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## Cortisol Response of Non-Suicidal Self-Injurers versus Non-Self-Injurers Exposed to a Social Rejection Laboratory Stressor

Patrick L. Kerr

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CORTISOL RESPONSE OF NON-SUICIDAL SELF-INJURERS VERSUS  
NON-SELF-INJURERS EXPOSED TO A  
SOCIAL REJECTION LABORATORY STRESSOR

by

Patrick L. Kerr

Bachelor of Science, University of Central Florida, 2002

Master of Arts, University of North Dakota, 2005

A Dissertation

Submitted to the Graduate Faculty

of the

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in partial fulfillment of the requirements

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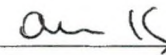
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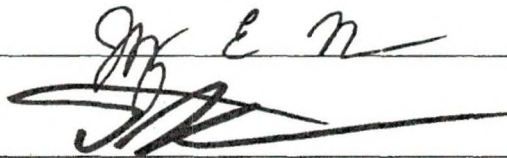
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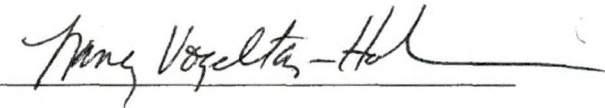
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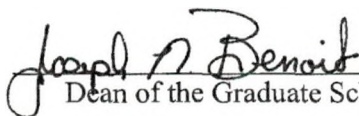
  
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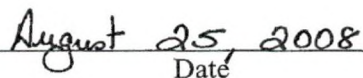






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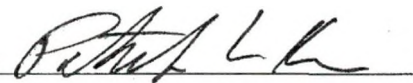
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For those who suffer.

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## ABSTRACT

Non-suicidal self-injury (NSI) represents a growing area of concern in a variety of clinical settings, yet remains a poorly understood phenomenon. An influx of research on the functions of NSI over the past decade has suggested a biopsychosocial emotional regulation model of this behavior. This model proposes that self-injurers engage in NSI to reduce negative emotions, and presupposes that self-injurers are characterized by emotional dysregulation. The present study evaluated the biological component of this model by assessing hypothalamic-pituitary-adrenocortical axis (HPAA) functioning in a group of self-injurers (n=26) and non-injuring healthy controls (n=28). HPAA functioning was assessed via measuring salivary cortisol levels across 65 minutes following exposure to an interpersonal rejection stressor or neutral comparison condition. Results of the experiment did not support the biological facet of the proposed biopsychosocial model. A complex time x condition x group x gender interaction effect was found, which was counterintuitive to study hypotheses. However, self-reported difficulties with emotional regulation were in the hypothesized direction, with self-injurers reporting greater difficulties in most domains. Future research must seek other potential lines of evidence in support of the biological aspects of emotional regulation in self-injurers.

## CHAPTER I

### INTRODUCTION

Over the past two decades, non-suicidal self-injury (NSI) has received an increased degree of attention in a number of mediums, including the mainstream media (e.g., Marano, 2004) and popular culture (e.g., Reznor, 1994), psychopathology research literature (e.g., Favazza, 1996, 1998; Gratz, 2001; Muehlenkamp, 2005; Nock & Prinstein, 2004; Ross & Heath, 2002; Ross & Heath, 2003), and disciplines outside of the mental health field (Hafeez & Goodyear, 2003). More specifically, the study of NSI has grown exponentially as an area of research focus in recent years, especially over the past decade. Such recent research indicates that this behavior occurs in about 1-4% of general, non-clinical populations (Briere & Gil, 1996; Klonsky, Oltmanns, & Turheimer, 2003), with higher rates in adolescents (10-15%; Hawton & Rodham, 2006; Muehlenkamp & Gutierrez, 2004; Ross & Heath, 2002) and college students (17-35%; Gratz, 2001; Whitlock, Eckenrode, & Silverman, 2006).

Evidence from anthropological findings suggests that self-mutilative practices have been engaged in for much of human evolutionary history (Casteret, 1951; Janssens, 1957). In a more recent historical context, case studies of self-mutilation became more prevalent in the medical literature near the close of the nineteenth century and the beginning of the twentieth century. However, following Menninger's (1938) early review of the literature concerning self-mutilation, the gap in publications on this topic suggests that interest in this phenomenon was largely dormant for some time. The



attention of the psychiatric community was directed back to this topic about twenty years later when “wrist slashing” among psychiatric inpatients gained notoriety (Favazza & Simeon, 1995). It was unclear at that time what differentiated self-mutilating behavior from suicidal behavior, besides the inevitable consequences of these behaviors. Although it is difficult to ascertain the exact function of patients’ self-mutilation from accounts in this early literature, the behaviors, and contexts in which they are described, seem consistent with modern day accounts of NSI. Therefore, it can be reasonably assumed that this literature base represents the origin of the study of NSI in psychiatry and psychology.

Kreitman (1977) was the first to devote an entire text to self-injury. His work documented one of the first systematic epidemiological studies of NSI, which he termed “parasuicide” (a term discussed in more depth in subsequent sections of this paper) among United Kingdom residents in Edinburgh over a period of eight years beginning in the mid-1960’s. It was at this time that interest in this phenomenon regained prominent attention in the psychiatric literature. This area was further developed by subsequent work of Ross and McKay (1979) and Pattison and Kahan (1983), which also served to increase research and clinical interest in NSI. Since that time, the research literature on NSI has expanded substantially, incorporating a variety of theories regarding potential etiological and phenomenological models for this behavior.

#### Defining Non-Suicidal Self-Injury

As noted earlier, self-injury has historically been referred to by numerous other labels (e.g., Gratz, 2001; Kreitman, 1977; Linehan, 1993a, 1993b; Simeon & Favazza, 2001). Indeed, the diversity of labels reflects a central difficulty in conceptualizing this

behavior within this area of study. Moreover, it reflects the underlying divarication in the extant literature regarding definitions of self-injury, in that each label has at times been used to describe slightly different variations of this behavior. One primary point of disconnect seems to be whether or not failed suicide attempts or self-injurious behavior with an intent to die should be included in a definition of self-injury.

Kreitman (1977) coined the term “parasuicide,” defining it as “a non-fatal act in which an individual deliberately causes self-injury or ingests a substance in excess of any prescribed or generally recognized therapeutic dosage” (Kreitman, 1977, p. 3). Alcohol consumption was excluded from the criteria of this definition because there is no standard amount of alcohol that is commonly acknowledged as a regular, prescribed, or therapeutic dosage. Linehan (1993a) posited that this definition also included failed suicide attempts in which there was minimal intent of death, and she adopted the term in her own seminal work. Most recently, Walsh (2006) forwarded a definition of self-injury as “intentional, self-effected, low-lethality bodily harm of a socially unacceptable nature, performed to reduce psychological distress” (Walsh, 2006, p.4). Walsh’s definition is useful from a clinical perspective in that it portends a specific functional hypothesis of self-injury, i.e., that this behavior serves an ameliorative function for psychiatric symptomatology. However, a definition that is functionally specific may be limiting in clinical research exploring the potential functions of this behavior.

Other definitions of self-injury have been forwarded in the literature. Like Walsh (2006), some have also defined NSI from a functional perspective. For example, Miller (1995) proposed that NSI in women (which she termed “Trauma Reenactment Syndrome”) is a symbolic reenactment of childhood trauma, citing the wealth of

literature connecting self-injury with early trauma (e.g., van der Kolk, Perry, & Hermann). Favazza (1996) and others (e.g., Muehlenkamp, 2005) have submitted that NSI, with its associated features and sequelae, represents a separate psychiatric disorder.

Favazza (1996) proposed what he termed “Repetitive Self-Mutilation Syndrome,” and indicated that this disorder would best be classified as an impulse control disorder. The proposed criteria for Repetitive Self-Mutilation Syndrome included “ (1) preoccupation with harming oneself physically; (2) recurrent failure to resist impulses to harm oneself physically, resulting in the destruction or alteration of body tissue; (3) increasing sense of tension immediately before the act of self-harm; (4) gratification or a sense of relief when committing the act of self-harm; and (5) the act is not associated with conscious suicidal intent and is not in response to a delusion, hallucination, transsexual fixed idea, or serious mental retardation” (Favazza, 1996, p.256). Although there are some components of this suggested classification that necessitate refinement, one may infer from these criteria that this definition of self-injury assumes that (1) NSI is distinct from suicide, and (2) it facilitates the regulation of tension. Self-injury in the present study is conceptualized in the context of non-suicidal self-injury. Specifically, NSI is any form of self-directed behavior that causes or has the potential to cause immediate physical (i.e., tissue) damage to the individual without intent to cause death.

#### Differentiating Non-Suicidal Self-Injury from Suicidal Behavior

Much confusion has arisen regarding the difference between NSI and suicidal behavior. This confusion has been promulgated by previous usage of the term “parasuicide,” which implies a suicidal component to the self-injury that may not have been engaged in to terminate the individual’s life, as well as the use of the term

“deliberate self-harm” in multiple definitional contexts (e.g., Haw, Houston, & Townsend, 2002). There is evidence that a correlation between suicidality and NSI exists. Empirical research indicates that as much as approximately 40% of self-injurers may experience suicidal ideations during episodes of NSI (Favazza, 1996; Pattison & Kahan, 1983), and approximately 50-85% of these individuals have attempted suicide at least once in their lifetime (Stanley, Winchel, Molcho, Simeon & Stanley, 1992). However, there is also evidence to suggest that suicide attempters who self-injure are a unique sub-group (Stanley, Gameroff, Michaelson, & Mann, 2001). On the other side of this conceptual coin, there is also evidence that self-injurers who attempt suicide differ from their non-suicidal counterparts by way of longer histories of NSI and a higher number of NSI methods, thus making them a higher risk group in multiple ways (Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006; Whitlock & Knox, 2007). Parallel to this, NSI has been conceptualized as falling along a continuum of self-harm where severity of the behavior ranges from low-lethal compulsive NSI (e.g., trichotillomania, onychophagia) up to and including suicide (Linehan, 1986). Such a conceptualization suggests that while NSI and suicide may be conceptually or categorically related, they are also functionally distinct phenomena.

Walsh (2006) summarizes the differences between these two behaviors as being primarily in the following areas: (1) intent; (2) level of physical damage and potential lethality; (3) behavioral frequency; (4) multiplicity of methodology; (5) helplessness and hopelessness; (6) psychological repercussions of a NSI episode. In the following sections, the evidence supporting this contention is discussed.

### *Intent*

Behaviors are frequently defined in terms of their purpose (i.e., attainment of reinforcement, reduction of punishment). The success or failure of a behavior in fulfilling that purpose is often a primary contingency that maintains it. Shneidman (1985) discussed how intent separates NSI from suicide. He contended that suicidality is characterized by a desire to terminate psychological pain; the suicidal individual does not typically wish to kill the body, but instead to end her or his painful experience of consciousness. Conversely, those who engage in NSI may do so as a way of changing their experience of consciousness; this may involve ameliorating an excess or paucity of emotion (Brown, Comtois, & Linehan, 2002; Conterio and Lader, 1998; Favazza, 1987; Linehan, 1993a; Muehlenkamp & Gutierrez, 2004). The intent of both groups is to escape from psychological pain; however, the degree to which that pain is averted (i.e., temporarily or permanently) is the differentiating factor.

#### *Level of Physical Damage and Lethality*

There is strong empirical evidence indicating that there are a limited number of methods employed in completed suicides. Self-inflicted gunshots, hanging, overdose, self-poisoning, and jumping from lethal heights are attributed to approximately 98.6% of the deaths that result from suicide, whereas cutting accounts for only about 1.4% (Centers for Disease Control, 2002). Because cutting has consistently been shown in both clinical and non-clinical populations to be the most common form of NSI (e.g., Favazza & Conterio, 1988; Suyemoto, 1998; Walsh & Frost, 2005), there is some indication that the majority of those who self-injure tend to use a relatively low-lethal method to engage in this behavior. Some authors (e.g., Muehlenkamp, 2005) contend that many moderate/superficial self-inflicted injuries incurred by NSI individuals are able to be

taken care of by the individual without medical attention. Thus, the physical damage caused by NSI is likely to be less severe medically and less lethal than suicidal behavior, although NSI can clearly become a more lethal behavior as the frequency and severity increase (it is important to note that there may be other reasons for not seeking medical attention even when such care may be warranted, such as prior experiences with pejorative care providers, as argued by Shaw [2002]).

