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April 6, 2017

Psychophysiological Correlates of Non-Suicidal Self-Injury in Adolescent Girls

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Abstract

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Linehan's biosocial theory posits that individuals at risk for developing Borderline Personality Disorder (BPD) have biological vulnerabilities to emotion dysregulation and exposure to invalidating environments, which would exacerbate these vulnerabilities. In this study, we aimed to characterize adolescents at risk for the development of BPD in two ways. First, by characterizing the biological vulnerabilities; and second, by understanding the association between biological vulnerabilities and functions of NSSI. Participants included two groups of mother-daughter dyads, one of which included adolescents at risk for BPD (i.e. adolescents who engage in repetitive self-injury) and the other with healthy control adolescents. We assessed skin conductance responses to maternal invalidation and self-reported functions of NSSI. Results indicated that healthy controls and self-injuring adolescents did not differ in their response to maternal invalidation, although there was some indication for greater sensitivity among self-injuring adolescents, and no significant association between biological vulnerabilities and functions of NSSI. The findings, if replicated in a study that corrects for this study's methodological limitations, suggest the need to reevaluate key components of the biosocial theory.

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Psychophysiological Correlates of Non-Suicidal Self-Injury in Adolescent Girls

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Psychophysiological Correlates of Non-Suicidal Self-Injury in Adolescent Girls

Borderline Personality Disorder (BPD), a chronic and impairing psychological disorder, is characterized by emotional, cognitive, behavioral, and interpersonal dysregulation or instability (Lieb, 2004). The prevailing theoretical account of the development of BPD is Linehan's biosocial theory (1993). According to Linehan (1993), the disorder develops as a result of the transaction between an individual's biological vulnerability to emotion dysregulation and chronic exposure to an invalidating family environment. Emotion dysregulation is premised as the core underlying dysfunction in BPD and characteristics associated with BPD are thought to be consequences of emotion dysregulation (Linehan, 1993). In particular, non-suicidal self-injury (NSSI) – the direct and intentional destruction of one's body tissue in the absence of suicidal intent (Nock, 2010) – is theorized to be a maladaptive mechanism aimed to reduce emotion dysregulation (Linehan, 1993).

The biosocial theory posits that emotion dysregulation is rooted in biologically-based vulnerabilities that characterize individuals at increased risk for BPD. Yet, despite the emphasis on biological vulnerabilities to emotion dysregulation, few published studies report empirical support for these claims in adolescents at risk for BPD. As a critical first step in validating the biosocial theory and understanding emotion dysregulation that characterizes at-risk individuals, we aimed to address this gap. Furthermore, adolescents' engagement in NSSI is reinforced, internally or socially, which relates to the purposes or functions that it serves (Nock & Prinstein, 2004). However, the extent to which functions of NSSI relate to the theorized biological vulnerabilities remains unknown. To characterize adolescents at risk for the development of BPD, the current study addresses two main aims: first, to test support for the theorized biological

vulnerabilities of adolescents engaging in NSSI, and second, to test the extent to which biological vulnerabilities are associated with self-reported functions of NSSI.

Prevalent among female adolescents in particular (Lloyd-Richardson, Perrine, Dierker, & Kelley, 2007; Lundh, Wangby-Lundh, & Bjarehed, 2011), NSSI represents both a diagnostic criterion of BPD (American Psychiatric Association, 2013) and a precursory indicator of risk for the development of BPD (Crowell, Beauchaine, & Linehan, 2009). More than half of female adolescent inpatients with a history of NSSI have a BPD diagnosis (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006). In terms of NSSI as a predictor of BPD, adolescents who engage in NSSI, especially repetitive NSSI, appear to be at elevated risk for developing full-blown BPD (Klonsky & Olino, 2008). In addition, a longitudinal study has demonstrated that, of the nine criteria for the diagnosis of BPD, self-injury or suicidality was the best predictor of the presence of BPD two years later (*Positive Predictive Power* = 0.48; Grilo et al., 2007). NSSI is thus demonstrated to be a useful indicator for discriminating adolescents at risk for BPD. Therefore, we tested our hypotheses on female adolescents who engage in NSSI.

The Biosocial Theory

Linehan's (1993) biosocial theory provides a useful framework for understanding the development of BPD. Linehan proposes that emotion dysregulation is the central dysfunction in individuals with BPD. Given this premise, the characteristics of BPD, such as NSSI, are consequents of emotion dysregulation. In her model, BPD develops from the transactions over time between one's biological vulnerabilities to emotion dysregulation and an invalidating family environment. These biological vulnerabilities are proposed to be characterized by three features: (1) heightened emotional sensitivity, (2) greater emotional reactivity, and (3) slower recovery to pre-arousal levels. Linehan proposed that these biological vulnerabilities are then

exacerbated by an invalidating family environment, one that delegitimizes, undermines, or mocks expressions of private experiences (1993). The transactional association between the individual's biological vulnerabilities and invalidating family environment then unfold in that when confronted with invalidation, the child's negative emotions escalate in intensity, in turn eliciting more invalidation. Thus, a transactional process ensues in which, over time, the child and her environment reciprocally influence each other, ultimately leading to the development of BPD.

The current study aimed to empirically evaluate a key aspect of the theory by assessing the extent to which biological vulnerabilities that characterize emotion dysregulation differentiate adolescents who engage in NSSI from healthy adolescents. We tested hypotheses on physiological indicators of the three proposed features of biological vulnerability to emotional dysregulation characterized by (1) heightened emotional sensitivity, (2) greater emotional reactivity, and (3) slower recovery to pre-arousal levels. Further, we aimed to incorporate the specific invalidating-environment aspect of the biosocial theory by testing maternal invalidation as a stressor to elicit emotional arousal.

Heightened sensitivity. According to Linehan (1993), the first biological vulnerability to emotion dysregulation is heightened sensitivity. An individual who has high sensitivity may be hyper-vigilant to emotional stimuli, have greater tendency to react quickly and have a lower baseline threshold for reacting to stressors compared to those with normal sensitivity (Fruzzetti, Shenk, & Hoffman, 2005). Given the complexity of the construct, heightened sensitivity has been assessed via a range of methods, although more often with self-report measures or behavioral responses to lab-based stressors than with psychophysiological measures.

In terms of self-report, adolescents who engage in NSSI have been shown to score higher on self-report measure of emotional sensitivity, with items such as “I tend to get emotional very easily” ($d = 0.68$; Glenn, Blumenthal, Klonsky, & Hajcak, 2011). At the behavioral level, consistent with the theory, adolescents who engage in NSSI, relative to controls, were likely to abandon a performance-based distress tolerance task earlier ($d = 0.52$), suggesting a lower tolerance to stress and thus greater sensitivity to the demands of the stressor task (Nock & Mendes, 2008). Likewise, a study that assessed automatic information processing using a dot-probe performance task showed that relative to healthy controls, adolescents with elevated BPD features demonstrated an attentional bias towards perceived threat and had trouble disengaging attention from threatening stimuli ($d = 0.93$; Jovev et al., 2012). That is, adolescents at risk for BPD showed heightened attentional vigilance toward threatening stimuli.

