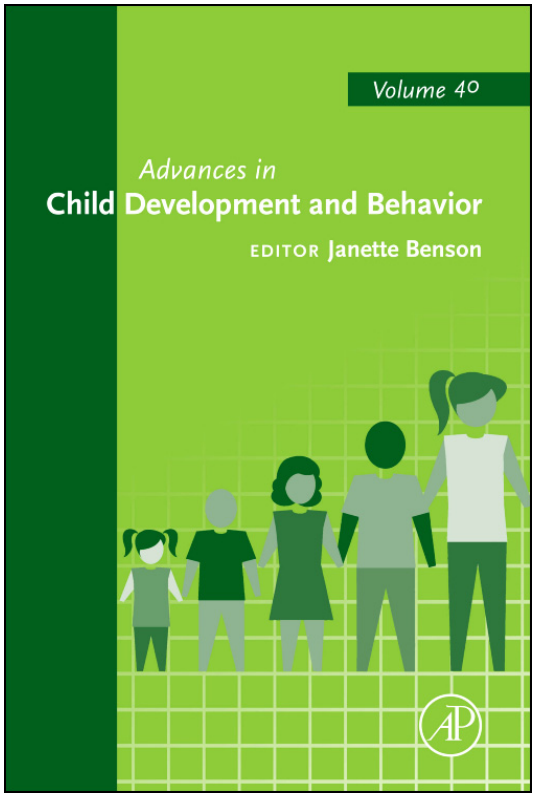

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DEVELOPMENTAL PERSPECTIVES ON VULNERABILITY TO NONSUICIDAL SELF-INJURY IN YOUTH

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Abstract

Nonsuicidal self-injury (NSSI) is defined as intentionally causing bodily harm to oneself without the intent to kill oneself. Recently, there has been an increase in research aimed at understanding why individuals, especially youth and young adults, engage in NSSI. This chapter explores the emergence and maintenance of NSSI from a developmental perspective. Epidemiological research suggests that rates of NSSI increase

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dramatically from early adolescence to young adulthood. No study has investigated NSSI in youth younger than age 10. Current understanding of how emotion and cognitions as well as interpersonal processes play a role in the emergence and maintenance of NSSI is explored. Further, the role of biology (e.g., neurological underpinnings, genetic associations, HPA-axis functioning) on NSSI is explored. Throughout the chapter, particular limitations (e.g., sample selection, measurement issues) in the extant corpus of knowledge are highlighted. Finally, we consider future research directions that may inform developmentally sensitive understanding of the proximal and distal risk factors that may affect the emergence and maintenance of NSSI across the life span.

I. Developmental Perspectives on Nonsuicidal Self-Injury

The study of self-injurious thoughts and behaviors (SITB; see Nock & Favazza, 2009), which includes suicidal and nonsuicidal thoughts and actions, is a serious public health concern that has gained increasing scientific attention. Nonsuicidal self-injury (NSSI), one of the various forms of SITB, is defined as the destruction of one's body without the intent to die. Until recently, much of the knowledge of NSSI came from empirical studies of patient populations and case studies of young adults who reported engaging in NSSI. Not until the past decade have researchers begun to explore NSSI in youth (e.g., Jacobson & Gould, 2007; Klonsky, 2007). Yet despite this recent attention, these reviews do not explore potential theoretically relevant vulnerabilities for the ontogeny of NSSI across different developmental stages and do not address developmental precursors to elucidate why this maladaptive behavior might emerge around adolescence. As this chapter will show, most of the research on NSSI has focused on adults and adolescents with little to no empirical study of NSSI in children. Thus, there is a need for life-span developmental psychopathology research on NSSI.

This chapter aims to appraise the literature on NSSI in youth and to elucidate gaps in this body of literature. Specifically, this chapter will (1) review basic definitions and epidemiology of NSSI in youth (e.g., gender differences in NSSI), (2) explore the functions of NSSI in adolescence and adulthood, (3) review proximal (e.g., psychopathology) and distal (e.g., early childhood maltreatment) risk factors and processes of NSSI, and (4) summarize the interpersonal and intrapersonal risks for the onset and maintenance of NSSI across different developmental stages.

II. Defining the Behavior

NSSI is considered a behavior that individuals engage in, and it is typically repeated. Further, NSSI is both deliberate and direct, in that the act is intentional and meant to physically harm one's skin (Nock & Favazza, 2009). For example, tobacco use leading to lung cancer would not be considered a form of NSSI because the act of using tobacco does not directly harm one's skin and is not necessarily used with the intention to harm oneself. Further, NSSI is a behavior that is not culturally sanctioned. Destruction of body tissue in the form of tattooing and body piercing are culturally acceptable and reflect different motives than NSSI does (Aizenman & Jensen, 2007).

There is currently no psychiatric disorder that captures NSSI in youth in the Diagnostic and Statistical Manual of Mental Disorders, Revised Fourth Edition (DSM-IV-TR; American Psychiatric Association, 2000). Yet, engaging in NSSI is one symptom of borderline personality disorder (BPD), which is characterized by extreme emotional and self-dysregulation, as well as pronounced instability in relationships. Approximately 50–90% of those diagnosed with BPD report engaging in NSSI (see Skodol et al., 2002; Zanarini et al., 2008). However, personality disorders (PDs), such as BPD, cannot be diagnosed in children according to the DSM-IV. By definition, a PD is enduring, and although symptoms of PDs may emerge in youth, these symptoms must prevail into at least early adulthood in order to constitute diagnosis of a PD. Consequently, it is during the adolescent years that patterns associated with PDs tend to emerge and earlier in childhood that formation of interpersonal processes associated with PDs may commence. Although much of the literature on PDs in youth focuses on adolescence, as this time-period is more proximal to early adulthood, it is still relevant to understand the development of symptoms, such as NSSI for BPD, of PDs in both childhood and adolescence.

NSSI varies in severity. Major NSSI is defined as behavior that occurs with low frequency, sometimes even just once, and causes a great deal of physical harm. An example is an individual experiencing an episode of psychosis who cuts off a limb. The rest of this chapter will not focus on major NSSI, as this behavior is infrequent and occurs within the context of nonnormative situations. Additionally, stereotypic NSSI, a behavior that causes minor harm and occurs as frequently as several times a minute, will not be explored in this chapter. This behavior is usually seen in individuals with developmental disabilities and neuropsychological disorders (see Luiselli, 2009).

Research with high-school aged youth reveals that there are many different behavioral methods of NSSI that are utilized (Muehlenkamp & Gutierrez, 2004, 2007). Cutting oneself was reported as the most frequent

method of NSSI (47–48%), followed by scratching (28–36%) and hitting oneself (12–15%). Other NSSI methods include burning, punching or kicking, and banging oneself. More than half (over 60%) of these youth also reported using more than one method of NSSI at some point in their lifetime. To our knowledge, no research has explored potential age and gender differences in NSSI methods in youth samples.