### *Behavioral Frequency*

In light of the above discussion regarding level of physical damage incurred by suicidal and NSI behavior, it follows logically that the frequency with which these behaviors occur is a differentiating factor. While there are subsets of suicidal individuals who remain suicidal for protracted periods of time due to chronic psychiatric disturbance (e.g., Major Depressive Disorder, Recurrent; Bipolar Disorder; Borderline Personality Disorder), many suicidal behaviors or suicide attempts occur in singularity (Walsh, 2006). However, it is important to note that some data suggests that up to 69% of suicide attempters have previously engaged in suicidal behavior (Haw, Houston, Townsend & Hawton, 2002). Moreover, Klonsky and Olinio (2008) report that there may be one subset of self-injurers that is characterized by substantially higher levels of suicidality. Nevertheless, although suicidality or suicidal ideation may be chronic, suicidal behavior is less likely to be so in these individuals. Conversely, Walsh (2006; Walsh & Rosen, 1988) reports that NSI tends to be chronic, with the typical self-injurer engaging in 20-100 episodes over several years.

### *Multiplicity of Methodology*

Research suggests that suicidal individuals to be single attempters if indeed they do make a suicide attempt (Holden & Johns, 1997). Furthermore, those who do engage in repeated suicide attempts tend to employ the same method each time. In the case of repeat attempters, a number of things may prevent the behavior from resulting in death, including both accidental and purposeful discovery (although categorizing pre-death purposeful discovery as “suicide” versus NSI remains debatable). Conversely, research indicates that many NSI individuals are likely to use more than one method of NSI (Favazza, 1992; Favazza & Conterio, 1988; Osuch, Noll, & Putnam, 1999). This may be due to circumstance, such as removal of access to preferred NSI method, habituation to the sensation produced by each method, or due to personal preference. Alternatively, this may reflect a subtypology of NSI, as suggested by latent class analyses forwarded by Klonsky and Olino (2008), in which at least 11% of participants reported multiple methods of NSI.

### *Helplessness and Hopelessness*

Research suggests that feelings of hopelessness are substantially associated with suicidality, and constitute a major risk factor for suicidal behavior in depressed individuals (Beck, 1996; Gutierrez, Osman, Kopper, Barrios, & Bagge, 2000; Johns & Holden, 1997). Beck and colleagues’ (1979) cognitive theory of depressive illness posits that depression is frequently characterized by perceived hopelessness. This hopelessness may extend to one’s view of the potential for their suffering to end. As a provenience for suicidality, hopelessness reflects a maladaptive or perseverative problem-solving process. In this problem-solving process the depressed individual is unable to reframe, revise, or restructure their self- and world-schemas to incorporate evidence contradicting their

perceived hopelessness. In this same vein, helplessness, specifically learned helplessness, is a well-established contributing factor for depression and suicidality (see Seligman, 1975, for a review). Suicidally depressed individuals commonly feel as if they are beyond being helped (Beck, 1996). Such a perception then further contributes to a view of their situation as hopeless, thus further supporting the notion that suicide is the only effective solution for ending their suffering. These two facets of suicidality are clearly complimentary to each other. Walsh (2006) proposes that self-injurers do not experience the same hopelessness that suicidal individuals do because they are engaging in a behavior that relieves the distress contributing to hopelessness. It may be that suicidal individuals feel little control over their circumstances and their pain, and self-injurers may feel some sense of control via their NSI. Walsh (2006) further contends that, although self-injurers are clearly not precluded from experiencing depressive cognitions, they may be less likely to see the future as completely bleak because the NSI they engage in may function to acutely reduce distress.

#### *Psychological Repercussions of a Self-Injury Episode*

Suicide attempts not resulting in death are frequently followed by a continuation or exacerbation of depressive symptomatology (Walsh, 2006). This effect may result from the individual's perception of the attempt as another failure, buttressing their view of themselves as inept. Furthermore, the fact that suicide is viewed as the key strategy that will eliminate the attempter's pain suggests that survival of an attempt will not reduce the tension leading up to the act. Conversely, NSI is frequently reported to have a distress reducing effect (Linehan, 1993a; Nock & Prinstein, 2004, 2005). Although there



is also shame and guilt that may go along with NSI, there is also a tendency for tension and stress to decrease following an act.

#### Etiology of Non-Suicidal Self-Injury

Much attention in the research literature has been devoted to developing an accurate model to explain how NSI develops and is maintained. Early hypotheses explaining NSI were based on psychodynamic theories (e.g., Menninger, 1935), but these theories have lacked empirical support. More recent theories of NSI are empirically grounded in developmental, behavioral, and neurobiological perspectives. Some of these theories have recently been integrated by Linehan (1993a) and others (e.g., Chapman, Gratz, & Brown, 2006; Brown, Comtois, & Linehan, 2002) into the biopsychosocial model of BPD, which has also been used to explain NSI.

#### *Biopsychosocial Model*

Linehan's (1993a) biosocial model remains the most comprehensive explanation of NSI. In her reformulation of BPD, Linehan proposed that borderline features, which commonly include NSI (Zanarini, Frankenburg, Hennen, & Silk, 2003) are part of the sequelae of dysregulation of fundamental emotional regulation processes. These processes may include inhibition of inappropriate behavior related to strong negative or positive affect, self-regulation of physiological arousal related to affect, refocus attention while experiencing intense affect, and the coordination of action for accomplishment of a non-mood-based objective via self-organization (Gottman & Katz, 1990). The biopsychosocial model posits that such emotional dysregulation originates from a transactional relationship between inherent biological characteristics and specific types of developmental environments that are invalidating (i.e., the invalidating environment).

Invalidating environments are typically characterized by people and other factors that question, discount, or disregard current and long-term needs, as well as feelings and subjective experience of the individual. Continuous invalidation frequently results in the individual doing the same to her/himself. The concept of biosocial transactions is consistent with a comprehensive model of BPD, and provides a systemic context in which to discuss features of this disorder, such as NSI.

This model, which is nicely summarized in a recent review of the NSI literature by Klonsky (2007), also suggests that there are secondary effects of dysregulated emotions. It is proposed that emotional dysregulation contributes to further dysregulation of emotions through dysregulation of environmental factors, which is also supported by recent work in an adolescent population by Lloyd-Richardson and colleagues (2007) indicating that adolescents who report engaging in this behavior report doing so not only to help regulate either affect or an emotional state (i.e. “to stop bad feelings” was one of the most frequently endorsed reasons for NSI), but also commonly endorsed motivations for NSI that involved a social context that may be related to the initial internal dysregulation (e.g., “to get control of a situation,” to get a reaction from someone”). Hilt, Cha, & Nolen-Hoeksema (2008) also reported a contextually contingent functional model of NSI among adolescent girls consistent with the secondary effects model above. In this model, NSI engaged in as a response to internal distress was performed in an attempt to regulate emotions via an automatic negative reinforcement mechanism (see Nock and Prinstein, 2004), whereas, NSI in response to stress in interpersonal relationships was aimed at achieving social positive or social negative reinforcement. In sum, research and theoretical models proposing a secondary effects process suggest that frequently extreme

or atypical behaviors may contribute to unstable or tentative relationships with others, withdrawal of affection from others, or loss of opportunities for personal gain or accomplishment. Vulnerability toward invalidating environments may thereby be created through this secondary pathway, thus exacerbating the effects of emotional dysregulation and perpetuating the potential for future dysregulation.

There are other factors that may contribute to exacerbation of emotion dysregulation symptoms. According to the biosocial model, features of emotional dysregulation in an individual with a biological predisposition for difficulties with emotional regulation may be amplified when the individual is in an environment that is perceived as negative (e.g., unsupportive, unstable, or excessively demanding). Furthermore, Linehan (1993a) contends that, due to a “higher sensitivity” (Linehan, 1993a, p. 44) to emotional stimuli, such individuals may evaluate their environments, or experiences, as being negative more readily than others who do not have such difficulties in regulating their emotions. Early research in emotion and emotional expressive and attention processes supports the notion that higher emotional arousal is associated with greater focus on emotionally relevant aspects of a situation or environment (Bahrick, Fitts, & Rankin, 1952; Bursill, 1958; Callaway & Stone, 1964; Cornsweet, 1969; Easterbrook, 1959; Mcnamara & Fisch, 1964). According to Linehan’s (1993a) model of BPD, this may be a component of or related to borderline individuals’ quick emotional escalation and subsequent slow return to baseline emotional functioning. In short, the biosocial model suggests that the myriad BPD behaviors develop as a result of a transaction between biological, social, and environmental factors.

Although NSI is only one of the features of BPD, it is the feature that is of most relevance to this discussion of the biosocial model. The biosocial model of BPD conceptualizes BPD behaviors as maladaptive attempts by the individual to regulate their emotional experience. This conceptualization thus suggests that these behaviors are somehow effective in intervening in the present dysregulation, and either result in the restoration of homeostasis, or initiate a process that does so. As an emotional regulation strategy, NSI appears to meet this criterion. Although the exact mechanism is not clear, self-injuring borderlines and other self-injuring individuals report a reduction in anxiety and other aversive emotions (e.g., hostility) after engaging in NSI (Liebenluft, Gardner, & Cowdry, 1987; Ross & Heath, 2003). Others, such as Walsh (2006) cite clinical anecdotal evidence based on self-reports from patients that indicate this as well. Thus, NSI seems to be effective in providing some intervention in dysregulated affective states by either directly or indirectly terminating the dysregulation or the affective state itself. It is likely that the directness or indirectness of this effect is not ubiquitous and varies from person to person.

The biosocial model provides a comprehensive and useful template for explaining NSI. As applied to NSI, this model is an important advent for research on this phenomenon in that it delineates the conditions and factors through which this behavior may develop. The present study approaches the study of NSI from the perspective that it serves an emotional regulatory function, a perspective which has been forwarded in the biosocial model. Therefore, understanding how the various factors of the biosocial model transact and result in this behavior is of paramount importance in understanding the premise of the research proposed in this manuscript. In his review and expansion of

Linehan's (1993a) model, Walsh (2006) has proposed that the biopsychosocial theory may be segmented into five primary components. These include environmental, biological, cognitive, affective, and behavioral. In the following sections, these proposed components are reviewed to provide the reader with an overview of how each contributes to the biosocial model, and to NSI.

### *Environmental Components*

The environmental factors affecting NSI behavior include elements of the self-injurer's personal and familial history, and components of the current environment. The family of origin provides an environment for ongoing learning and development of behavioral repertoires for children. NSI is associated with disturbance within the family of origin, including familial psychiatric illness and substance abuse (Walsh & Rosen, 1988), abusive and violent family member interactions (Shapiro & Dominiak, 1992), as well as suicide and NSI among family members (Favazza, 1996, 1998). Because parental/caretaker and peer modeling and socialization of emotion-related coping behaviors contributes substantially to children's acquisition of behaviors (Bandura, 1986; Eisenberg, Cumberland, & Spinrad, 1998), observation of self-destructive behavior, where there is the potential for modeling of maladaptive coping behaviors, may be particularly detrimental. Children who are repeatedly exposed to maladaptive coping strategies such as NSI or substance abuse by their caretakers are thus at an increased risk for development of these maladaptive coping strategies themselves.