In the one published study we found to have tested Linehan’s proposed heightened sensitivity as a psychophysiological construct, Crowell and colleagues (2005) tested the theory with measures of two psychophysiological systems, respiratory sinus arrhythmia (RSA), a measure of the parasympathetic nervous system’s regulatory influences on cardiac activity, and skin conductance responses (SCRs), which reflects the sympathetic nervous system’s skin conductance responses to emotional arousal. They found support for heightened resting arousal among self-injuring adolescent females relative to health controls, reflected by lower RSA ($\eta^2 = .25$), but not SCR ($\eta^2 = .01$). Thus, overall, evidence for Linehan’s theorized heightened sensitivity as a psychophysiological construct has received limited and mixed empirical support from studies of adolescents who engage in NSSI. Thus, our study, like that of Crowell and colleagues (2005), measured sensitivity at rest and improved on the current literature by also

providing a psychophysiological measure of distress tolerance - shorter latency to stress response in the face of an acute stressor.

Emotion reactivity. The second biological vulnerability to emotion dysregulation proposed in Linehan's biosocial theory (1993) is emotion reactivity. In contrast to emotion sensitivity that is identified as low baseline threshold and quicker responses, emotion reactivity is described as the heightened intensity of emotional reactions to a stressor. In support of the theory, adolescents who engage in NSSI behaviors, compared to controls, have been found to self-report higher levels of subjective emotional distress in response to lab-based, standardized stressors ($d = 0.74$; Glenn et al., 2011).

However, the literature regarding psychophysiological measures of emotion reactivity has yielded mixed findings, which varied with the particular psychophysiological system that was measured. In support of emotion reactivity, one study showed exaggerated reactivity in skin conductance levels in response to a performance-based stressor amongst adolescents who engage in NSSI compared to healthy controls ($d = .57$; Nock & Mendes, 2008). However, a study using measures of the neuroendocrine system predicted, and found that adolescents who engage in NSSI had significant hypo-reactivity of cortisol responses to experimentally-induced stress (Trier Social Stress Test, TSST) compared to healthy controls ($d = .75$; Kaess et al., 2012). The hypothesized lack of reactivity reflected in this study may be unique to cortisol and the authors of this study interpreted these findings as poor down-regulation of arousal which is still reflective of emotion reactivity. A third study, which measured RSA and SCR, yielded contradictory findings on physiological characteristics of emotion reactivity in female adolescents (Crowell et al., 2005). Specifically, the study showed that when exposed to a lab-based sad mood induction, female adolescents who engage in NSSI showed significantly reduced RSA ($\eta^2 = .06$), reflecting

heightened arousal, but did not differ in SCR relative to healthy controls ($\eta^2 < .01$). However, this study did not report whether the mood induction stressor was successful in significantly increasing skin conductance arousal from baseline, thereby limiting our ability to conclude that the stressor successfully induced arousal relative to baseline levels. Therefore, SCR measures of baseline may be indistinguishable from that of reactivity, which may explain the lack of group differences. Overall, across these three studies that used a lab-based stressor to elicit a stress response, there was mixed support for the biological vulnerability of emotion reactivity theorized in the biosocial theory (Linehan, 1993). We aimed to improve on this literature and help to resolve the discrepancies by using maternal invalidation, an ecologically-valid stimulus, that is the key stressor in the biosocial theory.

Slow recovery to rest. The third and final biological vulnerability to the development of emotion dysregulation proposed by Linehan (1993) is a slow recovery to rest levels of psychophysiological arousal following an emotionally arousing stressor. Linehan (1993) posited that the slow recovery to pre-stressor arousal levels perpetuate the heightened sensitivity to subsequent emotional stimuli, emphasizing the particular importance of understanding this index of biological vulnerability in the continuation of emotion dysregulation. In support of this component of the biosocial theory, one study showed that relative to healthy controls, adolescents who engage in NSSI retrospectively self-reported longer persistence of emotions ($d = 0.70$; Glenn et al., 2011).

Despite the centrality of this construct to the biosocial theory, we found only one published study that reported having examined recovery to resting levels of psychophysiological arousal following stressors among adolescents who engage in NSSI relative to controls. In that one study, Crowell and colleagues (2005) found that healthy control adolescents and adolescents

who self-injure showed significant differences in RSA ($\eta^2 = .35$) but not in SCR ($\eta^2 = .01$) during the recovery period that followed sad mood induction. This study, however, did not report whether the mood induction stressor elicited significant SCR in the participants compared to baseline levels, which may explain why skin conductance responses during recovery did not significantly differ by group. Thus, support for the biological vulnerability of slower recovery is sparse, relying on one study that yielded mixed support and that one study did not report effects of the stressor necessary to demonstrate that self-injuring adolescents have slow recovery to rest.

Overall, the literature to date provides limited and mixed support for self-injuring adolescents' biological vulnerabilities to emotion dysregulation, as posited in the biosocial theory. However, as the review suggests, the mixed pattern of findings may be due to studies having fallen short of fully testing the theory. With the current study, we built on this literature in two ways. First, emotion sensitivity is nuanced and has two distinct components: lower threshold for reacting to stressors and faster reactions to stressors. Yet researchers assessing physiology have typically relied on only one of these two components. To build on the literature, in the current study, we examined both components of emotion sensitivity within a single physiological system by operationalizing sensitivity as (a) resting arousal and (b) latency to stress response in the face of an acute stressor.

Second, the published empirical studies assessing psychophysiological measures of emotion sensitivity, reactivity and recovery relied on generic stressors such as performance in front of a critical audience or sad mood inductions rather than the key purported stressor in the biosocial theory: invalidating environment. Therefore, the current study added to the literature by examining invalidation as the stressor. Specifically, we studied adolescent girls' responses to invalidating statements by their own mothers as a personalized, ecologically-valid stressor to

assess all three components of the proposed biological vulnerability, with psychophysiological measures of emotion sensitivity, emotion reactivity and slow recovery to rest.

Among psychophysiological systems, we focused on galvanic skin conductance response (SCR) because we aimed to examine differences in arousal rather than differences in regulatory function, the latter of which are best captured by RSA or cortisol. That is, skin conductance measures changes in the sympathetic nervous system, providing a direct, psychophysiological measure of emotional and sympathetic responses rather than an indirect measure of the parasympathetic nervous system's regulatory function, as is the case for RSA.

Invalidating environment. In addition to the biological vulnerabilities to emotion dysregulation proposed by Linehan, the biosocial theory also posits that a biologically vulnerable individual must also be exposed to an invalidating environment in order to enhance risk for BPD to develop. Linehan (1993) describes the invalidating family environment as one where the expression of the youth's emotional experiences are met with intolerance or trivialization. An invalidating environment can exacerbate biological vulnerabilities to emotion dysregulation and it is the transaction between these two factors that, over time, contribute to the development of emotion dysregulation, the central dysfunction in NSSI and BPD.