III. Epidemiology of NSSI

A. RATES

Rates of NSSI tend to increase with age across development from childhood through adolescence (see Figure 1). In a community sample of preadolescents (grade 6–8, $N = 508$), 7.7% of youth reported engaging in NSSI behavior (Hilt, Nock, Lloyd-Richardson, & Prinstein, 2008) and 13.9–21.4% of high-school adolescents reported NSSI (e.g., Muehlenkamp & Gutierrez, 2004). In college age samples, rates of NSSI can be as high as 38% (see Rodham & Hawton, 2009). Research with clinical samples

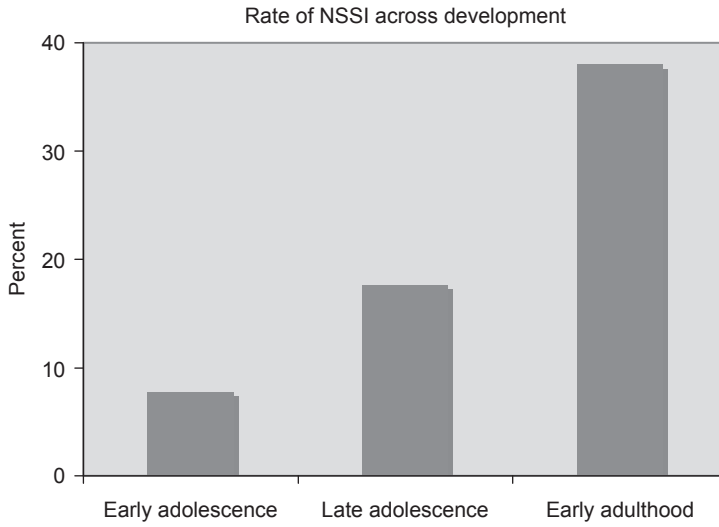

Fig. 1. Epidemiological trends in NSSI. Note*: This figure is meant to convey the likely increase in NSSI across development based on separate studies using multiple measures. Rates for early adolescence are from a study by Hilt et al. (2008), rates for late adolescence are from a study by Muehlenkamp and Gutierrez (2004), and rates for early adulthood are from a study by Rodham and Hawton (2009).*

shows even higher rates, with about 40% of adolescent inpatients engaging in NSSI. Further, among inpatient youth with a history of NSSI, up to 70% report a suicide attempt in their lifetime (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006). Finally, the average age of onset of NSSI is around ages 12–14 (see Rodham & Hawton, 2009).

However, despite these valuable epidemiological data on NSSI, we note two limitations and major gaps in the knowledge base. First, to our knowledge, there is no research exploring rates of NSSI in children younger than middle school (i.e., age 10 or younger). Research with younger populations is necessary to allow for a more complete understanding of the developmental trajectories and risk factors associated with NSSI. Little is known about the developmental course of NSSI. In an exception, one relatively small study Hankin and Abela (2011) used the Functional Assessment of Self-Mutilation (FASM; Lloyd-Richardson, Perrine, Dierker, & Kelley, 2007), a frequently used checklist of NSSI behaviors, as part of a clinical interview across two time-points to assess NSSI in youth. They showed that 8% of early adolescents (ages 11–14) engaged in NSSI, and then 18% reported NSSI at a follow-up 2½ years later (youth now ages 13–17; 14% of youth initiated NSSI for the first time). Of those who engaged in NSSI in early adolescence, moderate continuity was noted such that 50% continued to harm themselves 2½ years later. It is unknown how well youth younger than age 10 would report on NSSI, as this is an area that, to our knowledge, has yet to be researched. In fact, empirical studies assessing NSSI using multiple informants to corroborate engagement in NSSI has not been conducted, and this is an area for future exploration.

B. GENDER

Gender differences in NSSI have also been investigated. Reviews of the NSSI literature report that in both community samples (Laye-Gindhu & Schonert-Reichl, 2005; Muehlenkamp & Gutierrez, 2007; Ross & Heath, 2002) and clinical samples (e.g., Guerry & Prinstein, 2010), girls report up to four times greater rates of NSSI compared to boys. However, methodological factors may have contributed to these findings. Some studies report that a gender difference in NSSI has not investigated samples of pure NSSI but have combined NSSI with other groups. For example, Laye-Gindhi and Schonert-Reichl reported on NSSI ideation and not NSSI behavior, and Muehlenkamp and Gutierrez noted greater rates for girls in their NSSI plus suicide attempt group but not the NSSI only group. Other studies that have examined gender differences

in NSSI using a pure NSSI group have not shown significant gender differences in NSSI (Hilt, Nock, et al., 2008; Muehlenkamp & Gutierrez, 2004). Given that support for greater rates of NSSI in girls is mixed, future research is needed to better understand the magnitude of any gender differences in NSSI and under which conditions and contexts it may be found. It is possible that the inconsistency in the literature on gender and NSSI may be accounted for by the role of age or pubertal development, yet we located no studies that longitudinally investigated the emergence of the gender difference in NSSI and how age, puberty, or other developmental factors affected the gender difference.

C. CONCURRENT PSYCHIATRIC OUTCOMES

NSSI is associated with several other maladaptive outcomes during adolescence. Most notably, there is an association between NSSI and suicidal thoughts and behaviors. Nock et al. (2006) conducted a study of adolescents age 12–17 with multiple inpatient admissions. Among this sample, 70% of those who reported NSSI also reported a lifetime history of at least one suicide attempt. Additionally, Prinstein et al. (2008) found that following inpatient hospitalization, adolescents engaging in NSSI at greater rates had a higher likelihood of being hospitalized in the subsequent 1½ years, even after controlling for suicidal ideation. These studies suggest a link between NSSI and suicidal behavior, even though NSSI and suicidal behavior are distinct. Yet to our knowledge, no study has examined NSSI as a risk factor to lifetime suicide attempts in a community sample of youth. The association between NSSI and suicidality in the general population may be lower than that reported by Nock, Prinstein, and colleagues because more severe psychopathology and comorbidities are typically seen in clinical samples, as noted earlier.

Exploring the association between NSSI and psychiatric diagnoses, Nock et al. (2006) found that in a sample of inpatient adolescents who reported NSSI, 41.6% were diagnosed with major depressive disorder (MDD), 23.6% with posttraumatic stress disorder, 15.6% with generalized anxiety disorder, 44.9% with conduct disorder, and 59.6% with oppositional defiant disorder. Moreover, of the girls in the same study, 51.7% met criteria for a diagnosis of BPD, 21.0% for avoidant PD, 20.7% for paranoid PD, 6.9% for dependent PD, 6.9% for histrionic PD, and 5.2% for narcissistic PD. Recent evidence also suggests a link between posttraumatic stress symptoms and NSSI (Shenk, Noll, & Cassarly, 2010). Psychiatric correlates in parents have also been shown to relate to NSSI in youth. Deliberto and Nock (2008) found that the youth engaging in NSSI had parents who reported a

history of greater alcohol abuse, drug abuse, violence, and their own suicidal ideation. Differences were not found for parental history of self-injury, bipolar disorder, depression, anxiety, schizophrenia, and eating disorder in this study. However, Hankin and Abela (2011) found that parental history of clinical depression as well as parents experiencing a depressive episode when prospectively assessed over 2½ years predicted NSSI in youth.

D. METHODOLOGICAL CONSIDERATIONS

Information on the continuity/discontinuity of NSSI over time and age of new NSSI engagement is essential to inform an enhanced understanding of developmental pathways. Literature on developmental pathways emphasizes investigating early precursors of later developing phenomena to better understand the process by which predisposing vulnerabilities eventually lead to these phenomena in some individuals and not in others (Pickles & Hill, 2006). It may be that different groups (e.g., childhood onset of NSSI vs. adolescent onset of NSSI) exhibit distinctive pathways (i.e., multifinality), or within a group, different pathways exist due to multiple factors (i.e., equifinality). For example, in the depression literature, there is evidence that young children may experience a broad array of negative temperamental characteristics that lead to different outcomes over time, such as an anxiety disorder or depressive disorder (see Lahey, 2004), suggesting the notion of multifinality. However, numerous processes, such as early life trauma, genetics, or acute stressors to name a few, may all play a role in the emergence of a later depressive disorder (see Colman & Ataullahjan, 2010 for a recent review), suggesting equifinality. Therefore, further research with younger children is necessary to explore developmental pathways in NSSI.