Other life-experiences of NSI individuals also may play a role in the onset of the behavior. For example, a wealth of research suggests that there are higher prevalence rates of NSI in clinical and non-clinical adults who experience childhood sexual and

physical abuse than those who do not (Briere & Gil, 1998; Gratz, Conrad, & Roemer, 2002; van der Kolk, Perry, & Herman, 1991). Separation from, or death of, a primary caretaker during childhood also appears to be associated with NSI (Briere & Gil, 1998; Gratz et al., 2002; Walsh & Rosen, 1988). Recent work by Gratz et al. (2002) has demonstrated that males and females who self-injure are affected differently by familial and developmental environments. Gratz and her colleagues reported significant gender differences in the way that familial and developmental experiences affect and predict NSI. Sexual and physical abuse, maternal emotional neglect, and insecure maternal and paternal attachment were significant risk factors for females, while paternal separation and physical abuse were highly predictive of NSI in males. This data is consistent with Linehan's (1993a) concept of the "invalidating environment" in which a child's physical and/or emotional needs are discounted, unacknowledged, and/or not attended to. Such an environment may elicit progressively stronger expressions of need up to a point of extremity at which point those needs still may or may not be responded to. The intermittent response to such expressions of need serves to reinforce extreme expressions of emotion as a way of getting one's needs met (Linehan, 1993a). Gratz et al.'s (2002) data support this by suggesting that elements frequently present in chaotic family systems, especially loss or threat of loss of a parent or other caretaker, significantly predict NSI.

Components of one's developmental environment also contribute to NSI, especially for those who may have experienced chaotic and abusive developmental environments (Walsh, 2006). Those who have experienced parental/caretaker separation via death or removal from the home during childhood may be more sensitive to loss, or the potential for loss, of other relationships later in life. This may be particularly true for

those who engage in NSI as Gratz 2002 demonstrated. Here, a risk for NSI is present especially if the childhood loss impeded their development of healthy and adaptive coping skills.

While developmental environments may be important antecedents to the onset of NSI, aspects of the current environment may be a key factor in the perpetuation of this behavior. Any number of events may serve as a “trigger” for NSI and it is likely that a combination of factors (e.g., psychological state, pre-existing stress level, access to alternative self-soothing methods, circumstances) is the final determining factor (Favazza & Rosenthal, 1990). Psychological states may also be precipitants of NSI. Indeed, research indicates that circumstances precipitating NSI typically include aversive levels of stress and aversive affective states (Simeon & Favazza, 2001). Difficulties in occupational or educational performance, conflict in one’s relationship with an intimate partner or close friend, interactions with the legal system, and financial difficulties are examples of common stressors that may precipitate an episode of NSI. Any of these may include themes of being rejected or abandoned, which are also commonly reported themes in the precipitating factors reported by self-injurers (Favazza & Rosenthal, 1990). Additionally, Walsh (2006) contends that individuals may be more sensitive to current aversive experiences, or, moreover, the threat of current aversive experiences, that are similar to those experiences that have historically been aversive for the individual. Therefore, someone who experienced physical or sexual abuse as a child may react more quickly to the perceived threat of similar abuse as an adult than someone who did not experience such abuse, even in normal interpersonal interaction. From this discussion, it may be concluded that both the historical/developmental and current environment of an

individual, as well as the transactional relationship of these factors, may play an important role in the onset and course of NSI.

### *Biological Components*

As noted by Linehan (1993a), the biological underpinnings of NSI are likely to be heterogeneous. Data in the extant literature implicates the limbic system in the pathogenesis NSI. The limbic system, which mediates affective feeling, along with memory, learning, and perception, is one of the primary biological subsystems regulating emotional processes. Others include the brain stem, which mediates activation of purposeful behavior and general arousal, and the cerebral cortex, which functions as a mediator of attentional processes, working memory, perception, and volitional control (Lewis & Stieben, 2004). Individuals who engage in NSI are believed to have significant difficulties regulating their emotional experiences. However, the evidence for these problems with emotional regulation has been inferential, derived mostly from data based on the self-reported reduction of intense affective states following NSI. It is important to incorporate evidence from related literature bases in examining the evidence for biological mechanisms of emotional regulation.

There is some peripheral supporting evidence for such biological processes suggesting that some NSI individuals respond to treatment with anticonvulsants, which are now commonly used as mood stabilizers in Bipolar affective disorders (Chengappa et al., 1999; Hirdes et al., 2002). Recent small-sample and case studies have shown an association between administration of medications from this class of pharmacological agents and decreases in self-injury in Bipolar Disorder and BPD patients (Cassano, Latanzi, Pini, Osso, Battistini, & Cassano, 2001; Chengappa et al., 1999). Research in



bipolar patients indicates that anticonvulsants reduce the reactivity of the amygdala (a central component of the limbic system) to emotional stimuli (Drevets et al., 2002; Krystal et al., 2002). In short, studies suggest that anticonvulsants exert their effects through their potentiation of gamma-aminobutyric acid (GABA; an inhibitory neurotransmitter), limitation of electrical activity in the anterior cingulate cortex (a component of the limbic system) which is associated with emotional lability, and stabilization of the neuronal membrane at the sodium ion channel by inhibition of the release of aspartate and glutamate (Blumberg et al., 2000; Chengappa et al., 1999; Krystal et al., 2002; Theoharides, Dessain, & Shuster, 1992).

This research suggests, first, that commencement of anticonvulsant therapy is associated with reduction of NSI in some cases, and second, that there are observable reductions in other indices of emotional responsivity (outside the context of NSI) that are associated with commencement of anticonvulsant therapy. Furthermore, this research suggests that anticonvulsant medications act on those areas of the brain known to be involved in emotion and emotion regulation processes. Thus, it may be concluded that there is preliminary evidence to support an association between emotional regulatory processes at the biological level and NSI. Specifically, when stabilization of biological emotional regulation functions is achieved pharmacologically, individuals who engage in NSI do so less frequently. Research evaluating the biological mechanisms of emotional regulation (and dysregulation), while promising, is still in a relatively nascent stage, and some empirical findings suggest that only a subset of self-injurers may respond to anticonvulsant treatment (Favazza, 1996). Any conclusions from this data must, therefore, be made judiciously. Additional research is presently needed to further

elucidate the exact relationship between the biological substrates underlying emotional regulation and the NSI.

Studies of the biological components of NSI have often also conceptualized this behavior as a form of aggression, thus research on aggressive behavior is also of relevance to this discussion. Much effort has been focused on identifying significant differences in the prevalence of specific neurotransmitters as well as abnormalities of neuroanatomical structures that mitigate aggressive behaviors. In this vein, a number of neurotransmitters and neurotransmitter substrates have been implicated in the mitigation of aggressive behavior. Of these, the serotonergic, noradrenergic, and endogenous opioid systems are among the most frequently discussed as possible mitigating substrates.

There are some empirical findings from studies of serotonin levels in self-injurers that provide some evidence of reduced serotonergic functioning (see Grossman & Siever, 2001 for a review). Simeon, Stanley, Frances, Mann, Winchel, & Stanley (1992) reported that cerebrospinal fluid (CSF) levels of 5-HIAA were 44% lower in non-suicidal self-injurers than controls. This study also found that imipramine platelet binding (considered to be an analogue of serotonergic functioning) was significantly lower in self-injurers than controls. Similarly, Markowitz (1995) also concluded that NSI was associated with lower levels of serotonin in self-injurers. Inferential support for the role of serotonin has also come from evidence that selective serotonin reuptake inhibitors (SSRI) are sometimes effective in reducing self-injury (Grossman & Siever, 2001), although evidence of the iatrogenic effects of SSRIs, such as increases in suicidal ideation, have also been well-documented (e.g., Donovan et al., 2000; Grounds et al., 1995; King et al., 1991).

Although different from NSI in some important ways, studies of suicidal behavior also bare some relevance to this discussion. Research has shown a relationship, between reduced levels of 5-HIAA and suicidal behavior in suicide attempters. Specifically, findings from post-mortem studies of brainstem concentrations of serotonin and completed suicides have been relatively consistent in demonstrating a relationship between low 5-HIAA levels and suicide (Russ, 1992). Additionally, Mann and Malone (1997) documented lower levels of 5-HIAA in cerebrospinal fluid of depressed individuals who attempt suicide compared with controls from a psychiatric population. More recently, Arango et al. (2001) reported a 40% smaller concentration of 5-HT<sub>1A</sub> (serotonergic receptor sites) in the dorsal aspect of the raphe nucleus of depressed suicide completers when compared to nonsuicidal, nonclinical controls. Furthermore, in related research van Heerigen and colleagues (2001) found significantly lower binding potentials in frontal 5-Hydroxytryptophan<sub>2A</sub> (5HT<sub>2A</sub>) receptors (an index of serotonergic activity) in suicide attempters compared to non-clinical controls. Conversely, Mann, Stanley, & Malone (1996) reported finding no differences between suicide attempters and non-clinical controls in serotonin levels; however, this study did find a significant correlation between 5-HIAA and both planning of suicide attempts and extent of medical damage incurred by suicide attempts. Thus, there is some empirical support for an inverse relationship between serotonergic activity and features associated with self-harm behaviors, implying that NSI may also be related to low serotonergic functioning.

Evidence of relationships between aggressive behavior and other neurotransmitters has also been reported. Research on norepinephrine in animals (Eichelman, 1987) has indicated that levels of this neurotransmitter are negatively

correlated with aggressive behavior. Further evidence of this relationship derives from findings that noradrenergic agents that block or reduce the activity of norepinephrine are associated with a decrease in aggressive behavior. Similar results have been found in human studies, which have used CSF levels of a norepinephrine metabolite (3-methoxy-4-hydroxyphenylglycol; MHPG) as a reference index, demonstrating that CSF levels of MHPG are associated with aggression. Brown, Goodwin, Ballenger, Goyer and Major (1979) reported a significant correlation between MHPG levels in CSF and a history of aggressive behavior in soldiers with "personality pathology." However, Traskman, Asberg, Bertilsson, & Sjostrand (1981) reported contradictory findings in their sample of suicide attempters, where CSF levels of MHPG were not significantly different in their sample of depressed and non-depressed controls. It is conceivable that the unique attributes of the specific group being examined in this latter study may have contributed to their lack of significant findings. Overall, the literature appears to suggest that elevations in norepinephrine levels is associated with increases in aggression, however, this relationship may be moderated by factors such as individual psychopathology.

The endogenous opioid system (EOS) has also been implicated as a mitigating biological factor in NSI (Oquendo & Mann, 2000; Russ, 1992; Winchel & Stanley, 1991). The EOS has been discussed at length as potentially playing a role in the onset and maintenance of NSI since many self-injurers report analgesia when they self-injure. The role of the EOS in NSI can be explained in terms of operant behaviorism. It has been posited that some individuals who engage in NSI may have inherently low levels of opiate activity (Oquendo & Mann, 2000; Winchel & Stanley, 1991), and that NSI serves as a mechanism for the release of additional opioids into the regions that are deficient.

This restores the opiateergic “tone” (i.e., a standard level of endogenous opiate activity, presumed to be inadequate in such individuals) to an adequate level (Winchel & Stanley, 1991). Consequently, the NSI is reinforced by the pleasant physiological changes produced by the release of endorphins and enkephalins. In line with this model, research has demonstrated a significant correlation between severity of NSI and the plasma levels of met-enkephalins in self-injuring individuals (Winchel & Stanley, 1991). Thus there is evidence that the EOS is indeed involved in NSI at some level, although its specific contribution to this behavior has yet to be fully elucidated.

Research on pain-sensitivity in self-injurers, an area of biological research encompassing multiple neurobiological systems, has expanded beyond specific biological substrates to incorporate multi-systemic symptomatology. Specifically, diminished sensitivity to pain has also been examined as a potential explanation for chronic NSI (Walsh, 2006). When considering both adults and adolescents, approximately 47 to 60% of individuals who engage in NSI report analgesia when engaging in this behavior (Bohus et al., 2000; Nock & Prinstein, 2005).