In addition to serving as an etiological factor in Linehan's transactional model of risk for the development of BPD (1993), invalidation is also proposed to serve as an acute stressor, which would elicit the three features of biological vulnerability. In line with this idea, (Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)(Shenk & Fruzzetti, 2011)college students randomized to receive invalidating feedback from the researcher following a performance-based stressor

showed greater heart rate (*Effect Size* = 1.10) and skin conductance levels (*Effect Size* = .73) compared to students who received validating feedback (Shenk & Fruzzetti, 2011). In support of Linehan's biosocial theory (1993), the study provides a direct link between invalidating feedback and emotional arousal. In the current study, we took a step further by studying invalidation directly from each participant's mother rather than from a stranger.

Functions of NSSI

Our second aim of this study was to understand potential associations between biological vulnerabilities to emotion dysregulation and self-reported functions of NSSI to characterize adolescents at risk for BPD. To better understand NSSI behavior in adolescents, researchers have proposed that NSSI may serve different functions across individuals and/or episodes of NSSI. Taking a functional approach to NSSI, these researchers classify NSSI based on the antecedents, which may precipitate the behavior, and consequents, that likely reinforce the behavior. Utilizing the functional approach has led to the consensus that NSSI behaviors are maintained through two broad functions, intrapersonal and interpersonal functions (Klonsky, 2007; Nock & Prinstein, 2004; Nock, Prinstein, & Sterba, 2009). These two functions have been shown to be significantly related to different BPD symptom clusters in young adults (Sadeh et al., 2014). Specifically, intrapersonal functions of NSSI were significantly related to affect regulation symptoms of BPD ($\beta = .63$), including affective instability, anger, and fear of abandonment, whereas interpersonal functions of NSSI were significantly associated with interpersonal dysregulation symptoms ($\beta = .54$), such as chaotic relationships, paranoia, and identity disturbance. The Sadeh et al. (2014) study illustrates the value of obtaining information about different functions of NSSI because each function is differentially associated with clusters of BPD symptoms. Therefore, in our objective to better characterize adolescents at risk for BPD, we extended the literature on self-

reported functions of NSSI and examined the psychophysiological correlates of functions of NSSI to capture individual differences in adolescents at risk for BPD.

Intrapersonal functions. Intrapersonal functions are the most commonly reported functions of NSSI among adolescents (Klonsky, 2007) and consist of functions to alleviate emotional arousal, to end dissociative feelings, to replace or avoid the desire to commit suicide, to mark internal distress, or to express anger towards oneself. Adolescents who endorse these functions are thought to experience changes in emotional or other internal experiences, from before to after engaging in self-injury, which reinforce NSSI (Nock & Prinstein, 2004).

Given the prevalence of adolescents' endorsement intrapersonal functions of NSSI, it is important to better understand these functions. In particular, given the centrality to the theory of biological vulnerabilities to emotion dysregulation, albeit with mixed empirical support, one key question is the extent to which endorsement of intrapersonal functions is associated with the biological vulnerabilities. Yet we found only two published studies to have examined these associations. In one, Nock and Mendes (2008) found a weak, positive association between greater endorsement of intrapersonal functions and increase in skin conductance reactivity (the second of the three biological vulnerabilities) in response to a lab-based stressor (Spearman's rho = .25). In the second study we found, young female adults with BPD who engaged in NSSI at least once per week showed lower heart rates when administered incisions compared to those who did not receive the incision, suggesting that the injury was associated with reduced distress ($d = .43$; Reitz et al., 2012). Taken together, these two studies support an association between intrapersonal function of NSSI and one of the three biological vulnerabilities - emotion reactivity. However, these studies used either a lab-based stressors to induce arousal or directly caused an injury. An important next step is to test associations between the intrapersonal

function of NSSI and all three biological vulnerabilities and to do so with the specific stressor proposed in Linehan's theory – invalidation. Therefore, in order to assess whether self-reported intrapersonal function of NSSI is reflected in the biological vulnerabilities proposed by Linehan, the current study examined the association between endorsing intrapersonal functions and psychophysiological measures of emotion sensitivity, emotion reactivity and slow recovery to pre-arousal levels in response to invalidating maternal feedback among adolescents who engage in NSSI.

Interpersonal functions. Interpersonal functions of NSSI refer to the self-injuring act that is reinforced by the social environment such as being granted an increase in one's autonomy, being seen as distinct from others, eliciting help from others, or by generating excitement among others. In each of these ways, NSSI is reinforced by changes to the individual's social environment and thus comes to serve that function. As with intrapersonal functions, interpersonal functions are endorsed by adolescents who engage in NSSI albeit to a lesser extent (Klonsky, 2007). However, we found no published studies that examined the associations between the three biological vulnerabilities to emotion dysregulation and interpersonal functions of NSSI among adolescents who self-injure.

Despite the absence of published studies of associations between biological vulnerabilities and interpersonal functions of NSSI, a few studies have shown support for self-reported correlates of NSSI that are consistent with interpersonal functions of NSSI. Although studying young adults rather than adolescents, one such study (Muehlenkamp, Brausch, Quigley, & Whitlock, 2013) showed that repeatedly engaging in NSSI was associated with perceived lower quality support from others compared to healthy controls ($\eta^2 = .09$). In a more direct evaluation of interpersonal function of NSSI, Hilt, Nock, Lloyd-Richardson, and Prinstein (2008)

found that young adolescents perceived a significant improvement in their relationship with their fathers after engaging in NSSI behavior. The longitudinal nature of this study suggests that adolescents may perceive NSSI to be effective in eliciting caretaking from parents, which likely serves as reinforcement of NSSI. These studies suggest that endorsement of interpersonal functions of NSSI is associated with perceived deficits in social environment and resultant perceived improvements in the environment, consistent with the idea of interpersonal functions. However, we found no published studies of associations between interpersonal functions and self-injuring adolescents' psychophysiology. Specifically, it is not known whether interpersonal functions of NSSI are reflected in the biological vulnerabilities proposed in the biosocial theory (Linehan, 1993). Thus, to our knowledge the current study provides the first examination of the association between interpersonal functions of NSSI and the biological vulnerabilities.

The dearth in the literature regarding the association between functions of NSSI and the biological vulnerabilities, and the importance of understanding such associations for the purpose of characterizing adolescents at risk for BPD, justifies our study. We examined the associations between adolescents' self-reported functions of NSSI and psychophysiological indices of biological vulnerabilities in response to an ecologically valid stressor that reflected the proposed invalidating environmental influence in Linehan's biosocial theory (1993).

Current Study

In the biosocial theory, Linehan (1993) proposes that both biological and environmental vulnerabilities in adolescents place them at risk for engaging in NSSI behavior and, ultimately, the development of BPD. Studies to date provide mixed empirical support for biological vulnerabilities in adolescents at risk for BPD but have yet to examine the biological vulnerabilities of heightened emotion sensitivity, greater reactivity and slower recovery to rest in

response to a maternal invalidating stressor. Therefore, the first aim of the current study was to address this gap in the literature on Linehan's proposed biological vulnerabilities by examining the psychophysiological response to maternal invalidation in self-injuring adolescents compared to same-age control participants.