Second, much of the extant research on NSSI in youth has been conducted with clinical inpatient and outpatient psychiatric samples. Information on the risk factors, correlates, consequences, and functions of NSSI in clinical samples of youth is extremely important, as many youth who engaged in NSSI are inpatient or outpatient clients. Yet, research with psychiatric clinical samples exhibits known and problematic biases. For example, Goodman et al. (1997) showed that adolescents using outpatient mental health services had greater severity of psychiatric disorder compared to peers not using these services, were more likely to be non-Hispanic and White, have comorbid diagnoses, were postpubertal if they were female, and have parents who were more educated, yet less satisfied with family life. Moreover, research employing clinical samples is more prone to Berkson's bias, a specific selection bias with clinical samples

where some individuals with a certain outcome may use clinical services at greater rate than others and, therefore, be observed with this outcome at a greater rate. Effect sizes also tend to be less accurate and appropriate (both over and underestimation of effect sizes) in clinical samples (Cohen & Cohen, 1984). Given these known biases in psychiatric clinical samples, it is equally as important to investigate NSSI using general community samples. Of note, some research has utilized general community samples of adolescents (e.g., Muehlenkamp & Gutierrez, 2007). In the remainder of this chapter, we highlight whether a clinical or community sample was used, as the sample ascertainment likely affects epidemiological features, risk factors, and processes.

IV. Functional Models: Why Do Youth Engage in NSSI?

Given the preponderance of NSSI among youth, as well its many negative correlates, it is imperative that the function of this behavior be investigated. Nock and Cha (2009) use the word "function" to describe the antecedent of NSSI, in the way it would be used by learning theorists and behavioral therapists, and not to broadly describe the purpose of NSSI. In this respect, Nock and Prinstein (2004) suggested a functional approach to understand what precedes and maintains NSSI behaviors, and they delineated two dimensions: interpersonal versus intrapersonal and negative reinforcement versus positive reinforcement. These two dimensions constitute the four function model (FFM; see Figure 2) in which four proposed categories of potential functions of NSSI can be evaluated. In this model, the automatic categories represent an internal (or intrapersonal) dimension and the social categories represent an external (or interpersonal) dimension. Additionally, the positive

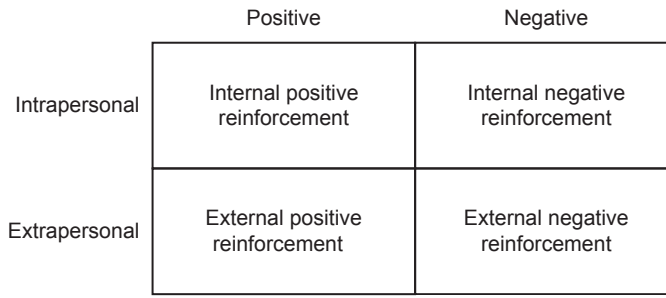

Fig. 2. Four function model of NSSI.

categories represent the desire to bring about a certain behavior or result and the negative categories represent the desire to stop an outcome from occurring. For example, the negative internal category includes wanting to stop feeling a negative emotion, and the positive social category includes wanting to get attention from parents.

In addition to the FFM proposed by Nock and Prinstein (2005), Klonsky (2007) has delineated additional factors that may play a role in the functions of NSSI. These include (1) affect-regulation, to alleviate negative emotional arousal; (2) antidissociation, to discontinue dissociation; (3) antisuicide, used as a replacement or avoidance of the desire to commit suicide; (4) interpersonal boundaries, to emphasize autonomy; (5) interpersonal-influence, to call upon others; (6) self-punishment, to inflict one's anger onto oneself; and (7) sensation-seeking, to produce positive feelings, such as excitement. As stated by Klonsky, it is important to recognize that these functions are not mutually exclusive, and there may be additional ways to categorize and define the functions of NSSI. Therefore, Klonsky's theoretical framework applied to NSSI and the FFM proposed by Nock and Prinstein may be viewed complementarily.

Despite relevant theoretical frameworks to describe the functions of NSSI, we located empirical work directly testing only the FFM. This research has predominantly focused on cross-sectional correlates. Still, support for the FFM comes from both adolescent clinical samples (e.g., Nock & Prinstein, 2004) and community samples (e.g., Lloyd-Richardson et al., 2007). Nock and Prinstein found that about 24–53% of adolescent inpatients engaging in NSSI reported using the behavior for internal functions (i.e., to stop bad feelings, to relieve feeling numb, or to feel something even if it was pain), and about 6–24% reported using NSSI for interpersonal functions (i.e., to avoid being with people, to get attention, and to be like someone respected). These findings suggest that the internal functions are more commonly used. We review evidence for internal functions first and then the interpersonal functions.

The extant findings support an internal negative reinforcement function of NSSI. Hilt, Cha, and Nolen-Hoeksema (2008) found that adolescent girls with greater internal distress, specifically those with greater depressive symptoms, engage in NSSI for internal functions, such as regulation of emotions. Additionally, Franklin et al. (2010) found a link between individuals with a history of NSSI and decreased arousal after a pain induction (used as a proxy for NSSI in the laboratory), whereas those in the control group showed increased arousal following the pain induction.

In support of the interpersonal functions of the FFM, Hilt, Cha, et al. (2008) found that peer victimization was associated with self-report of both social positive reinforcement and negative reinforcement functions

in a sample of adolescent girls. Peer communication quality moderated the association between peer victimization and social functions, such that girls with lower-quality peer communication were more likely to engage in NSSI for social reinforcement (i.e., an interpersonal positive function) when experiencing higher levels of peer victimization. Also consistent with the functional model, low social support prospectively predicted NSSI 2½ years later among youth ages 11–14 (Hankin & Abela, 2011). It is possible that these youth engaged in NSSI for positive social reinforcement (e.g., obtaining support). NSSI behaviors may also serve as a positive reinforcement strategy to elicit a response from parents, whether a negative reaction or an increase in perceived social support.

V. Risk Factors and Mechanisms for NSSI

We now shift the focus of this chapter to risk factors and processes associated with NSSI. Although there are many potential risks, we focus on three main areas that may affect NSSI in youth: (1) emotions and cognitive risk factors, (2) interpersonal relationships, and (3) biological factors. Of note, the distinction we make between these three areas is somewhat arbitrary, as they often overlap with one another (e.g., youths' emotional reactivity and parents helping children to regulate emotions).

A. THE ROLE OF EMOTIONS AND COGNITIONS IN NSSI

Theoretical models propose that individuals who engage in NSSI are more emotionally dysregulated and they use NSSI as a means of coping with intensely negative emotional responses (Chapman, Gratz, & Brown, 2006; Darche, 1990; Suyemoto & MacDonald, 1995). For example, adolescents report having strong feelings of sadness and anger prior to engaging in NSSI (Nock, Prinstein, & Sterba, 2009) and perceiving these emotions as being overwhelming and uncontrollable (Chapman et al., 2006). Further, as the internal negative reinforcement category of the FFM might suggest, after engaging in NSSI, these individuals report a significant increase in positive emotions (Muehlenkamp et al., 2009).

