only. These findings suggest that for females, emotion reactivity is a particularly salient path in the route from depression symptoms to SIB. This is consistent with research that females experience greater levels of depression than males (see Piccinelli and



**Fig. 2.** Emotion reactivity as a mediator of the relationship between probable BPD diagnosis and NSSI history (model 3) and SA history (model 4). Note: Standardized regression weights shown, \*\*\* $p < .001$ , \*\* $p < .01$ , \* $p < .05$ , + $p < .10$ , ♂ = weight for males, ♀ = weight for females.

Wilkinson (2000) for a review), beginning as early as age 13 and become marked during early adulthood (Hankin et al., 1998), the age of most of our participants. These findings are also consistent with studies that find females experience greater difficulty regulating emotions compared to men (e.g., McRae, Ochsner, Mauss, Gabrieli, & Gross, 2008).

The results concerning the probable BPD–SIB relationship were less straightforward. While emotion reactivity mediated the probable BPD–NSSI relationship in both genders, there was a difference in the type of mediation effect found. For females, the effect was partially mediated by emotion reactivity, while for males, the effect was fully mediated. This does not suggest moderation, as this would be indicated in a difference between genders in the path from emotion reactivity to NSSI. These results are not entirely in line with our hypotheses. Given that females tend to engage in NSSI because they are feeling sad and males tend to do so because they are feeling angry (Whitlock et al., 2011), it is possible that both sadness and anger could activate the pathway from emotion reactivity to NSSI within the context of BPD. Thus, the differences between males and females within this context are not in emotion reactivity, but in the specific emotions that trigger this pathway.

Finally, emotion reactivity did not mediate the relationship between probable BPD diagnosis and SA history in either gender. There was a non-significant trend for females, however. Thus, our hypothesis was not supported. Results suggest that individuals with BPD might not engage in SA due to emotion reactivity. Rather, there might be other pathways from BPD to SA. One such pathway is impulsivity, given that studies comparing suicide attempters with BPD to suicide attempters with major depression find that impulsivity scores are higher among suicide attempters with BPD compared to those with depression (Soloff, Lynch, Kelly, Malone, & Mann, 2000). Further, because men tend to be more impulsive (Cross, Copping, & Campbell, 2011), we might expect moderation in the sense that the mediated effect would be stronger for men when considering impulsivity.

In summary, two of our hypotheses were supported: emotion reactivity mediated the relationship between depressive symptoms and NSSI and depressive symptoms and SA in women only. One hypothesis was partially supported: emotion reactivity mediated the relationship between probable BPD diagnosis and NSSI history in both genders. One hypothesis was not supported: emotion reactivity did not mediate the relationship between probable BPD diagnosis. Taken together, our results suggest that emotion reactivity plays a role for females only within the context of depression symptoms, but plays a role for both males and females within the context of BPD and NSSI, and has no mediational role in the relationship between BPD and SA.

Two statistical points deserve discussion. First, many of the effects, although significant, were relatively weak compared to other studies of emotion reactivity (e.g., Nock et al., 2008). Our large sample, however, allowed us to detect effects of the size we observed. Across all models, the relationship between emotion reactivity and NSSI/SA were fairly modest, which likely contributed to weak indirect and moderated effects. Interestingly, in other studies (e.g., Nock et al., 2008), the relationship between emotion reactivity and NSSI was considerably stronger than in the present study. This might be a result of our study using an unselected sample and others using a clinical sample where there is likely a greater frequency of NSSI and SA. Thus, we might conclude that emotion reactivity is particularly relevant as a pathway from psychopathology to SIB among individuals with more severe, impairing psychopathology. Even the participants in our study that had the most severe levels of psychopathology still likely exhibited less impairment than those in an inpatient sample, for instance, by definition our participants were all attending college and actively fulfilling course requirements by completing the study. An alternate

explanation for our weak effects is that when the relationship between emotion reactivity and self-injurious behavior is examined in a larger sample, the effects are not as large as would be expected from previous studies, which use considerably smaller samples than ours. A second statistical point is that only one of the models demonstrated complete mediation (i.e., the direct path between psychopathology and NSSI/SA dropped to non-significance with the introduction of emotion reactivity). Such a pattern of results does not necessarily reduce the value of our findings because there are likely multiple pathways to SIB, and emotion reactivity is one such pathway.

Our findings are compatible with other models of SIB. Hamza, Stewart, and Willoughby (2012) propose a model where the relationship between NSSI and SA is mediated by factors such as psychopathology and interpersonal distress. Although we examined NSSI and SA separately (we could not have examined them in the same model because they were measured at the same time), emotion reactivity might be a proximal cause of SA in the relationship between NSSI, depressive symptoms, and SA. Our findings are also further in line with Joiner's et al. (2009) Interpersonal-Psychological Theory of Suicide. Within Joiner's theory, completed suicide requires both the desire and capability to die by suicide. These factors are acquired independently and emotion reactivity may contribute to the capability to die by suicide by exaggerating the need to escape from aversive emotional stimuli, possibly through self-injury (Bresin, 2010). Over time, this exposure to SIB might then lead to the capability to die by suicide, resulting in a suicide attempt.

It is important to discuss the implications of using an undergraduate sample. On one hand, finding observable (albeit small) effects of emotion reactivity in a non-clinical sample could mean that emotion reactivity is an important pathway to SA and NSSI even in typical undergraduate samples. Moreover, the effects of emotion reactivity differ among males and females even in non-clinical samples. On the other hand, given that BPD, depression, NSSI, and SA are all psychopathological processes, it is important to replicate these findings in clinical samples. This is especially important because as discussed earlier, the relatively weak effects we observed in the present study may have been a result of using a sample with a low base rate of psychopathology and self-injurious behavior. Doing so would help refine models of NSSI and SA and suggest avenues for potential intervention. Even in a non-clinical sample, however, our findings suggest that in a clinical setting, targeting emotion reactivity in those at with depression or BPD (especially females) might serve to reduce their risk of escalating from psychopathology to self-injury. There are several other limitations to the study that should be acknowledged. Most notably, the data collected were cross-sectional which did not allow a full test of mediation, but rather only allowed us to infer mediation. Another key limitation was that we used an unselected undergraduate sample and future studies that replicate our findings in clinical samples are needed. A final limitation was the use of self-report measures. Future studies are needed that utilize structured interviews. Strengths of the current study include the use of a large, ethnically diverse sample and NSSI and suicidal behaviors as outcome variables.

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