Psychophysiological research on pain perception in self-injurers is an emerging line of inquiry. Thus far, both Russ and his colleagues (1992, 1994) and Bohus and his colleagues (2000) have found lower perception of aversive stimuli in self-injurers. Russ et al.’s (1992, 1994) studies examined perception of induced pain in NSI individuals who reported no pain during NSI. These researchers found that participants who reported no pain during NSI also reported substantially lower levels of pain than both pain-perceptive self-injurers and non-self-injuring controls. Additionally, Bohus et al. (2000) studied pain perception in self-injurers diagnosed with BPD versus non-clinical controls and

found markedly diminished perception of pain during both distress and non-distress conditions in the BPD group compared to controls. When distressed, borderline participants' pain threshold was even higher than in the non-distress condition (Bohus et al., 2000). While such biological studies of self-injurers are promising, and highlight the importance of psychobiological processes to NSI, further research is necessary before more firm conclusions can be drawn.

### *Cognitive Components*

Walsh (2006) divides the cognitive dimensions of NSI into two categories: (1) cognitive interpretations of environmental events; and (2) self-generated cognitions. According to cognitive theory, an individual may experience environmental events as problematic when they perceived those events to be aversive (i.e., painful, overwhelming). This theoretical model further contends that the rationality of one's cognitions also influences one's perception of environmental events. For example, if one believes that they should have had control over an aversive or painful situation that they could not have realistically terminated or mitigated, then the already negative perception of the situation is likely to be exacerbated. Such cognitive appraisals, especially of oneself, can be a trigger for NSI. Self-generated cognitions occur without an identifiable external cue, and are presumed to be part of the reservoir of cognitions related to self and world. In self-injurers, these cognitions may frequently be derisive (e.g., "Today is going to be the worst day I've ever had, and tomorrow will be even worse."). Cognitive theory suggests that these cognitions may then become part of one's self-schema (i.e., the meaning-making 'structure' one uses to understand oneself), and as such may contribute

to further vulnerability toward potential misinterpretation of environmental events, and thereby also NSI.

Over time, such thoughts can also become a discriminant stimulus for NSI through repeated pairing of specific thoughts, or thoughts related to a specific fear (e.g., failure, rejection, abandonment) with acts of NSI (Walsh, 2006). If the self-injurer responds to a specific cognition invariably by engaging in NSI, then that cognition itself may become a cue for self-destructive behavior by creating a strong association between the thought and the consequences of the behavior (e.g., relief of negative affect, help from others). This associative process may be explained by the following logic-path statement: *if a, then b, and if b then c; then also, if a, then c*. Therefore, an association may be made such that the self-injurer believes that when they experience the specific cognition (a), NSI is the only viable response (b), and that when they self-injure (b) there will be some sort of positive change (c) in the environment (e.g., through caring behaviors of others or reduction of aversive circumstances). The connection between (a) and (b) is made to achieve (c), which in turn reinforces (a). Such a cycle may explain the seemingly self-perpetuating cycle of aversive thoughts precipitating NSI frequently seen in self-injurers (Favazza, 1996; Walsh, 2006).

#### *Affective Components*

Negative affective states are believed to precipitate NSI because these states are perceived as overwhelming or intolerable. As noted earlier, it is also believed that NSI functions to ameliorate the distress caused by these affective experiences through a mechanism that is not yet clear. A variety of emotions may precipitate NSI episodes. It is reasonable to posit that all of these are negative in some way (e.g., depression, shame,

guilt, anxiety/panic, anger). Many self-injurers report that their NSI functions to diminish feelings of distress related to negative affect (e.g., Liebenluft, Gardner, & Cowdry, 1987). This has been supported in more recent empirical research by Nock & Prinstein (2004, 2005) in samples of adolescents. Briefly, Nock & Prinstein (2004, 2005) reported one of the primary and most frequently reported reasons for NSI reported by self-injuring adolescent psychiatric inpatients was regulation of negative emotions. This research provides important preliminary support for the long-held hypothesis that regulation of emotional states is the underlying function of NSI. However, replication and further extension of this line of research in other population subsets is needed. Additionally, research examining the psychobiological correlates of this self-reported reduction in negative affect will provide important data regarding the mechanism by which such reductions take place.

### *Behavioral Components*

The behavioral aspects of NSI, and moreover the functions of this behavior, are critical in understanding its course and the reasons for its chronicity. These functional aspects encompass each of the other components discussed above. Earlier literature attempted to explain NSI in behavioral terms by placing it into the framework of operant behaviorism. Carr (1977) proposed that, like any other behavioral pattern, NSI functions as an operant. As such, it is maintained via systematic reinforcement or punishment from the individual's external or internal environment. Carr's (1977) model specifically distinguishes between two modes of contingent behavioral maintenance: positive reinforcement and negative reinforcement. The positive reinforcement hypothesis proposes that NSI is maintained through positive (typically social) reinforcement.



Positive reinforcement is generally provided through an increase in contact with, or an increase in the care exhibited by, others in the self-injurers life in response to the self-injury. This level of care and concern, or contact, may not be achieved outside of the context of episodes of self-injury, thus supporting a continuation and increase on the frequency of the behavior. Conversely, the negative reinforcement hypothesis suggests that the NSI is maintained through the termination or avoidance of a stimulus that the individual perceives as being aversive (Bennun, 1984; Carr, 1977). The escape from the aversive stimulus (e.g., an aversive emotional state or aversive life circumstances) follows the onset or completion of NSI, and leads to an increase in the behavior because of the escape that the behavior results in.

While the above model represents an early attempt to theoretically delineate the behaviorally-based functionality of NSI, recent empirical work has provided support for this contention. In their research on the functional dimensions of NSI in adolescent inpatients, Nock and Prinstein (2004, 2005) proposed a functional model of NSI, which posits that these behaviors may serve four functions: automatic-negative reinforcement (NSI functions to reduce the discomfort associated with a negative affect state); automatic-positive reinforcement (when NSI functions to induce some form of appetitive physiological state); social-negative reinforcement (when NSI functions to facilitate an individuals avoidance or escape from the demands of interpersonal interactions and relationships; and social-positive reinforcement (when NSI functions to facilitate contact between the self-injurer and others). These researchers found that the self-injurers in their sample were more likely to engage in self-mutilating behaviors when automatic and/or social reinforcement were available or provided, thus providing evidence

consistent with the proposed functional model. Additionally, as noted by Walsh (2006), a multitude of events may precipitate an episode of NSI, including interpersonal conflict with peers or family, or substance use. Behavioral components of NSI may also include preparatory behaviors associated with NSI, such as deciding on the place, time, and method of NSI. All of these components may become strongly associated with acts of NSI, and thus precipitate a cascade of both internal and external events leading up to an NSI episode (Walsh, 2006). The findings of Nock and Prinstein (2004, 2005) therefore support the contention that environmental contingencies play a central role in NSI.

NSI tends to elicit rapid, and in some cases intense, reactions from others (Barstow, 1995; Clarke & Whitaker, 1998; Conterio & Lader, 1998; Favazza, 1998; Gallop, 2002). In the context of BPD, episodes of NSI may be associated with frequent and dramatic fluctuations between emotional polarities (Linehan, 1993a). These fluctuations can engender an understandable desire for help of some kind, and NSI can elicit attention that may result in obtaining such assistance. Linehan (1993a) notes that the extreme behaviors exhibited by individuals with BPD are commonly engaged in as a way to “alert the environment to take better care of them” (Linehan, 1993a, p. 69). Here, “care” can be operationalized in behavioral terms as reinforcement. Therefore, the relationship between the individual and their environment in this context is simultaneously discordant and operantly reciprocal. In this respect, the paucity of reinforcement that the individual receives from their environment (the antecedent) precedes the NSI (the behavior), which in turn precipitates the desired attention (consequences), or social reinforcement. Outside of the context of BPD, this process may also be observed. If an individual who chronically engages in NSI perceives that

reinforcement (i.e., attention) is generated primarily through self-injuring acts, then this sets the stage for a potentially long-standing learning paradigm. It should be noted that use of the word *attention* in this discussion is in the context of caretaking attention to neglected or abused emotional, psychological, or physical needs, and not in the context of manipulative attention-seeking behavior.

The preceding behavioral components of NSI only address the way in which the behavior is maintained, and not its onset. This component of the model may be explained by social learning theory (Bandura, 1977). Based on the operant behavioral principles discussed earlier, for any behavior to be acquired by an organism there must be an initial reinforcement of some form. Social learning theory, as applied to humans, maintains that children acquire their initial behavioral repertoire through observation of the behavior of primary caretakers (e.g., parents or others) early in the child's life. These caretakers essentially function as behavioral models for a full range of behaviors, ranging from coping skills and strategies to interpersonal behaviors and emotional expression function as models for behavior the child. Research suggests that the forum for this initial schedule of reinforcement or punishment is quite often the childhood home environment of the individual (Birt et al., 1997; Linehan, 1993a; Green, 1978; Wolfe & Birt, 1997; & Wolfe & McEachran, 1997).

Consistent with Bandura's (1977) model of social learning, Suyemoto (1998) contended in her review of the NSI literature that social learning leads to the acquisition of the behavior in one of two ways. One way is via the experience of abuse by a parent or primary caretaker during the individual's childhood years. This abusive behavior provides a salient example of interpersonal interaction, which consists of caustic and

maladaptive behaviors. The abused individual is then likely to interpret these behaviors as appropriate via social referencing, and will consequently replicate them. Second, through a classical conditioning paradigm, the individual's model of care and nurturance is paired with aversive physical experiences such as pain and hostility, and aversive emotional experiences such as shame, guilt, and anger (Suyemoto, 1998). These two paradigms seem to work in concert to initially generate NSI via imitation of parental or caretaker behaviors, and then by reinforcing the behavior via regulatory effects (e.g., termination of dissociative states, reduced anxiety, attention to needs). This hypothesis is supported empirically by research that has demonstrated a positive linear relationship between child abuse and neglect and onset of NSI (Green, 1978; Wolfe & McEachran, 1997).

It seems clear from the research discussed above that there is probably not one unitary factor to which the pathogenesis of NSI may be traced. One common thread among all etiological models for NSI, however, is that they acknowledge the contribution of emotions and emotional experiences to this behavior. Emotions have a clearly established role in the course of psychopathology. More specifically, research suggests that the regulation or dysregulation of emotions may be a prolific factor in the pathogenesis of multiple dysfunctional behaviors (e.g., Gross, 1998; Mennin, Heimberg, Turk, & Fresco, 2002). Furthermore, although NSI is more prevalent in some specific disorders, this behavior has been observed in a wide variety of psychopathologies. Given the commonality among etiological models of NSI (i.e., that NSI may function to regulate emotional states or drives) and the diversity of comorbid clinical presentations observed

in NSI individuals, it may be reasonable to conclude that that NSI does indeed serve an emotional regulatory function.

Nonetheless, the available research suggesting a relationship between emotion regulation and NSI has been correlational. A review of the extant literature reveals no empirical studies that have attempted to establish causality in this posited relationship. One method of establishing a causal relationship is through examination of objective and observable markers of emotional regulation in self-injurers. Although emotion regulation is a multifaceted construct, there are psychobiological indices and analogues that may provide valuable information about individual emotion-regulatory capacities. The present study was predicated on the assumption that examination of these indices of emotion regulation in self-injurers would further our understanding of its role in NSI. In the following sections, research pertaining to emotion-regulation and psychobiological indices of emotion-regulation is reviewed.

## Emotion and Emotion Regulation

### *Emotion*

#### *Defining Emotion*

Although earlier researchers propagated a view of emotions as states of neural activation that were situationally disruptive, and not specific to the situation (Hebb, 1949, Young, 1943), it seems clear now that emotional behaviors have developed as a function of evolutionary necessity. William James (1894) viewed emotions as behavioral and physiological response tendencies that functioned to allow a species to adapt to significant events across, and as part of, the evolutionary process. The behaviors associated with anxiety, anger, sadness, disgust, and happiness help to maintain the safety

and integrity of the organism, and the survival of the species through their physiological and behavioral correlates (Gross, 1998).