Other evidence suggests that adolescents and young adults who reported NSSI showed a greater physiological arousal while completing a distress tolerance task (Nock & Mendes, 2008). Compared to controls, those with a history of NSSI had a significantly greater skin conductance response. In addition, trajectories of emotional response also differed between the NSSI and non-NSSI groups. While the non-NSSI group

showed a skin conductance response that increased initially, it tapered off midway through the task. In contrast, the NSSI group's skin conductance response continued to build throughout the task. These results suggest that NSSI individuals may have different physiological response patterns, and they may be more emotionally dysregulated.

Emotional responses have been proposed to comprise two separate processes: emotional reactivity and emotion regulation (Mennin & Fresco, 2010). Emotional reactivity refers to one's innate, unaltered emotional reaction. Emotion regulation refers to one's ability to change the emotion or alter the way the emotion is experienced (McRae, Ochsner, Mauss, Gabrieli, & Gross, 2008). It has been speculated that both of these processes are impaired in individuals reporting NSSI (Chapman et al., 2006). In fact, across childhood and adolescence, as youth begin to rely on the self for regulation of emotion instead of other (see Thompson & Goodman, 2010), dysfunction in emotional processes can impact the development of psychopathology (Cole, Michel, & Teti, 1994). Further, evidence from other bodies of literature point toward the presence of combined emotion regulation ability and emotional reactivity relating to other negative outcomes, especially during adolescence. For example, brain imaging studies on anxiety in adolescence have shown that adolescents show greater amygdala activity, and this related to heightened reactivity to emotions and a poor ability to regulate emotions (Hare et al., 2008). Very little research, however, has attempted to determine whether those with a history of NSSI are both highly emotionally reactive *and* poor at emotion regulation. Further, these processes have rarely been explored at periods across development, specifically before and during the ontogeny of NSSI, to elucidate any potential causal relationship between emotional responding and NSSI.

Nonetheless, research to date on NSSI and emotions suggests that emotion dysregulation among NSSI individuals may not be a result of heightened reactivity, as laboratory measures of emotional reactivity have not found differences between individuals reporting NSSI and controls (e.g., Davis et al., 2011; Niedtfeld et al., 2010). Therefore, another possibility is that dysregulation associated with NSSI arises from poor emotion regulation ability. Emotion regulation can be conceptualized as the use of cognitive, behavioral, or social strategies to reduce or change emotional responses (Gross & Thompson, 2007). Further, adaptive emotion regulation refers to the ability to use a variety of nondestructive strategies to regulate emotional responses (Gross & Thompson, 2007). Research has found that those with a history of NSSI are less successful at using adaptive emotion regulation strategies, supporting theoretical models suggesting that NSSI is used to regulate emotions. For example, youth

and adults using NSSI report impairment in regulation abilities such as emotional clarity, awareness, acceptance, strategies, impulses, and goals (Slee, Garnefski, Spinhoven, & Arensman, 2008). Much of the evidence demonstrates that individuals engage in NSSI behaviors to alleviate emotional distress or negative affect (Brown, Comtois, & Linehan, 2002; Chapman et al., 2006). It has also been shown that adolescents who have a history of NSSI tend to display higher levels of physiological reactivity to stress, as well as reduced stress tolerance and deficits in social problem-solving skills (Nock & Mendes, 2008). Adults engaging in NSSI additionally show decreased ability to reduce sadness using cognitive reappraisal (Davis et al., under review). Further, it has been proposed that with continued engagement in NSSI over time, an individual may become reliant on NSSI to regulate their emotional responses (Suyemoto & MacDonald, 1995).

Studies have begun to examine cognitive factors as distal risk factors predicting engagement in NSSI behavior. Given its stability, cognitive vulnerability, specifically negative cognitive style, may operate as a distal risk predicting NSSI. Negative cognitive style, which is defined as the tendency to generate pessimistic stable, global, and internal causal attributions about negative events and infer negative consequences and self-implications from these events (Abramson, Metalsky, & Alloy, 1989), has been shown to differentiate youth who engage in NSSI from those who do not 2½ years later (Hankin & Abela, 2011). It is also possible that the interaction of cognitive vulnerability with stress leads to higher levels of NSSI in at-risk youth. In fact, Guerry and Prinstein (2010) found that the interaction between cognitive vulnerability and stress prospectively predicted NSSI trajectories, such that only in the presence of high cognitive vulnerability did high amounts of stress lead to NSSI engagement. High levels of stress did not predict NSSI among optimistic youth (i.e., low negative cognitive style).

It has also been theorized that individuals engage in NSSI to provide relief from unpleasant thoughts, implicating certain cognitive vulnerabilities as a potential direct causal link to NSSI. For example, in his escape theory, Baumeister (1990) proposed that suicidal behavior serves to relieve internal distress, which can be applied to NSSI. Rumination, one candidate for studying escape theory in NSSI (Hoff & Muehlenkamp, 2009), is a cognitive process involving repetitive focus of attention on one's thoughts and emotions (Nolen-Hoeksema & Morrow, 1991). Studies have shown that rumination tends to exacerbate negative emotion (Moberly & Watkins, 2008; Thomsen, 2006), and the need to obtain relief from negative thoughts and emotions leads individuals to engage in self-injurious behaviors (SIB; e.g., Najmi, Wegner, & Nock, 2007; Nock & Prinstein, 2004).

Similarly, emotional cascade theory (Selby, Anestis, & Joiner, 2008) posits that high levels of rumination result in a cascade of increasingly negative affect, which would propagate engaging in behaviors to distract from the rumination. Indeed, studies examining this relationship in undergraduate samples have found positive correlations between rumination and NSSI (Hoff & Muehlenkamp, 2009; Selby, Connel, & Joiner, 2010). Additionally, Selby and Joiner (2009) propose that the use of NSSI serves as distraction from intense negative affect within the emotional cascade framework in individuals with BPD. They explain that the act of NSSI, possibly through seeing blood or feeling pain as a consequence of the self-injurious act, may distract an individual from ruminative thoughts associated with an emotional cascade. By distracting oneself from extremely dysregulated negative emotions, attention is shifted elsewhere and the level of dysregulation subsides. Therefore, NSSI behaviors propagate a negative feedback loop that downregulates intensely negative emotions and, therefore, distraction from cognitions such as rumination.

The thought suppression literature provides another line of research examining how NSSI may be a method by which individuals distract themselves from unwanted thoughts. Thought suppression is a method of cognitive control often utilized to control emotions when thoughts create unpleasant affect (Wegner, 1989). Najmi et al. (2007) found that NSSI might be used as a distracter to help suppress unwanted cognitions. Additionally, Chapman, Specht, and Cellucci (2005) found that thought suppression was associated with self-harm frequency in female inmates with BPD. Chapman et al. suggest that unsuccessful attempts at thought suppression may counterintuitively increase the frequency and intensity of the negative thoughts and the distress that accompanies them, thereby increasing risk for NSSI.

Hopelessness, or believing that the future will not get better and one is powerless to change it, may also relate to engagement in NSSI. Consistent with this, hopelessness has been found to relate to NSSI in adolescent community samples (Hankin & Abela, 2011; O'Connor, Armitage, & Gray, 2006) and an adolescent clinical sample (Dougherty et al., 2009), as well as an adult inpatient sample (Andover & Gibb, 2010). Further, state-dependent levels of hopelessness, or in other words, "feeling stuck in the moment," may play a unique role in the ontogeny of NSSI, as well as the decision to use this maladaptive coping strategy in the time immediately preceding NSSI behaviors. As Selby and Joiner (2009) speculate, NSSI may serve to release oneself from experiencing large amounts of increasingly negative emotions or cognitions, such as those described by an emotional cascade. It might be that being in a hopeless state in which one increases in negative cognitions over time may be another avenue

by which youth may turn to NSSI. Research using daily diary methods, which have begun to be used in the NSSI literature (e.g., Armey, Nugent, & Crowther, in press), could explicate how different levels of state-dependent and trait-like vulnerabilities differentially relate to NSSI in youth. Although Nock et al. assessed state-dependent feelings preceding NSSI engagement (i.e., angry, overwhelmed, worthless), to our knowledge no research has investigated state-dependent levels of hopelessness with NSSI.