Furthermore, understanding the extent to which physiological responses to maternal invalidation are associated with endorsement of intra- and inter-personal functions can be helpful in furthering our understanding of individual difference among adolescents who self-injure and thus can possibly lead to different treatment pathways. Therefore, the second aim of the current study was to investigate how the biological responses to maternal invalidation may differ by intrapersonal vs. interpersonal function among those who self-injure.

Specifically, we hypothesized that adolescents who engage in NSSI compared to same-age controls will have (1) heightened sensitivity shown by greater mean number of skin conductance response (SCR) during rest and shorter latency before first SCR once the invalidating stressor begins (*latency onset*) (2) greater reactivity and slower recovery in response to the maternal invalidating feedback stressor measured by greater mean number of SCRs while hearing invalidating feedback and greater mean number of SCRs after invalidating feedback ends. We proposed an exploratory hypothesis that examined the association of intrapersonal and interpersonal functions to biological vulnerabilities. Although our hypothesis is exploratory, the literature review provides some support for directionality. Thus, we hypothesized that (3) emotion sensitivity, measured by mean number of SCRs during rest, and latency onset, would be positively associated with mean scores on intrapersonal and interpersonal functions. We also expected that (4) reactivity and recovery, measured by mean number of SCRs while listening to

invalidating feedback and after hearing invalidating feedback, would be associated with mean scores on intrapersonal and interpersonal functions.

In order to test these hypotheses, we used a correlational, cross-sectional study design with repeated measures of SCR across an emotional challenge protocol. We studied two groups of mother-daughter dyads – the NSSI group that included adolescents who engage in repetitive NSSI and the control group that included healthy adolescents with no history of NSSI. Furthermore, to limit variability associated with age, we restricted our sample age range to 14 to 18-year-old adolescents. This age range is supported by prospective studies that report mean onset of NSSI at age 14 (Yates, Carlson, & Egeland, 2008). We restricted the sample to include only females as studies report higher rates of NSSI among female adolescents (Lundh et al., 2011) and that males tend to report different functions of NSSI (Zetterqvist, Lundh, Dahlstrom, & Svedin, 2013). Finally, we focused on maternal instead of paternal feedback, given that maternal and not paternal maltreatment was associated with engagement in NSSI (Martin et al., 2016), as well as, to limit variability.

Method

Participants

A total of 54 mother-daughter dyads participated in the larger study from which these data were derived. Technical difficulties during data collection, comorbid Axis-I diagnoses and/or a single instance of NSSI led to the exclusion of 15 dyads and resulted in a final sample of 39 mother-daughter dyads. Chi-Squared analyses indicated no significant difference ($p > .05$) in the proportion of adolescent in NSSI or control excluded or included. Of the included female adolescent participants, 16 had a history of NSSI and 23 were healthy controls. *T*-tests and chi-

squared tests revealed that the two groups did not differ significantly on age or ethnicity. Likewise, chi-squared analyses showed that the adolescents' mother's ethnicity, SES, employment status and marital status did not differ significantly by group, although one household from both groups reported high household incomes. Furthermore, one adolescent in the NSSI group was adopted. Participants were recruited through outpatient mental health clinics (23.1%), a database of community members enrolled for possible research studies (41%), flyers in the community (30.8%), and word of mouth (5.1%).

For all participants, the inclusion criteria were English as the primary language spoken at home and adolescents living at least part-time with their mothers. Exclusion criteria were maternal histories of participation in parenting strategy training or psychotherapy (e.g., Dialectical Behavioral Therapy, parenting training), and adolescents' diagnosis of a developmental disability. For adolescents considered healthy controls, exclusion criteria were a lifetime history of any Axis I disorder, with the exception of Attention Deficit Hyperactivity Disorder, and any lifetime history of NSSI. Chi-Squared analyses indicated that there was no significant difference ($p > .05$) in the proportion of adolescents in each group using stimulant medication. For self-injuring adolescents, use of psychotropic drugs was permitted, as many adolescents engaging in NSSI were prescribed psychotropic medications ($n = 12, 75\%$).

Procedure

All data were collected during one 2.5 hour laboratory visit. Upon arrival, adolescents and their mothers provided written informed assent and consent, respectively. Adolescents completed a structured clinical interview to assess psychiatric diagnoses and history of self-injury while mothers created audio recordings of neutral and invalidating statements. After the interviews and audio recordings were obtained, adolescent participants completed the emotional

challenge protocol during which their skin conductance responses were measured. The laboratory session ended after completion of the protocol, and the participants were debriefed and compensated \$60 for their participation. This procedure was approved by Emory University Institutional Review Board. These procedures were a subset of a larger study and the participants engaged in interactions with their mothers prior to the emotional challenge protocol.

Emotional challenge protocol. The emotional challenge protocol consisted of four segments and skin conductance data were collected continuously throughout the protocol: a 5-minute rest segment (*rest*); a 1.5-minute neutral segment (*neutral*) during which the participant listened to three 30-second neutral statements; a 1.5-minute invalidation segment (*invalidation*) during which the participant listened to the three 30-second invalidating statements; and a 5-minute recovery segment (*recovery*). Three statements were used in the neutral and invalidation segments as this was the minimum amount of data needed to reliably assess psychophysiology (Boucsein et al., 2012). For the duration of the rest and recovery segments, adolescents were instructed to sit quietly and listen to white noise. Furthermore, before the invalidating segment, adolescents were prompted to imagine that they had just experienced a series of stressors commonly endorsed by adolescents, such as problems with school, parents, friends and romantic partners (Stark, Spirito, Williams, & Guevremont, 1989). With those stressors in mind, they were asked to imagine eliciting support from their mothers. All audio recordings were administered through headphones.

Emotional challenge stimuli. The stimuli for the emotional challenge protocol were audio recordings by each adolescent participant's own mother, recorded during the lab visit. While the adolescent was engaged in other aspects of the protocol, each mother was asked to read out loud a total of six statements, with the instruction being to read each statement within 30

seconds, sounding as natural as possible. After several practice readings and feedback from research assistants, each statement was recorded individually.

The six statements consisted of three with neutral content and three with invalidating content (see Appendix A). The neutral statements were adapted from Hooley et al. (2009) and were descriptions of routine situations or recent events, such as the weather. The invalidating statements were based on invalidating responses in the Children's Negative Emotions Scale, a self-report measure assessing parental validation and invalidation (Remmes & Ehrenreich-May, 2014). To ensure that variability across mothers in affective tone of the recordings was not a significant confound, research assistants rated the audio-recorded statements for affective tone using a -3 to +3 scale (negative ratings indicated negative affective tones, 0 ratings indicated neutral affective tones and positive ratings indicated positive affective tones).