More recent empirical and theoretical work is also indicative of the adaptive functions that emotions serve. Schwarz and Clore (1983) posited that emotions convey data to the organism regarding the current or ongoing fit between the organism and the environment. Ekman (1992) noted that emotions appear to address adaptive problems by conveying information to the organism regarding their current biological needs. Oatley & Johnson-Laird (1987) reported that emotions assist with the decision-making process, and Frijda (1986), citing decades of psychophysiological research, concluded that emotions facilitate the preparation of an individual for a quick motor response by activating the autonomic nervous system. In aggregate, empirical research supporting this adaptive activation process has found that emotional responses in humans include shifts in behavioral, experiential, autonomic, and neuroendocrine systems (see Lang, 1995 for a review).

Emotions may also be viewed as systemic processes, as suggested by Scherer (1994) in his discourse on “modal emotions.” Scherer’s model proposes that an emotion is a progressive series of interconnected and coordinated shifts in the states of an organism’s physiological systems and subsystems, which may include neural circuits, respiratory and circulatory systems, and digestive processes and systems as well. Such a shift occurs as a reaction to the organism’s assessment of internal or external stimulus events that bare direct relevance to the primary needs of the organism (Scherer, 1994, 2000). For example, when an organism’s interface with its environment results in the organism being prevented from achieving a goal that is needed for survival (e.g., food),

the biological systems of the organism that require the goal's attainment will serve a motivational function. In the case of food as the organismic need, the caloric and nutritional needs of the organism are biologically and genetically determined based on the functionality of those needs. Evaluation of whether a particular need is satiated is made by the organism's biological systems through determining if the availability of the resource (e.g., calories) is sufficient to perform the necessary function (e.g., mobility, cognition). If the resources are not available or sufficient for the function of the system, the physiological systems and subsystems of the organism will work together to alter the organism's behavior as needed to achieve the goal and satiate the need (e.g., directing attention to food-related stimuli; Mogg & Bradley, 1998). Although hunger is not considered an emotion (Wierzbicka, 1999), there may be labels given to the behaviors that accompany hunger (or other aversive drive states, such as pain) in some humans (e.g., irritability, anxiety, hostility, sadness), which in turn correspond to the changes in the physiological systems that have taken place to motivate the acquisition of food. It is the pattern of change that defines the emotion, and even minute variations may be indicative of actual differences in the organism's emotional state.

Across species, emotional behavior may also be viewed as motivated behavior, especially in non-human animals and organisms. In this sense, motivation relates to action of some form, where the organism seeks to achieve a goal. This may take the form of the physiological reaction of an organism to a pleasant or aversive environmental change (Bradley, 2001), such as a threat from a predatory organism or the availability of a mating partner. In studies of animal behavior, motivated behavior is modulated by both direction (i.e., approach or withdrawal) and intensity (i.e., speed or strength of the

behavior), each of which vary as a function of the requirements of goal-attainment. In this vein, some researchers (Carver & Scheier, 1990) have proposed a view of human emotion as an index of the rate at which the current discrepancy between a particular goal and the realistic appraisal of one's proximity to attaining that goal is reduced. Here, a positive emotion is indicative of more rapid reduction of the discrepancy, whereas negative emotion is indicative of a slower reduction than would be expected. Emotion regulation, discussed in subsequent sections, is essentially a derivative of one's intention or action to reduce the discrepancy, but is not seen as an end-product itself.

In humans, such motivated behavior is referred to as emotional behavior, or more generally, emotion. While behavioral definitions of emotion facilitate theoretical understandings of this construct, there are numerous definitions that have been propagated throughout the history of emotion research. Although emotion may seem intuitively comprehensible (Bradley, 2001), an agreed upon definition has yet to be forwarded. Discussions of emotional behavior in humans are further complicated by the need to differentiate between the various temporal, intensiveness, and purposive facets of emotion. The differentiation typically required is between emotion, emotion episodes, mood, and affect. These constructs are interrelated, and sometimes used interchangeably (Gross, 1998); however, their separation may become important in describing the phenomenological and subjective emotional experiences of the individual.

An understanding of emotions and emotional experiences is integral to comprehending the full range of human behavior. Commonalities in human emotional behavior exist across cultures, albeit with different manifestations. It is possible that such manifestations are related to differences in cultural experience (Ekman, 1972), thus, the



importance of taking into account cultural differences in emotion must be considered in both research and clinical settings (APA, 2000). In humans there may be cultural differences in the expression and elicitation of emotion, which have perhaps historically served adaptive functions. Research on emotion has revealed both intercultural commonalities and differences. While there appears to be general agreement between cultures in identifying the type of emotion being expressed, there are cultural differences in judgments about the intensity of the emotional or affective state (Ekman, 1987), as well as rules for emotional expression, emotional terminology, and self-reported emotional experiences (Matsumoto, 1990, 1993). Clear and consistent differences in norms for affective and emotional expression have also been observed between collectivistic and individualistic cultures (Eid & Diener, 2001). Moreover, the impact of emotions and emotional regulation on health has been found to differ across cultures (Consedine, Magai, & Horton, 2004), with higher levels of emotional expression and inhibition affecting individual health either beneficially or adversely through a culturally-dependent contingency. In aggregate, this research supports a view of emotions as being evolutionarily adaptive and universal. Viewed in the context of emotion regulation, the adaptivity of emotions denotes the evolutionary necessity and adaptiveness of emotion regulation. It is likely that a failure to upregulate or downregulate emotions when needed would not be germane to survival of most animal species. From an evolutionary perspective, emotion dysregulation may then be conceptualized as either a deregulation of psychobiological processes or initiation of maladaptive psychobiological processes in response to environmental demands. The characteristics, contributing factors, and manifestations of such psychobiological deregulation have yet to be explored empirically

in the literature, and research in this area will be necessary to further our understanding of these processes.

### *Emotion, Mood, and Affect*

Distinguishing the constructs of emotion, mood, and affect from one another is most commonly oriented toward developing a more accurate understanding of emotion through a more precise and comprehensive description of individual emotional phenomenology. There is at present no clear concurrence in the psychological literature on how to define an emotion, although many theories have been proposed (e.g., Mayer & Salovey, 1988; Cole, Martin, & Dennis, 2004). Thus separating emotion from mood and affect is challenging. Gross (1998) has differentiated these constructs in the following way. Emotions transpire and develop during a relatively limited time-frame, whereas emotional episodes extend across longer periods of time and sometimes across multiple facets of a given situation. Emotion and affect are sometimes used synonymously; however, affect is also sometimes used to describe the behavioral or experiential manifestations of an emotion. One common practice-generated depiction of emotional experience (e.g., APA, 2000) suggests that affect describes “emotional weather,” whereas the term “mood” is used to describe the more persistent or consistent “emotional climate.” Describing mood as the emotional climate (APA, 2000) portends a sustained and/or persistent state of emotional experience for an individual, which not only includes multiple aspects of a single situation, but is present across multiple situations. A further clarification proposed by both Davidson (1994) and Fiedler (1988) is that cognitive processes are more susceptible to the influence of moods than are actions.

Earlier, Davidson (1994) differentiated between emotion and mood by stating that (1) moods are considered to be brief, whereas emotions are longer lasting; (2) emotions are thought to be accompanied by specific facial expressions, while moods are not; and (3) emotion is preceded by a readily recognizable antecedent event, whereas the antecedent precipitating a mood is not always apparent. Furthermore, emotions are thought to be experienced in response to antecedent events that are rapid and unexpected in their acute onset, whereas moods develop in response to longer-lasting, and slower progressing events.

The foregoing definitions of these constructs are useful on multiple levels. First, they help to unify the language by which researchers and clinicians alike describe the same emotional phenomena. Additionally, they clarify the constructs in some important ways. Most importantly perhaps, discussion of these constructs in terms of their temporal parameters and levels of intensity seems to point toward a functionally-based differentiation of these constructs. As noted above, a discussion of emotional processes and behaviors must incorporate a discussion of the functions they serve.

#### The Function of Emotions and Affect

Davidson (1994) has proposed that emotion differs from mood in function. Emotions occur during situations when some form of action is needed to facilitate the organism's adaptation to the circumstances, with concomitant autonomic activity (e.g., arousal or suppression/reduction). Conversely, mood functions to modulate information-processing and therefore also cognition; attention is directed more selectively to some cognitive content and limited to others.

As noted earlier, Frijda (1994) has proposed that emotion has two aspects, each of which serves different functions. The first is the appraisal of events as relevant and either pleasant or unpleasant. This cognitive appraisal answers the question of whether an event needs to be attended to and acted upon to obtain a goal or protect the interests and integrity of the organism. The second aspect of emotion is the elicitation of a behavioral, physiological, and/or experiential response to the event, which is related to protective and survival functions within the organism. It is the functionality of these facets of emotion that highlights the important roles emotions serve.

Functionally, the appraisal of events as relevant/irrelevant, and pleasant/unpleasant serves to alert the organism to the nature of an event relative to the organism's own interests (e.g., safety). The event-appraisal aspect of emotions can be considered a *relevance signaling mechanism*, which proposes that an emotion is an index of comparison between an *end-goal* and the *current state*. Concordance and discord between an *end-goal* and *current state* is signaled to the action system by emotion. This model presupposes that generalized, flexible action plans are formulated by a combination of neural circuitry and learning history of the organism. This combination may be conceptualized as the organism's action system. This system prepares and executes internal or external goal-oriented activity. The function of such preparatory and executive behavior is the remuneration of any discord or facilitation of further concordance.

The second aspect of emotion Frijda (1994) proposes is response elicitation. Here, emotion is viewed as a source of stimulation for initiating the behavior required to manage emotional events. Indeed, specific emotions directly relate to environmental

events. Fear functions to initiate self-protection or to prevent the event from occurring or recurring through minimizing exposure or reducing activity until a threat is no longer imminent; anger functions to influence a threat or a threatening other to cease the threatening behavior. In this way, emotions function to influence or modify the interaction between individual and environment, but do not modify the environment itself.

The data and models discussed above are indicative of the importance of emotions and emotional behavior across, species, culture, and individuals. The explanations of emotional processes that have been forwarded in the literature thus far and their supporting data are of relevance to more than just academic discourse. Empirically based principles that govern emotional behavior, especially the regulation and dysregulation of emotion, could be applied to the modification of maladaptive manifestations of such behavior. NSI, which appears to be associated with states of emotional dysregulation (Linehan, 1993a), may be one such detrimental manifestation. Although there does appear to be an association between NSI and emotion dysregulation, the current literature contains only correlational evidence of such a connection. Further understanding of emotional regulatory processes is necessary. In the following section, models and supporting research for emotion regulation are reviewed.

#### *Emotion Regulation and Dysregulation*

The study of emotion regulation was preceded by the study of the human coping response to “stress.” The focus of this research was centered on the theory that organisms exhibit similar physiological responses to different stress-inducing stimuli, or “stressors” (Seyle, 1956). A stress response is viewed as an individual’s attempt to cope with a challenge, be it physical, psychological, or both. Coping has generally been

conceptualized as a process involving cognitive and behavioral attempts at the modulation of both explicit exogenous and endogenous demands that are interpreted by the individual as subjectively strenuous or as extending beyond the limits of the individual's available resources for handling the demands of the situation (Lazarus & Folkman, 1984). Research has identified different types of coping, distinguishing between "emotion-focused coping" and "problem focused coping" (Gross, 1998, p. 274). The former is aimed at reducing the intensity of a negative emotion, and the latter is aimed at solving a problem.

In conceptualizing coping as a stress response, and stress responses as attempts to regulate emotions elicited by internal or external stimuli, coping itself may be thought of as a form of emotion regulation. However, this description of emotion regulation is too simplistic to accurately characterize the processes of emotion regulation as it is understood today. It is important that a clear definition of emotion regulation and the underlying components involved in this process are established.