B. DEVELOPMENT AND MAINTENANCE OF NSSI AND INTERPERSONAL RELATIONSHIPS

Since models of NSSI maintain that NSSI behaviors serve not only to express and externalize overwhelming or intolerable emotions but also to regulate these emotions (Suyemoto, 1998), it is important to examine the potential developmental origins of dysfunctions in emotion regulation that lead individuals to engage in NSSI. Emotional difficulties among those who report NSSI may begin early in childhood. Among the various influences that contribute to the development of affect-regulation, parenting behaviors and styles have received considerable attention. Parenting behaviors and styles exert a significant role in child and adolescent development and their views about their self and the world (Darling & Steinberg, 1993). Such views can be learned directly from parents' verbal feedback or can be inferred indirectly from parents' behaviors.

Caregivers also play a critical role in modulating their children's physiological arousal by providing a balance between soothing and stimulation in the early years of life (van der Kolk, 1996). Sensitive and responsive caregivers maintain optimal levels of physiological arousal in their children, which leads to the development of a normal biological stress response system, as measured by glucocorticoid levels in the limbic hypothalamic pituitary adrenal (LHPA) axis (Vasquez, 1998; Yates, 2009). In contrast, parents who are insensitive or inconsistent may promote chronic hyperarousal in their children, adversely affecting the structure, organization, and function of these physiological systems (Yates, 2009). Yates suggests that as children mature, caregivers teach their children skills so that they can modulate their own emotional arousal and derive comfort from social supports outside the family system.

Children raised in sensitive and supportive environments have caregivers who acknowledge their displays of emotion and trust that their emotional and cognitive responses reflect valid interpretations of life events (Linehan, 1993). Linehan's theoretical work on BPD maintains that an "invalidating

environment" interferes with children's ability to develop adaptive cognitive appraisal and communication abilities. According to Linehan, in an invalidating environment, the expression of emotional distress is trivialized or disregarded by the caregiver. Instead, it is attributed to overreactivity, oversensitivity, paranoia, a distorted view of negative events, or a failure to adopt a positive attitude. Similarly, children experiencing an invalidating environment can also be taught to dismiss positive emotions, ascribing these experiences to naiveté, overidealization, or immaturity. A child raised in an invalidating environment is taught that his or her evaluation of casual events, subjective experience of emotion, and outward expression of emotion are incorrect. Children who have experiences with caregivers who are insensitive and inconsistent have more limited opportunities to develop effective emotion regulation strategies from parents and other caregivers (Sim, Adrian, Zeman, Cassano, & Friedrich, 2009). As a result, children raised in this type of environment are at a high risk for engaging in NSSI later in life. In fact, Sim et al. found that adolescents who engaged in NSSI reported experiencing higher levels of invalidation from caregivers than adolescents who did not engage in NSSI.

Similarly, abused children often use avoidant emotional responses (Eisenberg, Cumberland, & Spinrad, 1998; Krause, Mendelson, & Lynch, 2003). These children may learn that their internal emotional cues are incorrect and that they must search the environment for cues on how to feel. As a result, these children experience a sort of emotional tug-of-war between their own emotional experience and what their environment requires them to feel. Ultimately, they are punished for choosing either strategy and, as a result, may oscillate back and forth between suppressing emotional responses and having extreme emotional responses (Linehan, 1993). Without the proper tools to identify, evaluate, and communicate emotional states to others, individuals raised in an invalidating environment or those with a history of abuse may engage in NSSI instead of effectively communicating their emotional state (Yates, 2009).

While the family system represents a formative environment that contributes to the development of emotion regulation strategies in infancy and early childhood, it can also act as a stressor that interacts with maladaptive emotion regulation skills to produce negative affect, which can lead to NSSI (Chapman et al., 2006). Research has shown that interpersonal stressors occurring within the family system, such as rejection, loss, or conflict, often immediately precede engagement in NSSI (for a review, see Prinstein, Guerry, Browne, & Rancourt, 2009). Individuals who possess poor emotion regulation skills may have a greater likelihood of engaging in NSSI behaviors when faced with interpersonal stress occurring repeatedly within the family system. Consequently, sexual abuse, physical abuse,

parental emotional neglect, parental overprotection, and control have been shown to be significant predictors of the frequency of NSSI (Gratz, 2003, 2006; Gratz, Conrad, & Roemer, 2002). Therefore, negative parenting may act as a stressor that interacts with poor emotion regulation skills to lead to NSSI behaviors.

Recently, there has been more attention given to peer relationships as antecedents and functions of NSSI. The increase in significance of peer relationships that characterizes the transition to adolescence parallels the rise of NSSI occurring within this developmental period (Centers for Disease Control and Prevention, 2004; Kessler, Berglund, Borges, Nock, & Wang, 2005). In particular, several theories suggest that peer relationships during adolescence play an important role in the etiology, maintenance, and exacerbation of NSSI (e.g., Heilbron & Prinstein, 2008, see Prinstein et al., 2009). Corresponding with the increase in time spent with peers and expanding social networks (Furman & Buhrmester, 1992; Gavin & Furman, 1989), adolescents also experience a greater number of interpersonal stressors (Hankin, Mermelstein, & Roesch, 2007; Rudolph & Hammen, 1999). Evidence suggests that interpersonal events may be especially relevant for NSSI. For example, interpersonal problems (e.g., peer conflict, peer rejection) are concurrently and longitudinally associated with suicidal ideation and behavior and frequently precipitate suicidal behavior (Berman & Schwartz, 1990; Prinstein, Boegers, & Spirito, 2001; Rigby & Slee, 1999). Peer victimization has also been shown to be a correlate of NSSI (Heilbron & Prinstein, 2008).

Recent theory on NSSI has also posited a vulnerability-stress model in which interpersonal stressors are associated with an increased risk in NSSI among youth who exhibit certain vulnerabilities, particularly social information biases (Prinstein et al., 2009). Such youth are more likely to encode and interpret social events in maladaptive ways, leading to distortions of the causes or salience of social cues (Abramson et al., 1989; Crick & Dodge, 1994). These biases then become problematic in the presence of interpersonal negative events. Since there is a paucity of research on vulnerability-stress models for NSSI, future research will likely benefit from examining peer stressors within vulnerability-stress models.

A growing body of research suggests that peer contagion factors may contribute to the development and maintenance of NSSI among adolescents. Several studies demonstrate that NSSI among patients in psychiatric treatment facilities are associated with increases in others' NSSI behavior (e.g., Ghaziuddin, Tsai, Naylor, & Ghaziuddin, 1992; Rosen & Walsh, 1989). One recent study found support for a transactional model among adolescent psychiatric inpatients suggesting that adolescents' NSSI predicted increases in frequencies of general SITB 9 months later among

their closest friends, which in turn predicted increases in adolescents' NSSI behavior another 9 months later (Prinstein et al., 2010). These associations were moderated by gender, with significant associations found only for girls. Prinstein and colleagues also found that in a community sample, best-friends' reports of NSSI prospectively predicted increases in adolescents' NSSI behavior 11 months later, but only among younger (sixth grade) adolescent girls. Taken together, these studies suggest that (a) peer contagion effects occur within dyadic relationships, as well as within broader friendship groups, (b) adolescent girls may be especially susceptible to peer influences of NSSI, and (c) contagion effects are transactional, such that an individual's NSSI behavior may influence peer engagement in NSSI, which in turn may maintain and exacerbate an individual's NSSI.