Measures and Materials

Clinical Interviews. Clinical interviews with the adolescents were administered by a Masters-level clinician and reviewed with a licensed Doctoral-level clinician to confirm diagnoses. Interviews were conducted without the mother present.

MINI-KID. Adolescents were interviewed using the Mini International, Neuropsychiatric Interview for Children and Adolescents (MINI-KID), a semi-structured diagnostic clinical interview (Sheehan et al., 2010). Presence of mood, anxiety and behavioral disorders was determined to confirm eligibility for the study. The interview has demonstrated good validity from its high concordance with the Schedule for Affective Disorders and Schizophrenia for School Aged Children. It also has been found to have high interrater reliability ($r = .89-.94$) and good test-retest reliability over 1-5 days ($r = .75-1.00$).

L-SASI. To obtain information on frequency of NSSI over the past year and lifetime

number of episodes, adolescents were also interviewed using the Lifetime-Suicide Attempt Self-Injury (L- SASI; formerly the Lifetime Parasuicide Count; Linehan & Comtois, 1996). Obtaining information regarding engagement in self- injury over the past year was assisted by a timeline follow-back methodology, where participants used personally relevant events as anchors in a calendar-based interview. There are no reports of the L-SASI's psychometric properties, however, items of the L-SASI are identical to a longer measure, the Suicide Attempt Self-Injury Interview, which has demonstrated adequate validity and very good inter-rater reliability (Linehan, Comtois, Brown, Heard, & Wagner, 2006).

NSSI Functions. Adolescent non-suicidal self-injury (NSSI) functions were assessed using the Inventory of Statements about Self-Injury (ISAS; Klonsky & Glenn, 2009). The inventory has 39 items that assess thirteen sub-functions of NSSI. Each of the thirteen sub-functions was assessed with three items. Adolescents are asked to identify the statements that are most relevant to their experience of self-injury, on a scale from 0 (*not at all relevant*), 1 (*relevant*) or 2 (*very relevant*). For each of the sub-functions, items were summed resulting in a subscale score ranging from 0 to 6.

In the current study, these thirteen functions were clustered into two overarching functions of intrapersonal (5 of 13 sub-functions) and interpersonal functions (8 of 13 sub-functions) according to factor analyses of these items (Klonsky, Glenn, Styer, Olino, & Washburn, 2015). The scores for intrapersonal and interpersonal functions are obtained as a mean of the summed subscale scores for each function and each score can potentially range from 0 to 6, with higher scores indicating more of the function. The two scores have been shown to have high internal consistencies (Cronbach's alpha = .80 and .87, respectively), good construct validity, based on correlations with clinical and contextual variables (Klonsky & Glenn, 2009),

and good test-retest reliability over one year ($r = .60$; Glenn & Klonsky, 2011). For the current study, internal consistencies for intrapersonal and interpersonal scores were also high, Cronbach's alpha = .94 and .83, respectively.

Psychophysiological measures. Throughout the emotional challenge protocol, skin conductance data were collected with Biopac TSD203 transducers placed on the distal phalanges of the middle and ring fingers of the participant's nondominant hand after applying electrode paste. Data were amplified with a GSR100C amplifier module which was attached to the MP150 acquisition system for Windows (Biopac Systems Inc.). The GSR100C module was set to collect tonic activity with a sampling frequency of 1,000 Hz. Acqknowledge was used to analyze the data and the units of skin conductance were recorded in microseimens (μS). After the data were resampled to 62.5Hz, phasic data were derived from tonic and nonspecific skin conductance responses (SCRs) were identified as fluctuations exceeding a threshold value of $0.05\mu S$.

The number of skin conductance responses for each segment of the emotion challenge protocol (rest, neutral, invalidation, and recovery) was averaged by the number of 30-second epochs within each segment. Only the last minute (last two 30-second epochs) of rest segment was considered for analyses to exclude skin conductance responses (SCRs) that were due to acclimation of the procedure, as done by other papers (e.g., Crowell et al., 2005).

Emotion sensitivity was operationalized in two ways: the mean number of SCRs exhibited during the rest segment and the latency to a SCR (in seconds) during the invalidation segment, with lower scores reflecting a lower threshold to a stress response and thus, higher sensitivity. Emotion reactivity was operationalized as the number of SCRs within the invalidation segment, relative to the neutral segment. Slow recovery was operationalized as the number of SCRs within the rest segment, relative to the invalidation segment.

Planned Analyses

We conducted preliminary analyses (*t*-tests and chi-squared tests) comparing adolescents who engage in NSSI to control participants and comparing the mothers of adolescents in each group on key demographic factors to assess possible confounds of these variables. Descriptive statistics of the variables were also analyzed to assess normality and to identify any potential outliers (mean values greater than ± 3 standard deviations). Analysis of mother's affective tone when reading emotional challenge stimuli statements were conducted to assess whether tone was a confounding variable.

In order to test our first hypothesis that adolescents who engage in NSSI compared to healthy controls would demonstrate greater mean SCRs during rest segment and the time taken for first SCR after invalidating stressor began (*latency onset*), we conducted independent samples *t*-tests on these two measures of emotion sensitivity. Our second hypothesis, that adolescents who engage in NSSI have greater reactivity and slower recovery demonstrated by greater number of mean SCRs in the invalidation and recovery segment, were tested using repeated measures analyses of variance (ANOVA) with group (NSSI status) X time (segment) interactions for mean SCRs. Post-hoc analyses were conducted where appropriate. For our third hypothesis, whether self-injuring adolescents' emotion sensitivity measures (number of mean SCRs at rest and latency onset) positively correlated with intrapersonal or interpersonal functions, we conducted a nonparametric Spearman's rho correlations, given that intrapersonal and interpersonal function scores were not normally distributed. Finally, for our fourth hypothesis that there is an association between intrapersonal and interpersonal functions and emotion reactivity and recovery, we conducted repeated measures analysis of covariance (ANCOVA) to assess whether there is a significant intrapersonal function X time (segment)

interaction for mean SCRs and a significant interpersonal function X time (segment) interaction for mean SCRs.

Results

Preliminary Analyses

T-tests and chi-squared analyses of demographic variables by group showed that there were no significant differences between the NSSI group and healthy control group (Table 1). Descriptive statistics of the variables (Table 2) revealed high skewness and kurtosis (Skewness > 3.0; Kurtosis > 10.0) for skin conductance data. As a result, we followed recommended standards to logarithmically transform skin conductance variables to approximate normal distributions (Hamilton, 1992). After transformation, analyses of outliers revealed a single univariate outlier and following recommended practices (Woodberry, Gallo, & Nock, 2008) the outlier was removed from the dataset. Finally, analysis of mother's affective tone confirmed that the tone in the emotional challenge stimuli did not significantly differ by group and thus did not need to be treated as a confounding variable (all p 's > 0.05).

Means and standard deviations for intrapersonal and interpersonal functions, as shown in Table 2, were low, especially for interpersonal functions. Moreover, the range of scores for interpersonal functions was very restricted, from 0 to 2.3, with only about one-third scoring even above a 1. In contrast, the range of scores for intrapersonal functions was from 0 to 4.8, with all but two participants scoring above 1. As noted in the previous section, intrapersonal and interpersonal function scores were not normally distributed.