#### *Defining Emotion Regulation*

Emotion regulation has been defined and described in a multitude of works by various authors (e.g., Campos, Campos, & Barrett, 1989; Cicchetti, Ackerman, & Izard, 1995; Fielder, 1989; Fox, 1994; Linehan, 1993a; Mayer & Salovey, 1995). Most recently, Cole and her colleagues (2004) forwarded an operational definition of emotion regulation as the changes that are associated with an activated emotion (regardless of what the activated emotion is) such as alterations in physiological functioning and overt behavioral changes. Such changes may also involve psychological mechanisms (e.g., cognitions) of the emotion itself. This conceptualization of emotion regulation views the emotion either

as a regulator or as being regulated. Emotion as regulating involves changes that are due to the activated emotion (e.g., changes in the interpersonal environment resulting from the expression of anger or sadness). Emotion as regulated involves changes in the intensity, valence, and/or duration of the emotion that has been activated resulting from behavioral efforts by the individual including interpersonal (e.g., an individual engages in behavior that makes a sad friend smile), or intraindividual efforts (e.g., an individual engages in self-soothing behavioral or cognitive strategies). The paucity of data supporting this conceptualization of emotion regulation represents a gap in the extant literature.

#### *Emotion Dysregulation and Psychopathology*

The idea that dysfunctional or maladaptive emotion regulatory processes are a main component of psychopathology is a generally accepted and supported perspective among researchers (Gross, 1998). This development is reflected in recent theoretical work, which has adopted an emotion regulation framework for conceptualizing various psychological disorders such as Generalized Anxiety Disorder.

Deficits in emotion regulation can be seen as falling into one of two categories (Cicchetti, Ackerman, & Izard, 1995). One category is the inability to “downregulate” intense emotional experiences, the second category is difficulty with “upregulating” emotions. Difficulties with downregulating are characterized by a high frequency of emotional behaviors (e.g., facial expressions, verbalizations, gross and fine psychomotor behavior) related to the experience or expression of emotion that are disproportionate (i.e., in duration or intensity) to the eliciting stimulus. This problem is typically incurred due to an inability to effectively utilize self-soothing strategies. Difficulty with

“upregulating” emotions or emotional behavior, involves the problematic, chronic suppression of emotional experience or expression. As discussed in subsequent sections, these difficulties have been identified by other notable researchers (e.g., Linehan, 1993a) as central to more severe forms of psychopathology relevant to the present study, such as Borderline Personality Disorder.

However, emotion dysregulation is not limited to the etiology of severe manifestations of psychopathology. Mennin, et al., (2002) propose an emotion regulation conceptualization of Generalized Anxiety Disorder. They posit that individuals with GAD experience emotions more intensely and more aversively than non-clinical individuals and that these negative emotions may be associated with aversive interpersonal consequences (e.g., rejection, isolation) that are difficult to understand or identify. The worry experienced by those with GAD may be conceptualized as a form of cognitive control, whereby the individual attempts to resolve the problematic experience by moderating these intense and aversive emotions. This pattern of cognitive functioning is typical in GAD, and is sometimes conceptualized as avoidance behavior.

The aforementioned cognitive pattern is common, though not endemic to GAD. A similar pattern may be noted among individuals who engage in NSI. As an example, it has been demonstrated that NSI often occurs in response to intense negative emotional experiences, such as anger (Brown et al., 2002). It is plausible that NSI functions as a form of control, much like the worry in GAD, to resolve the aversive emotional experiences. However, in this strategy the individual attends to anxiogenic stimuli (i.e., the object of worry) rather than to the acute aversive emotional experience. This results in perseveration to the ineffective, anxiety-inducing stimulus as the only problem solving



approach, which in turn leads to perpetuation of the anxiogenic sequelae. Furthermore, effective action-tendencies are blocked by avoidance of the aversive emotional experience. Failure to attend to aversive emotions may consequently lead to amplification of the emotional experience (i.e., the intensity and frequency with which neural impulses are transmitted) through an effort of the physiological protective mechanisms of the organism to modify the organism's exposure to the perceived environmental threat. In short, failure to acknowledge the experience of a fear response (1) does not make it dissipate; and (2) does not negate the presence of the anxiogenic stimulus that engendered it. Consistent with this proposal, Mennin, et al. (2000) reported that those in their study who met criteria for GAD reported more intense experiences of emotions, as well as greater difficulties with acceptance, identification, and description of their emotional experiences. Self-soothing of negative emotions was also impaired in these individuals when compared to non-clinical controls, suggesting a further connection with poor regulatory control of emotions.

Further evidence for Mennin et al.'s (2002) model has been published in the mood induction literature. It has been demonstrated that individuals with GAD exhibit increased worry and anxiety-related autonomic responses, and decreased acceptance of current emotional states after exposure to anxiogenic auditory stimuli (e.g., music) than nonanxious controls (Mennin, 2000); and such individuals develop catastrophic worry more readily when negative mood is induced (Startup & Davey, 2001). Together, these lines of research provide evidence that emotion dysregulation may play a role in multiple forms of psychopathology beyond the context of BPD where it is most frequently discussed.

Much of the psychopathology research literature on both emotion dysregulation and NSI has historically focused on Borderline Personality Disorder (BPD). Indeed, BPD is the only DSM disorder that is characterized explicitly by patterns of emotional dysregulation (APA, 2000), despite evidence for such dysregulation in other disorders (e.g., Mennin et al., 2002). Because of the high incidence rates of NSI in BPD patients (up to 80%, Zanarini et al., 2003), these two clinical phenomena are often portrayed as being inextricably linked. Anecdotal evidence suggests that NSI is sometimes viewed as a hallmark sign of BPD. Indeed, NSI has even been referred to as the “behavioral specialty” of individuals with BPD (Gunderson & Ridolfi, 2001). Recent literature (Zanarini et al., 2003), however, suggests that at least 20% of borderline individuals do not engage in NSI, and, moreover, that this behavior is not a stable trait across time in borderlines. Ergo, such stereotypical statements have not necessarily garnered empirical support.

Linehan’s (1993a) continuing work on the treatment of chronically suicidal and self-injuring borderline patients understands both BPD and NSI as a disorder of emotion regulation, with the NSI acting as an emotion regulation strategy. Linehan’s biosocial model presupposes that borderlines “are emotionally vulnerable” (Linehan, 1993a, p.43), that they lack the requisite skills to regulate their emotions, and that there are environmental factors that amplify this deficit and its manifestations. Based on this model, NSI may function as a method of changing subjective internal factors or environmental factors that are threatening to the individual’s emotional or physical integrity. Because many borderline individuals experience even low levels of emotional arousal as overwhelming, NSI may function as an acute, albeit maladaptive, intervention

to reduce emotional arousal by disrupting physiological processes related to the generation of emotion. The sensations induced by NSI (whether aversive or merely novel) may direct attentional resources away from the acute stressor and toward a more pressing stimulus.

Borderline individuals also frequently report experiencing dissociation before, after, or during NSI episodes (Linehan, 1993a). Himber's (1994) qualitative study of self-injuring female inpatients has provided one of the most detailed qualitative accounts of the subjective phenomenology of NSI in this respect. Specifically, 100% (n=8) of participants in this study reported experiencing dissociation in conjunction with NSI episodes. These experiences included "altered sensations, the sense of separateness from their bodies, memory disturbances and distortions in their agency" (Himber, 1994; p. 622). Dissociation may be reinforced by the amelioration of an acutely stressful stimulus through reduction of emotion-related neurotransmission below the perceptual threshold. Consequently, dissociation may be conceptualized as a method of downregulating an aversive emotional experience, although this phenomenon is sometimes reported to be aversive itself (e.g., Himber, 1994). The exact relationship between NSI and dissociative experiences has yet to be fully elucidated. Nonetheless, it is clear that there is a relationship between these two phenomena in at least a subset of self-injurers. Further research on this relationship may help to extend our understanding of NSI and its functions.

Altering the acute environmental stimuli that precipitate or perpetuate an aversive emotional experience is another function sometimes subserved by NSI. Environmental changes such as reduced acute demands of others or of situations may occur as a result of

NSI through common consequences of this behavior, such as the seeking of requisite medical care for or by the self-injuring individual. In this way, the individual may reduce his or her exposure to an aversive stimulus (e.g., abdicating or transferring responsibility for self-care; removing oneself from responsibility of the activities of daily living) via admission to a hospital or other medical treatment facility. Furthermore, the individual may modify their emotional environment. In the case of BPD patients especially, NSI may function to elicit previously perceived unexpressed concern or care from relatives, friends, or partners. Similar to increasing tolerance in chemical dependency, increasingly extreme behaviors may be required to elicit the same responses from others that were initially elicited by less severe actions. It is also this pattern of functioning that may contribute to self-injurers being erroneously viewed or categorized as borderline patients, with insufficient regard given to the actual idiographic clinical presentation.

#### *Psychobiological Aspects of Emotion Regulation*

The role of the central nervous system (CNS) in emotion regulation can be viewed as transpiring at multiple, sequential levels (i.e., the top-down processing model). The top-down processing model may be considered a transactional model in that the neural circuits and cortical pathways responsible for the regulation of emotional experiences interact with each other through perpetual bidirectional inhibitory and excitatory processes and responses. This top-down view also makes clear the simultaneously independent and interdependent nature of these central neural pathways. Different subsystems within the CNS may receive and process different incoming data (e.g., from the environment) or different parts of that data, or a singular set of incoming information may elicit diverse responses across different subsystems (Gross, 1998). The

specific neural circuitry involved in emotion regulation remains unclear. However, Mega and Cummings' (1994) model of subcortical activity regulation suggests that substrates found between the limbic structures and the prefrontal cortex imbue incoming information with "emotional meaning," as well as serving a modulating function.

Neuroscience has advanced our understanding of emotion regulation to a great extent. From a strictly neurobiological perspective, emotion regulation takes place at the systemic level through reciprocal afferent/efferent projections across neural circuits. These cross-circuit projections facilitate reciprocal regulation of each system by the others. As discussed in subsequent sections, the hypothalamus is an integral structure in the neurobiology of emotion regulation. The hypothalamus, along with the brain stem exert regulatory influence on the cortex through specific neurotransmitter substrates (e.g., serotonergic, noradrenergic, and dopaminergic) and dispersion of neuropeptides (Tucker, Derryberry, & Luu, 2000). The actions of the brain stem are moderated by the limbic system, which simultaneously directs cortical resources (i.e., receptors in the cortex and prefrontal cortex) toward incoming stimulation from the environment (Lewis & Steiben, 2004). Peripheral supportive evidence for this has also come from brain imaging studies of individuals with unipolar and bipolar depressive illnesses, who are thought to experience difficulties with emotion regulation in various forms.

The foregoing research indicates that mood disordered individuals exhibit irregular glucose metabolism and regional cerebral blood flow in the limbic system and prefrontal cortex (Baxter, Phelps, Mazziotta, Schwartz, Gerner, & Selin, 1985; Drevets et al., 1997; Mayberg, Lewis, Regenold & Wagner, 1994; Nobler et al., 1994; Soares & Mann, 1997), suggesting evidence of an association between pathophysiology in these

regions and overt indices of emotion dysregulation. These systems work in concert to provide a continuous feedback loop through which evaluative decisions about emotional behavior are made rapidly and adjusted according to incoming sensory information and environmental demands. When a disruption in these systems is encountered, research indicates that typical regulatory functions cannot take place in the same efficient manner.