Although initial evidence suggests that peer contagion effects occur for NSSI behaviors among adolescents, studies have not yet explored potential mechanisms of peer influence. Several theoretical models offer a framework for understanding how peer contagion effects may occur for health risk behaviors (Prinstein & Dodge, 2008), and these have been applied to contagion of NSSI (Heilbron & Prinstein, 2008). Heilbron and Prinstein have proposed several mechanisms of peer influence on NSSI. These include (1) a prototype/willingness model (e.g., Gibbons, Gerrard, Reimer, & Pomery, 2006), that suggests that youth may form a positive prototype of a person engaging in NSSI; (2) a pluralistic ignorance model (e.g., Prentice & Miller, 1993), in which youth believe that their private attitudes regarding NSSI are singularly different from others; and (3) a social learning model (Bandura, 1973), suggesting the possibility that youth may observe peers obtaining positive social reinforcement (e.g., high status, attention) when engaging in NSSI, which may lead to emulating NSSI behavior.

While understanding how peer influences may relate to NSSI is important, we must consider these influences differently across development. It has been widely established that the role of peer relationships changes over time for youth (e.g., Hartup, 1996; La Greca & Harrison, 2005). This knowledge warrants caution when concluding how peer processes may influence NSSI across development. Certain peer processes may influence engagement in NSSI across developmental periods while other peer processes may vary. Alternately, it is possible that peers may not play any role in the emergence of NSSI earlier than adolescence, as this is when peers become increasingly important (see Furman & Buhrmester, 1992). As it seems likely that differences in peer influences change over time, this highlight the importance of understanding if, in fact, some young children engage in NSSI, and subsequently if developmental influences may differ for these younger youth.

C. BIOLOGICAL PERSPECTIVES AND THE EMERGENCE OF NSSI IN YOUTH

So far we have explored the role of cognitive and emotional factors on NSSI, as well as how interpersonal relationships may precede the onset of and maintain NSSI across development. Biological processes, including the role of the brain, genetics, and specific hormones, are important to explore as well, as they may play a unique role in the emergence of NSSI. Although there is currently no research examining the neurological correlates of NSSI alone in either adults or youth, there are several studies providing preliminary evidence suggesting that several brain regions may play a role in NSSI behaviors given the link between NSSI and BPD (e.g., Kraus et al., 2010; Niedtfeld et al., 2010). Since significant neurological changes affecting emotion regulation occur during adolescence (Somerville, Jones, & Casey, 2010), this may be an area of research implicated in the ontogeny of NSSI during this developmental period. Brain regions of interest have included limbic regions (e.g., amygdala and orbitofrontal cortex) as they are implicated in processing and responding to emotional and threatening information (Davis & Whalen, 2001). Additionally, activation in the prefrontal cortex has been examined given its role in regulating limbic regions, cognitive reappraisal, and decision making (Ohira et al., 2006).

The majority of studies examining possible neurological correlates of NSSI focus on adult BPD samples, most likely due to the high prevalence of NSSI in this clinical population (Mack, 1975; Sansone, Wiederman, & Sansone, 1998). Emotional dysregulation is a core feature of both NSSI (Chapman et al., 2006; Gratz, 2006) and BPD (see Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004 for a review). Additionally, dysfunctional pain perception has also been noted in BPD patients who engage in SIB, including NSSI (Russ, Roth, Lerman, & Kakuma, 1992). Therefore, current research has mainly focused on investigating brain regions associated with emotion regulation and pain perception in this clinical population. Of note, most of the research on this population does not separate NSSI from other forms of SIB; therefore, we will refer to SIB, and not NSSI, in the following paragraphs.

Several studies with BPD patients have found elevated activation in limbic brain regions along with concurrent dysfunctional prefrontal regulation patterns, which are thought to contribute to poor emotion regulation (Minzenberg, Fan, New, Tang, & Siever, 2007; Silbersweig et al., 2007; Wingenfeld et al., 2009; see Niedtfeld & Schmahl, 2009 for a review). Brain activity related to pain perception in BPD has also been of interest, as patients report experiencing distress related to reduced pain perception,

which is often relieved by SIB (Lieb et al., 2004). Studies have found that BPD patients experience dysregulated fronto-limbic brain activity in response to heat pain stimulation (Schmahl et al., 2006) as well as differential activation of these regions in response to heat pain stimulation depending on their state of affective arousal (Niedtfeld et al., 2010). Interestingly, a recent pilot study had BPD patients and healthy controls listen to a standardized script describing an emotional situation followed by an act of self-injury while undergoing functional magnetic resonance imaging (fMRI; Kraus et al., 2010). The authors found that BPD patients exhibited decreased activation in the anterior cingulate cortex (ACC), an area of the brain that has been shown to be related to cognitive appraisal (Ochsner, Bunge, Gross, & Gabrieli, 2002) and pain responsiveness (see Klossika et al., 2006 for a review). Kraus and colleagues posit that the deactivation in the ACC seen in BPD patients may reflect their inability to successfully monitor their emotions during an emotional or stressful situation as well as dysregulation in pain perception during a self-injurious act.

In addition to a better understanding of possible neural underpinnings, there have also been significant advances in the study of how measured genes contribute to psychopathology and related traits, both in the context of direct gene-phenotype correlations (rGE) as well as interactions between genes and environmental factors (GxE) predicting specific outcomes. Despite these advances, there are currently no studies examining the genetics of NSSI in youth.

Although specific genes associated with NSSI have not been directly examined, there is a considerable body of research investigating genes related to associated psychological disorders and risk factors for NSSI reviewed earlier in this chapter. Candidate genes associated with the neurotransmission of serotonin have been widely examined in relation to several traits related to NSSI behaviors, such as emotion regulation (see Canli & Lesch, 2007 for a review), suicidal behaviors (see Mann, Brent, & Arango, 2001 for a review), and BPD (see Lis, Greenfield, Henry, Guilé, & Dougherty, 2007 for a review). Allelic variation in 5-HTTLPR and MAOA has been the focus of numerous investigations related to these traits and may therefore be of interest in future research aimed at identifying candidate genes associated with NSSI behaviors.

The association between 5-HTTLPR and emotion regulation has been seen across developmental stages and measurement type. For example, Kochanska, Philibert, and Barry (2009) found evidence for poor self-regulatory abilities in insecurely attached 15-month-old toddlers, and Gotlib, Joormann, Minor, and Hallmayer (2008) found 5-HTTLPR to be associated with higher and more prolonged stress reactivity to a laboratory stressor among adolescent girls. Additionally, there are several adult studies

examining the association between 5-HTTLPR and endophenotypes, such as emotion regulation, as measured by neurological activity in response to emotional or threatening stimuli (Canli et al., 2005; Hariri et al., 2002, 2005; Heinz et al., 2005, 2007; Pezawas et al., 2005). As previously noted, there is a high incidence of NSSI behaviors in those diagnosed with BPD, which could suggest that candidate genes associated with BPD may also be related to NSSI. However, studies of BPD and 5-HTTLPR have yielded mixed results with several demonstrating a link between 5-HTTLPR and BPD (Lyons-Ruth et al., 2007; Maurex, Zaboli, Öhman, Åsberg, & Leopardi, 2010; Ni et al., 2006; Steiger et al., 2005; see Lis et al., 2007 for a review) and one not finding an association (Pascual et al., 2008). However, the absence of a direct correlation between 5-HTTLPR and self-harm behaviors among BPD patients does not necessarily rule out the possibility of finding a GxE with 5-HTTLPR.