Between-Group Comparison of Psychophysiology

Heightened emotional sensitivity. We failed to support our first hypothesis, that

adolescents who engage in NSSI, compared to healthy controls, would demonstrate greater mean SCRs during the rest segment and take less time to react to the invalidating stressor – the two measures of heightened emotion sensitivity. Specifically, first, adolescents who self-injured, relative to healthy controls, did not have statistically significantly greater SCRs during rest, $t(23.3) = -1.44, p = .16$, although there was a large effect size for the differences between the two groups (Cohen's $d = 0.60$). Second, in terms of time to react, adolescents who engage in NSSI ($M = 10.1$), compared to healthy controls ($M = 19.1$) did not significantly differ in time to react to the invalidating stressor, $t(23.4) = 1.72, p = .09$, although, again, there was a large effect size for this group difference (Cohen's $d = 0.71$).

Greater reactivity and slower recovery to pre-arousal levels. We also failed to support our second hypothesis, that adolescents engaging in NSSI would show greater reactivity and slower recovery in response to invalidation, relative to control participants. The Mauchly's Test of Sphericity indicated that the assumption of sphericity had not been violated, $\chi^2(5) = 10.98, p = .052$. The repeated measures ANOVA failed to detect a significant group (NSSI status) X time (segment) interaction for mean SCRs, $F(1, 36) = 0.9, p = 0.35, \eta^2_{\text{partial}} = .02$.

Although not a direct examination of our hypothesis, the repeated measures ANOVA revealed that for the sample as a whole, a statistically significant difference in mean SCR across segments, $F(3, 108) = 7.24, p < 0.001, \eta^2_{\text{partial}} = .17$, was shown. This warranted a post-hoc examination using a Bonferroni correction to reduce Type I error, which revealed that participants demonstrated statistically significant increases in number of mean SCRs per segment from the *rest* to *neutral* segments (Cohen's $d = .62$), and from *rest* to *invalidation* segments (Cohen's $d = .64$), but not from the *neutral* to *invalidation* (Cohen's $d = .104$), or *invalidation* to *recovery* segments (Cohen's $d = .31$; see Figure 1), as predicted.

Functions of NSSI

The third, exploratory, hypothesis that self-injuring adolescents' emotion sensitivity (the first of the three of biological vulnerabilities) would be positively associated with intrapersonal and interpersonal functions was not supported. For intrapersonal functions, although not statistically significant, the correlation between adolescents' endorsement of intrapersonal functions on ISAS and the first index of heightened sensitivity, mean SCRs during rest, yielded a medium size association (Spearman's $\rho = .38, p = .15$), whereas the association between adolescents' endorsement of intrapersonal functions on ISAS and the second index of heightened sensitivity, latency onset after invalidation stressor began, was not statistically significant and yielded a small effect size (Spearman's $\rho = .075, p = .79$). For interpersonal functions, the correlation between adolescents' endorsement of interpersonal functions and mean SCRs during rest was not statistically significant and small (Spearman's $\rho = .21, p = .45$); similarly, the correlation between adolescents' endorsement of interpersonal functions on ISAS and latency onset after invalidation stressor began was not statistically significant and small (Spearman's $\rho = .27, p = .34$).

The fourth, exploratory, that measures of reactivity and recovery, would be associated with mean scores for intrapersonal and interpersonal functions, was not supported. Specifically, the repeated measures ANCOVA with Greenhouse-Geisser correction, $\chi^2(5) = 17.05, p = .005, \epsilon > .71$, failed to detect a significant time (segment) X intrapersonal function interaction for mean SCRs, $F(1.64, 21.3) = 2.36, p = 0.13, \eta^2_{partial} = .15$; and also failed to detect a significant time (segment) X interpersonal function interaction for mean SCRs, $F(1.64, 21.3) = .55, p = .65, \eta^2_{partial} = .041$.

Discussion

In the present study, we provided an empirical test of several components of Linehan's biosocial theory which proposes that individuals at risk for the development of BPD – those who engage in NSSI repeatedly – have the biological vulnerabilities of heightened emotion sensitivity, greater reactivity, and slower recovery to rest in response to a stressor. The study also explored the extent to which self-reported functions of NSSI might be associated with these biological vulnerabilities. We addressed gaps in the literature regarding associations between functions of NSSI and psychophysiology of self-injuring adolescents and by using a personalized, ecologically valid stressor to incorporate the invalidating environmental component of the biosocial theory. With our sample of adolescent girls who did or did not have a history of repeated NSSI, we found no significant difference between those who engaged in NSSI, compared to healthy controls, in psychophysiological measures of heightened sensitivity, greater reactivity or slower recovery to pre-arousal levels in response to maternal invalidating feedback. However, large effect sizes and statistical associations in the predicted directions suggest some support for the biosocial theory regarding some of these biological vulnerabilities. Further, despite theoretical and empirical support for two different functions of NSSI, we found no significant correlations between endorsed intrapersonal or interpersonal functions of NSSI and physiological measures of emotion sensitivity, reactivity or recovery.

Our findings that there were no significant differences between adolescents who engage in NSSI and healthy controls in skin conductance responses during rest and measures of latency onset (*sensitivity*); while hearing neutral and invalidating statements (*reactivity*), and after hearing invalidating statements (*recovery*) fail to support the biological vulnerability component of Linehan's biosocial theory. The lack of statistically significant group differences in our

findings are consistent with studies that do not show significant group differences in skin conductance responses to sad mood induction stimuli in adolescents who engage in NSSI (Crowell et al., 2005).

One possible explanation is that adolescents who engage in NSSI do not have these biological vulnerabilities to the development of BPD. Specific to the second of the three biological vulnerabilities, emotion reactivity, one study showed that it was poor ability to regulate emotions, not negative emotional reactivity, that explained differences between individuals who engage in NSSI and those who do not (Davis et al., 2014). This implies that adolescents who engage in NSSI do not have the biological characteristic of emotional reactivity that is theorized to predispose them to BPD but, rather, that they are lacking in their emotion regulation skills that result in emotion dysregulation. Future studies can address this possibility by directly comparing psychophysiological measures of emotion reactivity to that of emotion regulation.

There are several, additional, possible explanations for our failure to support this hypothesis. First, it is possible that our failure to support the biological vulnerabilities is specific to our having relied on galvanic skin conductance response as our index of biological vulnerability. For example, others provide supportive findings regarding heightened sensitivity, greater reactivity and slower recovery to rest indexed by heart rate variability, another measure of psychophysiology (Crowell et al., 2005). Second, some of our tests, such as our test for heightened sensitivity, indexed by latency to response to invalidating statements, yielded a large effect size despite the lack of statistically significant differences. This suggests that the small sample size may have provided insufficient power to yield support for this hypothesis. Third, our ninety-second invalidating stimuli may have been too short to detect significant group

differences in psychophysiological responses. In support of this concern, Nock and Mendes only reported significant differences between self-injuring adolescents and healthy controls in the eighth minute of a lab-based task. Rumination, a long, brooding cognitive process, has been found to have a moderating effect on negative affect and NSSI (Nicolai, Wielgus, & Mezulis, 2016). Thus, the short duration of the stressor, although providing the minimum amount of time needed in order to reliably measure psychophysiology, may not have allowed for such a cognitive process to occur and thus may not have differentially affected adolescents who engage in NSSI from healthy controls. Future studies should be designed to test these ideas.