The prefrontal cortex has also been implicated in emotion regulation. The prefrontal cortex is responsible for regulating subsystems lower on the neural hierarchy by exerting an inhibitory influence on instinctive, or stereotypical, behavioral response repertoires. This inhibition allows for processing of current or new incoming stimulus information that is then used to formulate conscious and purposeful action (Tucker et al., 2000). Some support for this proposed model of involvement of the prefrontal cortex has derived from lesion studies (Gross, 1998). Studies in which part of the prefrontal cortex is incised or ablated have found physical disruptions or ruptures in this structure in adult brains are associated with behavioral impulsivity and dysregulation of affect (Kolb & Taylor, 1990; Rolls, Hornak, Wade, & McGrath, 1994; Stuss & Benson, 1986; Tucker, Luu, & Pribram, 1995). There is also research in the developmental literature (e.g., Dawson, Panagiotides, Klinger, & Hill, 1992; Diamond, 1991) suggesting that changes in the structure of the prefrontal cortex during infancy are associated with the appearance of signs of emotion regulation (e.g., self-soothing techniques). The prefrontal cortex, it seems, is integrally related to the capacity of an individual to upregulate or downregulate their emotional behavior.

A corpus of research has been conducted regarding the neurobiology of emotion regulation and dysregulation in mood disorders. Studies of psychological factors

associated with emotional lability, such as impulsivity and aggression, which are partially attributable to serotonergic metabolites, specifically 5-hydroxyindoleacetic acid (5-HIAA), further support the proposed biological underpinnings of these aspects of BPD (Skodol, Siever, Livesly, Gunderson, Pfohl, Widiger, 2002a). Additionally, research on mood-congruent learning, recall, retrieval, and judgment (cf. Mayer & Salovey, 1988 for a thorough review of this literature) provides further theoretical support for a role of emotion dysregulation in identity disturbance. If borderline individuals are unable to determine and label their mood or emotional experience due to lability or behavioral inconsistency, then retention, recall, and retrieval of items such as personal preferences may also prove difficult, and may influence their perception of experiences. Moreover, chronic negative affect and emotionality may leave the borderline individual more vulnerable to recall and cognitive magnification of the aversive aspects of personal experiences, thus perpetuating the cycle of negative emotions. It is logical to presume that such disruptions, if chronic, may contribute to oscillations between emotional extremes in an effort to attain emotional homeostasis. Whether that emotional stability is attained through healthy strategies such as mindfulness-based skills (Linehan, 1993b; Hayes, Strosahl, & Wilson, 1999), or through maladaptive means such as NSI, varies from person to person. However, it is clear that underlying psychopathology is likely to complicate emotional-behavioral response tendencies.

In sum, emotion regulation seems to play a vital part in organismic survival through its role in the stress response. As research has documented, emotion dysregulation is associated with functional impairments that can impede survival. In humans, emotion dysregulation has been implicated as a key contributing factor for the

development of a variety of forms of psychopathology, many of which are associated with NSI. Explanations for the role of emotion dysregulation have come from a variety of extant bodies of research, most recently the neuroscience literature base. The neural pathways of emotional regulation lie in the hypothalamic-pituitary-adrenocortical axis, which is responsible for activating an organism's biological response to environmental threats and stressors (Kandel, Schwartz, & Jessel, 2000; Lovallo & Thomas, 2000). Appropriate activation of these responses is adaptive and necessary; however, abnormal, or dysregulated, patterns of psychobiological response to stress (e.g., exaggerated or protracted stress responses) may be detrimental to the physical and psychological integrity of the individual in a number of ways as discussed earlier. It is unclear what the relationship of NSI is to emotion dysregulation. One method of examining this is through observation of the psychobiological stress response system in self-injurers. A review of this neural system is provided in the following sections.

#### Summary of Cortisol and Hypothalamic Pituitary Adrenocortical Axis Functions

##### *The Adrenocortical System*

##### *Hypothalamic-Pituitary-Adrenocortical Axis*

At its most basic level, the hypothalamic-pituitary-adrenocortical (HPA) axis is a neuroendocrine circuit (Zeigler & Herman, 2002). The relationship of this circuit to organismic functioning, however, is complex affecting psychological, physiological, and immunological processes. The HPA axis encompasses the adrenal gland, hypothalamus, and pituitary gland. Excitatory afferent nerve projections converge on this circuit from the hippocampus and the hypothalamic parvocellular paraventricular nucleus (PVN). This circuit is responsible for regulating the secretion of glucocorticoids into the blood



stream as a component of an organismic stress response. As part of this stress response, cortisol secretion increases in preparation for management of the stressor or threat. This stress response has been observed to be nearly identical, neuroanatomically and neurochemically, in both animals and humans (McGaugh & Cahill, 1997).

### *Biosynthesis and Functionality of Cortisol*

Cortisol is one of two known glucocorticoids, but is the only glucocorticoid produced in humans (Lovallo & Thomas, 2000). Cortisol is considered a lipophilic adrenal steroid, and has a low molecular weight (Kirschbaum, n.d.). Like other adrenal steroids, cortisol is biosynthesized from low-density lipoprotein (LDL) cholesterol. It is secreted in pulses that are modulated by the frequency and amplitude of pituitary-based secretions of adrenocorticotropin hormone (ACTH). The center of secretory modulation of cortisol lies in the neural triad of the hypothalamus, pituitary gland, and hippocampus. The impulse for secretion originates in the hypothalamic PVN, which houses a large number of neuronal cells specialized for the production of corticotropin releasing factor (CRF). CRF, an orexigenic neuropeptide, is transported to the anterior pituitary gland via the pituitary stalk (Vale, Spiess, Rivier, & Rivier, 1981). Pro-opiomelanocortin, a complex protein produced by corticotrophic cells in the anterior pituitary where CRF conjuncts, is subsequently broken down by the transported CRF into both beta-endorphin (an opioid agonist) and ACTH. These substances are then released into the bloodstream and commence circulation through the body. Circulation of ACTH permits its transportation to the adrenal cortex, at which point it stimulates cortisol synthesis; cortisol is consequently secreted into the bloodstream. This process occurs both in spontaneity and via stimulus-response paradigms (i.e., as a biological reaction to

biochemicals or environmental stimuli; Kirschbaum & Hellhammer, 1989; Van Cauter, 1988).

The present study seeks to examine changes in salivary cortisol levels as an index of emotion regulation via its specificity as a biomarker of HPA axis functioning. A description of the molecular binding processes involved in the production and metabolism of cortisol is conducive to understanding the components of its distribution throughout the body. Understanding this binding process also elucidates the physiological mechanism by which cortisol is transported to saliva and is able to be measured therein. Cortisol shares a binding receptor with aldosterone (another adrenal steroid known as a mineralocorticoid). As noted by Arriza et al. (1997), this shared-receptor contributes substantially to the multifarious functions of cortisol in the human central nervous system (CNS). As described above, blood serves as the vehicle for the transportation and distribution of secreted cortisol, allowing it to penetrate all biological tissues. Upon secretion, most (approximately 90%) of the cortisol released binds to transcortin (also referred to as corticosteroid-binding globulin; CBG), albumin (Kirschbaum & Hellhammer, 1989), or to erythrocyte (red blood cell) membranes (Hiramatsu & Nisula, 1988). The majority of glucocorticoid receptors are located in the hippocampus, which is a primary point of corticoid regulation as well as the structure primarily responsible for the negative feedback component of glucocorticoid regulation (Jacobson & Sapolsky, 1991). This secreting-binding process leaves approximately 5-10% of cortisol unbound, or “free,” in circulation. Unbound cortisol is transported via the kidneys to the urinary tract and subsequently into urine. This unbound portion is also transported into saliva via the parotid gland (Lovallo & Thomas, 2000).

There is consensus in the literature that effective adrenocortical system, or HPA axis, functioning is a prerequisite for healthy and adaptive responses to stress. This system responds to stressors through a tri-faceted pathway (de Kloet, 1991). This pathway begins with the pituitary gland and hypothalamus, which receive biochemical stimulation via neurotransmitters and neuropeptides. The limbic system conducts afferent and efferent neural messages between the hypothalamus and the cerebral cortex. Finally, the brain stem conducts internal and external (i.e., sensory) stimulation to the hypothalamus. Effective HPA axis functioning includes an ability to generate an elevation in cortisol at the onset of a threat or stressor (i.e., upregulation), as well as to initiate a decrease in cortisol production to facilitate a return to baseline levels at the termination of the threat or stressor (i.e., downregulation). Such functions are a necessity for adaptation to everyday life events through preparing the organism to negotiate the demands of the external or internal environment.

In this vein, Stansbury and Gunnar (1994) have noted that cortisol in the HPA axis acts in conjunction with other physiological systems to extract the energetic resources necessary for a response to environmental challenges. Cortisol also regulates the immunological system, the endogenous opioid system, and central and peripheral catecholamine systems (Kandel et al., 1991; Lovallo & Thomas, 2000) thereby facilitating the maintenance of poly-systemic homeostasis. Furthermore, the presence of both glucocorticoid and mineralocorticoid receptors in the amygdala, hippocampus, and hypothalamus suggests that, in addition to ACTH and CRF, cortisol's neural-hormonal activity also influences emotional functioning, memory, and learning processes (Lovallo & Thomas, 2000; Stansbury & Gunnar, 1994).

The preceding description contributes to the conceptual basis the present study in that receptor location is related to receptor activity, that is, a receptor's activity will affect (i.e., facilitate or impede) the functioning of the area of the brain in which it is located (Kandel et al., 1991). The cortisol and mineralocorticoid receptors in the amygdala are likely to affect emotional functioning and emotional behavior. Researchers have posited that the effects of glucocorticoids are facilitated by the actions of glucocorticoids on steroid receptors in the reticular formation of the brain stem (Stansbury & Gunnar, 1994). Moreover, research suggests that when glucocorticoids are elevated in stress-response quantities, there are definitive effects on hippocampal functioning, such as delayed and immediate recall (Lupien et al., 1998). There are transitory decreases in long-term neuronal potentiation, which may be connected with variability in working memory functioning (e.g., variability observed diurnally and at post-stress intervals). Additionally, there is evidence that hippocampal neurogenesis is inhibited, and that dendritic degeneration may occur (Lovallo & Thomas, 2000; Lupien et al., 1998; McEwen, 1997). The degradation of neural pathways through dendritic degeneration may impede the process of regulating the functions served by those pathways.

#### *Factors Affecting Cortisol Secretion*

Research has established that cortisol is secreted in a diurnal cycle (i.e., circadian rhythm) regulated by the hypothalamic suprachiasmatic nuclei, which is dependent on ACTH secretion (Stansbury & Gunnar, 1994). Within the diurnal cycle, the peak in basal cortisol secretion occurs during the last few hours of nocturnal somnolence until approximately 30 minutes after awakening, exhibiting an increase from daytime baseline of 50-100% (Schmidt-Reinwald et al., 1999; Wust et al., 2000). There is remarkable

intra-individual consistency in this pattern of secretion, indicating that cortisol may be a preferred index of HPA axis functioning (Pruessner et al., 1997; Schmidt-Reinwald et al., 1999). Research has revealed that this rhythm emerges around three months of age in humans, and is fully attained by approximately two years of age (Stansbury & Gunnar, 1994).

Despite the documented stability of cortisol secretory rhythm, there are some additional physiological conditions which may alter the typical pattern of cortisol secretion across the diurnal cycle. These include both pregnancy and ingestion of oral contraceptives. Both of these conditions alter the synthesis of CBG in the liver, such that higher levels of this substance are produced, thus making available a greater supply of CBG for cortisol to bind to. Because the liver is unable to metabolize cortisol molecules that are CBG-bound, cortisol levels in plasma are elevated. Nonetheless, the available research on cortisol levels in pregnancy has historically been mixed at best.