Similar to 5-HTTLPR, MAOA plays a critical role in the neurotransmission of serotonin, norepinephrine, and dopamine via degradation of these amine neurotransmitters. Recently, MAOA has been examined in relation to brain activity during emotion processing and cognitive inhibition tasks, implicating that this gene could be related to neural networks associated with impulsivity, aggression, and emotion regulation (Eisenberger, Way, Taylor, Welch, & Lieberman, 2007; Passamonti et al., 2006). Additionally, MAOA differentiates activation of limbic regions in response to negative facial emotions (i.e., sadness), which could indicate that MAOA is also related to emotion regulation (Lee & Ham, 2008). The association among MAOA, impulsivity, and aggression is pertinent, as research has demonstrated that many individuals who engage in NSSI spend less than 5 min thinking about the act (Nock & Prinstein, 2005) and report greater impulsivity on several measures of impulsivity (Glenn & Klonsky, 2010; Janis & Nock, 2009).

Dopamine dysfunction has been thought to play a role in several psychological disorders including BPD (e.g., Friedel, 2004) and schizophrenia (e.g., Egan et al., 2001). *COMT* is a gene that contributes to dopamine levels via metabolic processes (e.g., Chen et al., 2004; Sabol, Hu, & Hamer, 1998) and therefore could serve as a candidate gene of interest when examining the role of dopamine in NSSI related traits. COMT has been associated with greater limbic-prefrontal activation to unpleasant emotional pictures (Smolka et al., 2005, 2007) and to both conscious and unconscious processing of fearful faces (Williams et al., 2010), as well as heightened amygdala–ventral medial frontal connectivity when matching negative facial expressions (Drabant et al., 2006). Drabant and colleagues suggest that this pattern of brain activation seen with COMT may confer inflexibility in processing emotion stimuli, which could contribute to poorer emotion regulation.

Additionally, COMT has also been studied in relation to suicidal behavior and aggression, but with mixed results. Several studies have found an association with suicidal behavior (Kia-Keating, Glatt, & Tsuang, 2007; Nolan et al., 2000; Ono, Shirakawa, Nushida, Ueno, & Maeda, 2004) and impulsive aggression in schizophrenia (e.g., Kotler et al., 1999; Lachman, Nolan, Mohr, Saito, & Volavka, 1998) as well as in suicide attempts (Rujescu, Giegling, Gietl, Hartmann, & Möller, 2003). However, other studies have not found an association with increased aggression (e.g., Baud et al., 2007; Zammit et al., 2004) or with suicide in mood disorder patients (Zalsman et al., 2008). The mixed findings in relation to suicide and aggression may be related to the context in which these behaviors are examined (e.g., varying by diagnostic group status). As youth who engage in NSSI report experiencing various psychological disorders (Nock et al., 2006), it may be important to carefully determine the presence or absence of specific diagnoses when investigating the role of COMT in NSSI behaviors.

Turning the focus to hormonal risk factors, one of the most robust biomarkers for psychopathology is dysregulation of the hypothalamic pituitary adrenal (HPA) axis (Burke, Davis, Otte, & Mohr, 2005): Humans and animals have an interconnected set of complex physiological systems for managing physical, cognitive, and socioemotional challenges. One of these systems is the HPA axis. In humans, the primary product of this system is the steroid hormone cortisol (Hennessy & Levine, 1979). Associations with the HPA-axis system and NSSI behaviors have been documented in both animal models (Sandman, Hetrick, Taylor, Marion, & Chicz-DeMet, 2000) and in humans (Symons, Sutton, Walker, & Bodfish, 2003). Although the existing literature on NSSI and dysregulation in both basal cortisol patterning and cortisol stress reactivity is limited, both under- and overactivation in HPA-axis functioning have been noted. For example, Symons and colleagues documented altered diurnal salivary cortisol patterning in adults with developmental disabilities and who exhibited SIB (but not NSSI expressly) relative to matched controls. Specifically, such patients with severe SIB were found to have higher levels of diurnal cortisol. Additional evidence for hypercortisol and NSSI comes from a single case where nocturnal urinary cortisol levels were assessed (Sachsse, von der Heyde, & Huether, 2002). Increases in cortisol in the evenings preceding an episode of NSSI were documented, followed by a return to baseline. Others, however, have documented hypocortisol and NSSI (Verhoeven et al., 1999). This patterning is in line with early work by Yehuda and colleagues (e.g., Yehuda, Giller, Southwick, Lowy, & Mason, 1991) demonstrating hypocortisolism in individuals with PTSD (an early risk factor for the development of NSSI).

It is possible that the role of HPA-axis functioning on NSSI, or on processes associated with NSSI, changes across development or with the emergence of puberty. Recent literature suggests that HPA-axis activity changes across development in samples of healthy youth (e.g., Gunnar, Wewerka, Frenn, Long, & Griggs, 2009) and youth at high risk for MDD (e.g., Hankin, Badanes, Abela, & Watamura, 2010). Specifically, greater cortisol reactivity to a stressor or challenge is noted in postpubertal youth (i.e., hyperreactivity), whereas relatively reduced cortisol reactivity to a challenge is observed in prepubertal youth (i.e., hyporeactivity). Since preliminary evidence on HPA-axis functioning in individuals reporting SIB suggests a possible link to NSSI, developmental trends, such as those in general youth samples and samples with MDD, will be important to explore.

VI. Future Directions for the Study of NSSI in Youth

A. LIKELY AVENUES FOR FUTURE RESEARCH

As NSSI is still a relatively new area of study, especially among youth, there are large gaps in the literature. Studies are needed to better determine how the multitude of risk factors change across the life span. Longitudinal studies are also greatly needed to elucidate causal mechanisms between these distal and proximal risk factors and engagement in NSSI. Moreover, without research on prepubertal children, it is not possible to determine temporal precedence with respect to which risk factors and processes predict first engagement in NSSI and to embark upon eventual prevention studies for NSSI.

Specific targets for future work on NSSI during childhood and adolescence have been highlighted throughout this chapter. First, epidemiological studies beginning in childhood, ideally during the early elementary school years, would offer better insight into the role of gender on NSSI. Although there is no evidence to suggest high rates of NSSI in children of this age, this cannot be concluded without proper empirical studies. It will be imperative that these studies investigate not only gender differences, but also how patterns observed in the risk factors for NSSI may change by gender and over time as well as how gender may serve as a moderator of other proximal and distal risk factors for NSSI. An important consideration for conducting research on NSSI with youth is the question of whether there is an increased likelihood for engaging in NSSI by asking participants about potential NSSI engagement. To our knowledge, this question has not been directly addressed with NSSI,

although it has been investigated with suicidality. The consensus is that asking youth about suicidality does not increase the likelihood of youth initiating suicidal behaviors (Hall, 2002). Thus, there is little reason empirically to believe that there are ethical concerns about asking youth about NSSI, especially for those who are not engaging in NSSI.