Although not hypothesized, when we merged data across the two groups, we found significant differences between the skin conductance responses during rest and while listening to their mother's neutral feedback as well as between rest and while listening to their mother's invalidating feedback. However, there were no significant differences in skin conductance responses while listening to neutral feedback compared to listening to invalidating feedback for both control and self-injuring adolescents. This suggests that it was hearing the mother's voices and not necessarily their invalidating feedback that elicited skin conductance responses in adolescents. That is, the invalidating feedback did not elicit the expected stress responses. The effect of the invalidating feedback on the adolescents may have been dampened by the use of scripts, which may not reflect how mothers typically respond. However, this script based approach has the benefit of having been derived from work of Hooley and colleagues (2009), which allowed us to create stressors that were personalized (the voice of each adolescent's mother) while also being standardized. On the other hand, Hooley and colleagues (2009) allowed for guided but unscripted feedback from mothers to assess effects on depressed girls and found significant differences between depressed and non-depressed girls. Therefore, assessing the

psychophysiology during a guided but unscripted, face-to-face interaction, using a dynamic systems approach such as in Crowell and colleague's most recent study (2017), can be applied specifically to invalidating feedback that would emphasize the reciprocal driving influence of mother's invalidation and daughters' responses, which may better capture the transactional process.

Our final two hypotheses assessed the possible associations between self-reported intrapersonal and interpersonal functions of NSSI and the three biological vulnerabilities to emotion dysregulation proposed in Linehan's biosocial theory. Contrary to our hypotheses, there were no statistically significant associations between self-reported intrapersonal or interpersonal function of NSSI and our indices of the three biological vulnerabilities (although one of the associations, between intrapersonal function and heightened emotional sensitivity, yielded a medium effect size). This contradicts prior studies that showed that administering NSSI-proxy incisions lead to reduced psychophysiological arousal (Reitz et al., 2012). Our null findings suggest that psychophysiological responses to an environmental stressor are not associated with either intra- or inter-personal functions of NSSI. Our null finding is also consistent with findings in this and other fields where physiological measures sometimes yield different findings than self-reports. In this field, for example, Nock et al. (2009) found that subjective reports of internal distress (negative affect) were a strong predictor of subsequently engaging in NSSI, a finding consistent with the notion of vulnerability to emotional dysregulation (albeit not biological vulnerability) being associated with functions of NSSI (Nock et al., 2009). On the other hand, our failure to support our hypothesis may have been due to the invalidating feedback not having been sufficiently stressful or inadequacies of the self-report measure of functions of NSSI. Specifically, the scores for interpersonal function was highly skewed and some subscales of this

function yielded a score of 0. Furthermore, the instructions provided for this self-report measure was ambiguous as it did not specify whether adolescents were to reflect on all experiences of self-injury or a single episode of NSSI. Given that all participants had more than one episode of self-injury and that function of NSSI can change by episode (Nock & Prinstein, 2005), the ambiguity of the instructions need to be addressed. Therefore, replication with a longer stressor, a larger sample size, and alternative measures of NSSI functions is warranted.

Limitations and Strengths

In addition to concerns already raised about sample size and length of the stressor, our study findings need to be interpreted within the context of additional limitations. Our sample mainly consisted of non-Hispanic White mother-daughter dyads from a high socioeconomic background with an average annual household income of \$127,865 compared to the Atlanta average annual household income of \$47,527. Despite this constraint, the two groups were matched on income and it is interesting to consider the findings in light of this being such a high-income group of families. However, given these characteristics, our sample limits the generalizability of our findings and future studies should therefore aim to obtain a more diverse sample.

Strengths of our study included how we improved on prior literature by using a stressor that was standardized and ecologically valid. The stressor also incorporated the invalidating environment component of the biosocial theory and, combined with our assessment of the biological vulnerabilities, encompassed all aspects of the biosocial theory to characterize adolescents at risk for the development of BPD.

Clinical implications. If replicated with a longer stressor and larger sample size in a more demographically diverse sample, the findings have clinical implications. Given our overall

lack of support for the biosocial theory, the clinical interventions based on the theory, such as dialectical behavioral therapy, may be misguided. Conversely, the findings would suggest that the success of dialectical behavioral therapy, evidenced in numerous randomized clinical trials (e.g., Fleischhaker et al., 2011) may not be rooted in the components of the biosocial theory from which it was initially derived. Nevertheless, such an interpretation is made cautiously given the size of our sample and other limitations of our study. With the goal of treatment in mind, future studies can assess how current treatment interventions affect emotion sensitivity, reactivity and recovery and whether such interventions can deter the development of BPD.

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Table 1

Demographic Variables of Adolescents and Mothers

Variables	Control (<i>n</i> = 23)	NSSI (<i>n</i> = 16)	Range	Statistic
Mean (<i>SD</i>) age in years				
Adolescent	15.50 (1.34)	16.07 (1.26)	14 -18	<i>t</i> (37) = -1.33
Mother	47.70 (4.55)	47.75 (4.25)	38 - 59	<i>t</i> (37) = - 0.04
Race (%)				
Adolescent				$\chi^2(2) = 2.62$
Caucasian/ White	73.91	93.75		
Multiracial	21.74	6.25		
Other	4.35	0		
Mother				
Caucasian/ White	78.26	87.5		
African-American/ Black	4.35	0		
Asian/ Asian-American	4.35	0		
Other Hispanic/ Latino	4.35	0		
Multiracial	4.35	12.5		
Other	4.35	0		
Mother's Mean (<i>SD</i>) years of schooling	17.26 (2.62)	16.25 (1.24)	12 - 25	<i>t</i> (37) = 1.43
Household Mean (<i>SD</i>) annual income (in thousands, \$)	130 (65)	123 (50)	40 - 250	<i>t</i> (37) = 0.356
Mother's employment status (%)				$\chi^2(3) = 4.77$
Homemaker	34.8	18.8		
Full-time outside of home	34.8	50.0		
Part-time outside of home	30.4	18.8		
Unemployed	0	12.5		
Mother's marital status (%)				$\chi^2(2) = 3.81$
Married	95.7	75		
Living with a partner	0	6.3		
Divorced	4.4	18.8		

Note. NSSI = Non-Suicidal Self-Injury

Table 2

Descriptive Statistics of Variables

Variable	<i>M(SD)</i>	
	Control (<i>n</i> = 23)	NSSI (<i>n</i> = 16)
Mean SCR per segment		
Rest ^a	1.11(2.54)	1.29(1.27)
Neutral	1.93(3.14)	1.67(1.53)
Invalidation	1.77(1.61)	1.92(1.84)
Recovery	1.20(1.05)	1.34(1.44)
Latency Onset	19.08(19.26)	10.11(9.10)
ISAS Scores per Function		
Intrapersonal		3.15(1.45)
Interpersonal		0.75(0.65)

Note. SCR = Skin Conductance Response, ISAS = Inventory of Statements About Self-injury ^a
 Only last minute of Rest considered; Raw values for mean SCR are reported but logarithmic transformations were conducted for Mean SCR.

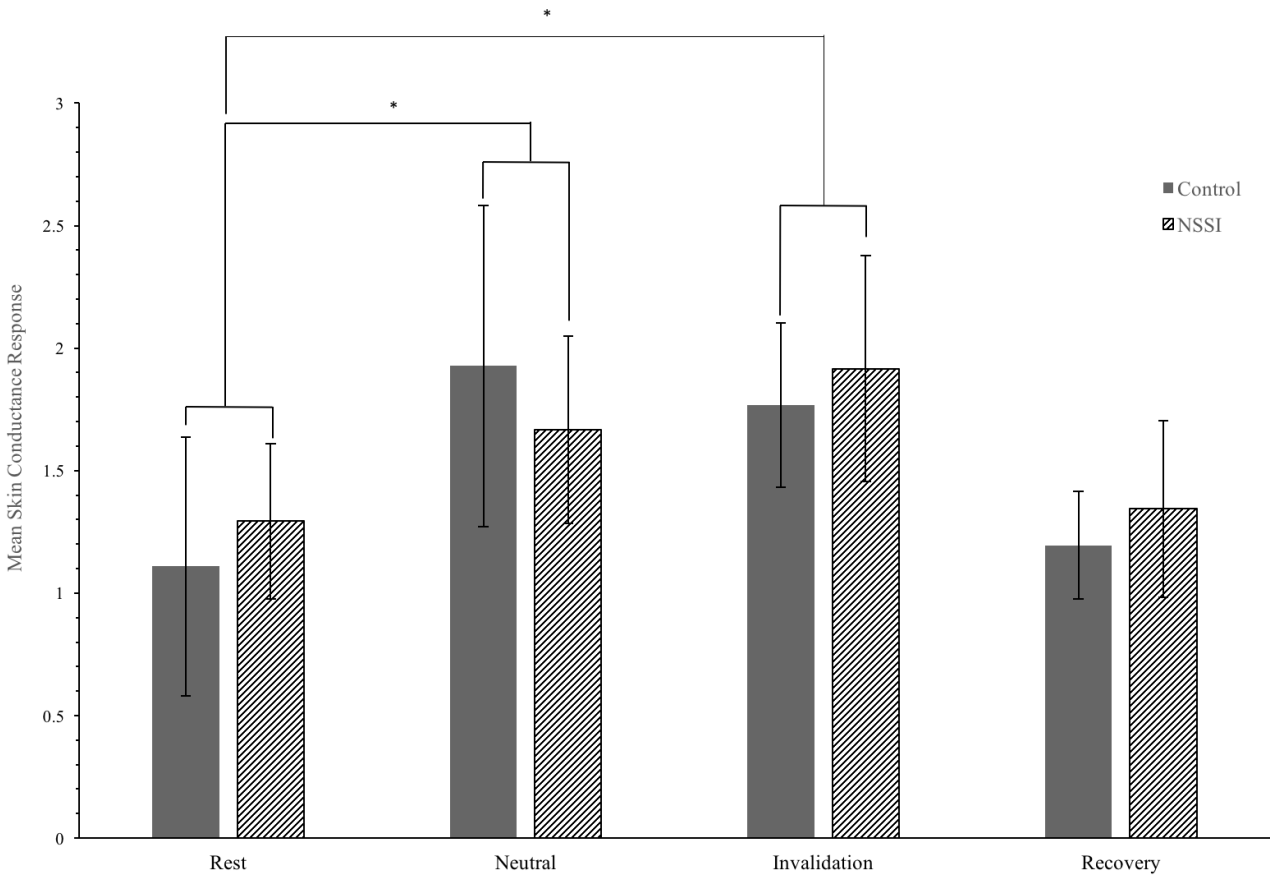


Figure 1. Mean Number of Skin Conductance Responses by Segment.

Note. Raw mean number of skin conductance was used to display figures for clarity and readability. Statistical analyses used logarithmically transformed data. * denotes $p < .05$.

Appendix A

Mother's Script of Neutral and Invalidating Feedback

1. “[Adolescent’s name], let me tell you a little bit about the weather forecast for tomorrow. It should be a really warm day tomorrow, with lots of sunshine in the morning and early afternoon. The forecast says that it should be somewhere in the high 80s. I will need to remember to wear my sunscreen if I’m outside tomorrow. Then, sometime in the late afternoon or early evening, it is supposed to get a little bit cloudy and there is even a chance that it might rain for a little while. I’ll need to pack an umbrella just in case.”
2. “[Adolescent’s name], let me tell you a little bit about the construction project going on close to this clinic. A couple of weeks ago, there was a construction crew that tore down the building that used to be on the lot across the street. Then they shut down half of the street, which caused a lot of traffic to build up for a few weeks. Now they are working on rebuilding the entire building and they are adding on a parking garage. The building will be another medical clinic, with lots of office space and rooms to see patients in.”
3. “[Adolescent’s name], let me tell you a little bit about a news story that I heard on the radio a couple of weeks ago. There is a light bulb in a California firehouse that has been burning for 989,000 hours. That is almost 113 years. It was first installed in the early twentieth century and has rarely been turned off since then. Some people have become very interested in figuring out where the light bulb came from and why it’s lasted so long.”
4. “[Adolescent’s name], you should really not make such a big deal out of this situation. There is no reason for you to be so emotional about this. Your day has really not been so bad; there are lots of people who have it much worse than you do. There is really nothing to be so upset about. You’re blowing things out of proportion; things aren’t as bad as you’re making them out to be. I really think you are over-reacting to all of this and you’re making a big deal about nothing.”
5. “[Adolescent’s name], you need to straighten up and stop sulking. I’m sure the reason that your day went so poorly is probably because you made some really bad decisions and because you didn’t handle yourself better. And then on top of that, now you’ve gotten so emotional that you are out of control and you’re making things worse for yourself. You really need to learn how to handle these kinds of things on your own and deal with them better. If you don’t calm yourself down, there are going to be consequences for your behavior.”

6. “[Adolescent’s name], I really don’t have time to deal with you being upset right now. There probably isn’t anything I can do to help you anyway. And besides, I just don’t understand what you’re getting so upset about. I really don’t know what to tell you- you’re going to have to figure it out and deal with this on your own. I have a lot of my own problems that I need to take care of right now, so I can’t take the time to figure out all of your problems, too. I just don’t have the energy to take care of you right now.”