Second, because knowledge on the relationship between NSSI and suicidal thoughts and behaviors currently comes only from clinical samples, longitudinal research on this relationship using community samples will be informative. We speculate that the current conceptualization of high concurrence of suicidal ideation and behavior in those reporting NSSI is likely a sampling bias (i.e., Berkson's bias), and future studies using community samples of youth will show lower rates of suicidal ideation and thoughts in these youth. Third, we found no research using parents as informants reporting on their children's engagement in NSSI. This is an area for future research, given that multi-informant studies generally can be more informative, reliable, and valid than studies with a single informant (Cole, Hoffman, Tram, & Maxwell, 2000). Research on concordance of report of NSSI between youth and their parents may shed light on how reliable and valid others' reports of NSSI may be and, further, if better concordance may be indicative of better or worse prognosis in the continuity/discontinuity of NSSI among these youth.

Fourth, research on emotion regulation and emotional reactivity, as well as the cognitive vulnerabilities that are implicated in NSSI in youth, has begun to provide insight into the development and function of NSSI. Yet none of the research on NSSI and emotions, and very little of the research on NSSI and cognitive vulnerability, has taken a developmental perspective and assessed constructs of emotion at or around the time of NSSI onset. Prospective studies are needed to better understand the temporal relationship between difficulties of emotional processing and NSSI. We speculate that there may be specific emotional processes and cognitive vulnerabilities as well as possible attentional biases seen in youth who go on to engage in NSSI. Specific targets for future research in this area may include assessment of state and/or trait levels of hopelessness and other cognitive vulnerabilities, understanding how both emotional reactivity and emotion regulation ability during childhood may stabilize and be reinforced thus leading to NSSI, and if attention to or away from negative stimuli, especially those that are self-referencing, may somehow play a role in the emergence of NSSI over time.

Fifth, several theoretical models suggest that peers are especially relevant to understanding NSSI during adolescence. However, there is a dearth of studies testing these models, and research investigating the contribution of peers in the onset, maintenance, and exacerbation of NSSI

has been mostly neglected. Future research on NSSI and peers will need to investigate possible differential roles of dyadic peer relationships versus peer groups in NSSI as peer groups, such as cliques, may influence NSSI. Finally, it will be important to consider how the interplay of various peer processes may contribute to the development and maintenance of NSSI, especially at different times across development as there is evidence from the general peer literature that salience of peer relationships changes throughout childhood and adolescence.

Sixth, although biological factors may be especially helpful in understanding why youth engage in NSSI and why the ontogeny of the behavior may occur at a specific period in development, limited knowledge on this topic exists. There are currently no published studies on measured rGE or GxE predicting NSSI behaviors, especially among samples of youth. Plausible candidate genes for future studies include those related to psychological disorders and risk factors associated with NSSI, such as emotion regulation and BPD (e.g., 5-HTTLPR) and aggression and impulsivity (e.g., COMT and MAOA). Future investigations, especially using imaging techniques and measuring biological markers such as HPA-axis activity, are also necessary to illuminate the possible neurological etiology of NSSI.

Finally, investigators conducting future research will need to appreciate that there are most likely multiple, dynamic, and complex pathways at different levels of analysis by which these risk factors and processes could contribute to NSSI.

B. DEVELOPMENTAL CONSIDERATIONS IN UNDERSTANDING NSSI

The majority of the literature we have reviewed in this chapter examined risk factors and processes that may predict the onset of NSSI, yet this research has been conducted with youth who already engage in NSSI. To our knowledge, very little research has used a developmental perspective to look at distal and proximal precursors to NSSI. This raises the question: What happens throughout development to bring about engagement of NSSI around adolescence—the modal age of NSSI onset?

There are significant cognitive, social, and physical transitions during adolescence that are of particular relevance for NSSI. Cognitively, there are substantial gains in reasoning and perspective taking skills, as well as the acquisition of better emotional understanding. Socially, peer relationships take on greater significance. Physically, the onset of puberty leads to hormonal and physical changes.

The social developmental changes that occur in adolescence (Habermas & Bluck, 2000) result in the desire for more independence and autonomy (Erikson, 1968; Ryan & Lynch, 1989) and the formation of new and more salient social networks (Furman & Buhrmester, 1992). However, by themselves such social changes seem inadequate to explain why NSSI is common and rates increase greatly throughout adolescence. What happens when this novel desire for autonomy comes face-to-face with more difficult changes and challenges during adolescence? It seems plausible that some adolescents may not yet be fit with adequate emotion regulation strategies and coping skills. Taking into account that there is a protracted period of development in the prefrontal cortex of the adolescent brain (Romine & Reynolds, 2005), some adolescents have difficulty coping with the new demands that are placed on them without the help of others. Same age peers, who are also lacking in regulatory skills, may not be adequate to help with emotion regulation at times of conflict or heightened stress. Moreover, without the cognitive capacity to fully control emotions, the onset of NSSI in adolescence may come about in an effort to reduce heightened negative emotions and affect without the help of others.

It is particularly important to explore potential biological factors and their role in the ontogeny of NSSI, as there is evidence for an effect of certain biological pubertal changes on the emergence of psychopathology (e.g., Ge, Conger, & Elder, 2001; Natsuaki, Biehl, & Ge, 2009). A key feature of puberty, the surge in gonadal hormones, is thought to affect the central nervous system (Geidd, Castellanos, Rajapakse, Vaituzis, & Rapoport, 1997), stress reactivity system (Gunnar et al., 2009; Hankin et al., 2010), as well as other neurotransmitters (e.g., GABA, glutamate; Terasawa & Fernandez, 2001). Moreover, there is evidence that the effects of genes on psychopathology may be more robust during adolescence and adulthood (e.g., after the pubertal transition), which suggests a potential interaction between biological processes and psychosocial factors that would predict NSSI as an outcome (see Uher & McGuffin, 2008, 2010 for research on this interaction for depression).

As Prinstein (2008) explains, NSSI may be an "overdetermined" behavior, which involves multiple risk factors and processes, as well as serving numerous functions at one time. Additionally and already alluded to, there may be an interactive process among these changes in adolescence that are theoretically linked to NSSI. In one of the first longitudinal studies of NSSI in an adolescent population, Guerry and Prinstein (2010) found that interpersonal stressors interacted with a cognitive vulnerability, specifically attributional style, to predict NSSI over the course of 9 months. Hankin and Abela (2011) also found that stressors

prospectively predicted NSSI over the course of 2 years in a community sample of adolescents. In addition, implicit in a study by Hilt, Cha, et al. (2008), girls who reported a history of NSSI had experienced a greater frequency of peer victimization (measured as both overt and relational victimization together).

C. SUMMARY

This chapter has shown that NSSI is a prevalent behavior, especially during adolescence, that may relate to other maladaptive outcomes. Youth reporting NSSI may have difficulty regulating their emotions and exhibit negative cognitions about themselves and life events, and these may be learned and reinforced early on from parents. Peer relationships may also play a role in both the ontogeny and maintenance of NSSI in youth. Further, research on NSSI in adults shows that several biological factors may also relate to NSSI, and this underscores the importance of a better understanding of the relationship between these factors (e.g., neurological correlates, genetics, hormones) and NSSI across development. We have begun to understand cross-sectional correlates, distal vulnerability factors, and the rates and functions of NSSI in young adults and adolescents. However, research on these topics in younger youth, especially in childhood and early adolescence, is lacking. It is particularly important to examine these constructs not only during and after but also before the onset of NSSI in order to understand what developmental processes lead to the ontogeny of NSSI in youth. Further, even though NSSI has been a budding research topic for the past decade, there is still much room for growth, specifically investigating influences to NSSI with a developmental lens.

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