Earlier researchers reported null findings in comparisons of cortisol assays for pregnant versus non-pregnant women (Guechot et al., 1981, 1982; Landon et al., 1984; Peters et al., 1984). However, more recent research (Nierop, Bratsikas, Klinkenberg, Mater, Zimmerman, & Ehlert, in press) indicates that women in the third trimester of pregnancy exhibit higher baseline levels of salivary cortisol and a greater degree of cortisol reactivity compared to those in the second trimester and non-pregnant women. Furthermore, this research suggests that cortisol recovery time (i.e., time required for return to baseline levels) was significantly protracted for women in the second trimester of pregnancy compared to non-pregnant women, although it did not differ significantly from that observed in third trimester women. These findings partially buttress earlier

reports in the literature (e.g., Bustamante & Crabbe, 1984; Stahl & Doerner, 1982; Vining et al., 1983) of elevated cortisol levels during the third trimester. Kirschbaum and Hellhammer (1989) contend that such elevations are the result of biomolecular supply and demand principles. An increase in plasma progesterone levels occurs during both pregnancy and oral contraceptive intake, leading to an increase in the number of molecules (of cortisol and progesterone) competing for the same CBG and target cell binding sites. Hence, there is greater bio-demand, but unchanged bio-supply, creating a circumstance in which some molecules are naturally excluded from the binding process and remain unbound.

In addition to physiological factors, psychosocial factors such as socioeconomic status, educational level, and ethnic origin also appear to influence the secretion of cortisol. Bennet, Merritt, and Wolin (2004) examined waking cortisol peak (30 minutes after awakening) and baseline levels in 63 non-Hispanic Caucasian and African American males and females. These researchers also examined the independent contribution of educational level on cortisol variation. After adjustment for education and managerial status, results indicated that higher levels of cortisol secretion were found in Caucasians. Additionally, those with higher levels of education had significantly higher cortisol peak levels after adjustment for ethnicity and BMI. Bennet et al. (2004) further reported no significant between-groups differences in overall cortisol secretion for the Ethnicity x Education interaction. However, additional analyses revealed that African Americans with lower educational levels had significantly lower cortisol levels at awakening than any other group (i.e., lower educated Caucasian, higher educated Caucasian, and higher educated African American). After 30 minutes, however, lower-

educated African Americans were only significantly different from higher-educated Caucasians; whereas lower-educated Caucasians were now significantly different from higher-educated Caucasians. All significant interactions and non-significant differences remained after controlling for perceived level of stress. The source of these differences is not clear, and these findings must be replicated before any conclusions may be drawn. However, these findings are of relevance to present research in that the population from which the sample was drawn was comprised of generally homogenous ethnic origins (i.e., European).

#### *Cortisol and Psychological Stress*

Inconsistent and contradictive findings within the literature have made the utility of cortisol as a biomarker for psychological stress somewhat unclear. Some researchers (e.g., Hjortskov, Garde, Ørback, & Hansen, 2004) have suggested that such inconsistencies may be the result of substantial variability in (1) types of psychometric instruments used to assess mental stress; (2) study design, or design-related issues (e.g., controlling for extraneous variables such as oral contraceptive use); or (3) choice of cortisol derivative used in statistical analyses.

In their brief review of the literature, Hjortskov et al. (2004) examined studies that included both cortisol and measures of self-reported psychological stress. The authors identified 73 studies, of which 14 met stringent criteria for inclusion in their review. Hjortskov et al. (2004) set an a priori criterion of 75% agreement between all studies as being indicative of consistency of evidence for or against a relationship between cortisol and self-reported psychological stress, with anything less than 75% being indicative of ambiguity. Four studies (27%; i.e., Ockenfels et al., 1995; Schulz et al., 1998; Steptoe,

Cropley, Griffith, & Kirschbaum, 2000; Zeier, 1994) reported a positive relationship, and two studies (13%; i.e., Steptoe et al., 1998; Yang et al., 2002) reported a negative relationship. Eight studies (60%), however, (i.e., Burton et al., 1996; Evans & Steptoe, 2001; Fischer et al., 2000; Fischer et al., 2002; Fox, Dwyer, & Ganster, 1993; Hanson, Maas, Meijman, & Godaert, 2000; Pruessner et al., 1999; van Eck et al., 1996) reported no relationship. The authors concluded that there are several reasons why a clear relationship between cortisol and self-reported stress may not be discernible at present. For example, in addition to design and procedural explanations, many studies of stress and cortisol relationships have examined work-related stress and/or stressors, or have examined cortisol response to on-demand performance of mental tasks (e.g., mental arithmetic). Some researchers (cf. Adam & Gunnar, 2001; Frankenhauser et al., 1986) have posited that there is a prerequisite level of stress required for activation of the HPA axis, which would likely not be met by common work-related stress. Operating from this theory base, the low to moderate mean levels of stress reported in many of the studies evaluated by these authors would have been insufficient for HPA axis activation (Hjortskov et al., 2004).

It is clear that a basic question must be asked regarding whether or not cortisol is a valid index of stress. Based on the research described above, it may be reasonably postulated that there is a relationship between psychological stress and cortisol levels. However, this research does suggest that there is potentially a threshold at which cortisol secretion is activated and/or elevated. It is possible that individuals who experience chronic emotional or psychological stress develop a lower threshold for cortisol activation over time in response to that stress. It is also possible that individuals who



utilize maladaptive coping strategies for handling stress, such as NSI, have an inherently lower threshold for cortisol activation, thus leading to a cortisol response that is exaggerated in either intensity, duration, or both.

### *Cortisol and Psychopathology*

Research over the past two decades has provided consistent evidence of a relationship between various clinical disorders and cortisol. It is important to note that the relationship between cortisol and psychopathology is not exclusive, but rather is a function mediated by HPA axis disruption or functionality (e.g., Davidson et al., 2002). Cortisol is not considered a precipitant of psychopathology, but is an index of systemic dysfunction that may be associated with, or manifested in, various forms of psychological disturbance. However, chronic elevation and inhibition of cortisol may be a perpetuating factor for psychopathology in that these endocrinological states have the potential to produce adverse immunological and physiological effects (e.g., immunodeficiency, parasomnia, anxiety, and other stress-related health problems). Such effects have clear implications for psychological functioning as well.

There is a relatively strong literature base that links cortisol levels with some forms of psychopathology, particularly among individuals with depression or anxiety disorders. Clinical research on the relationship between cortisol and psychological functioning first centered on depressed patients (Stansbury & Gunnar, 1994). Aggregated research of the relationship between cortisol and depression has indicated that, when compared with non-clinical individuals, depressed individuals exhibit inflated cortisol responses to stimulation of ACTH and higher plasma and urinary levels of free cortisol secretion during diurnal rhythmic secretion periods (Davidson, Lewis, & Alloy,

2002; Drevets, Price, Bardgett, Reich, Todd, & Raichle, 2002; Garlow, Musselman, & Nemeroff, 1999). With few exceptions (Birmaher et al., 1996), it is now well-established that there is a disruption of HPA axis functioning in mood disorders. This is perhaps most prolific in Bipolar Disorder (Young, 2004), as evidenced by higher basal levels of cortisol, and suppression failure in dexamethasone suppression tests (Carroll, 1981). It is posited that HPA axis disruption is the result of a dysregulation in the reciprocal connective mechanisms of the HPA axis, where depressed individuals do not exhibit suppression of precursor hormones when administered cortisol exogenously (Young et al., 1991). Several studies also suggest that a pattern of HPA axis hyperactivity may precipitate disruption of the feedback mechanism in depressed patients (Davidson et al., 2002; Soares & Mann, 1997; Young, 2004), such that hypercortisolemia develops, creating a psychoneuroimmunological vulnerability in these individuals. Similar adrenocortical disruptions may also be associated with other related and frequently comorbid disorders, such as Anorexia Nervosa (Stansbury & Gunnar, 1994). Thus, there is evidence that disorders in which dysregulation of emotional and emotion-related behavior are present may be characterized by a common theme of abnormal psychobiological stress responses.

While the literature pertaining to mood disorders is relatively clear, that pertaining to other forms of psychopathology is mixed (Heim & Nemeroff, 2001). For example, Obsessive-Compulsive Disorder (OCD) patients have been shown to exhibit basal hypercortisolemia. However, Panic Disorder patients tend to exhibit normal basal levels of cortisol (Heim & Nemeroff, 1999), whereas those diagnosed with Posttraumatic Stress Disorder (PTSD) have been shown to exhibit hypocortisolemia (Yehuda, 2000). There

has been a failure to replicate this latter finding in earlier studies by other researchers (Lemieux & Coe, 1995; Maes et al., 1998), despite recent supporting evidence from Neylan et al. (2005), who found that PTSD symptom severity significantly predicted lower pre-dexamethasone awakening cortisol levels in traumatized police officers. Furthermore, Gerra et al. (2000) reported increases in ACTH levels but not cortisol levels in children diagnosed with GAD who were exposed to an anticipated laboratory stressor, thus contributing further ambiguity to an already equivocal literature.

Perhaps of greatest relevance to the present study is a nascent line of research (currently consisting of a single case study) examining salivary cortisol in NSI among individuals with BPD. In a brief report presented as a letter-to-the-editor, Sachsse, von der Heyde, and Huether (2002) described the diurnal cycle of urinary cortisol secretion in a female borderline patient. This case study revealed an atypical pattern of cortisol excretion in this patient, characterized by substantial fluctuations in nocturnal excretion; her mean nocturnal excretion was also lower than normative levels. Interestingly, a pattern of excretion and correlated behavior emerged such that periods of consistently low nocturnal secretions were followed by periods of consistent elevations in cortisol excretion; excretion levels above 20 nanomolecules/liter were associated with NSI episodes that involved “one or more acts of self-mutilation” (Sachsse et al., 2002, p. 672). Immediately following the NSI episode, nocturnal urinary cortisol levels returned to the lower end of her baseline measurements and remained at this level for “several days.” This small case example represents the first attempt to investigate the relationship between NSI and biological indices of stress regulation. Expansion of this line of inquiry in much larger samples is necessary for this relationship to be elucidated.

The research cited above provides important supporting data for the premise that dysregulation of emotion is an integral component of psychopathology. Evidence from neuroendocrinological studies has revealed consistent differences in HPA axis functioning of individuals with mood disorders, which are commonly and frequently characterized by disruption of typical emotional processes and behavior. NSI is a characteristic that is found in many forms across the spectrum of clinical disorders. There appears to be an association between dysregulated emotional behavior and the occurrence of NSI, however to date only correlational evidence exists. More empirical approaches to examining the relationship between NSI and emotion dysregulation are needed. One such approach is observation of psychobiological correlates of the human stress response (e.g., cortisol) after initiation of the stress response system. Stress induction has been conducted in laboratory procedures for over five decades in stress-related research, and has evolved in effectiveness over that time. The following section reviews the research documenting the development of various stress-induction procedures, as well as the evidence for their effectiveness with various populations.

#### Salivary Cortisol and Stress Induction

Research using animal models lends support to the hypothesis that there could be stressor specific paths to cortisol secretion (see Weiner, 1992). In their recent meta-analysis, Dickerson and Kemeny (2004) proposed a theory of cortisol response that is rooted in a *social self-preservation system*. The function of this system is to continuously monitor the individual's environment for threats to their social esteem or social status. This system coordinates psychological, physiological and behavioral responses to

manage these threats through specific biological processes including activation of the HPA axis.

In their proposal of the social self-preservation system, Dickerson and Kemeny (2004) provided a thorough review of the stress-induction and cortisol secretion literature base. Their review revealed that the type of stressor as well as the degree of control participants had over the stressor were the factors with the greatest influence on cortisol secretion. Dickerson and Kemeny (2004) compared the effects produced on cortisol secretion by different types of laboratory stressors in different quantities (i.e., some studies included a single stressor; some included two or more stressors). In aggregate, the data in this literature base pertaining to stress induction and salivary cortisol suggests that conditions that included social-evaluative threat, (i.e., where performance was captured on permanent record, an evaluative audience was present, or a person offering negative social comparisons was present) is associated with significantly higher effect sizes for cortisol responses. Specifically, both conditions with and without social evaluative threat (SET) elicited significant cortisol increases. However, conditions with SET produced significantly greater cortisol increases than those without. Additionally, different numbers of types of SET resulted in progressive increases in effect size for cortisol responses. In an applied sense, larger effect sizes translate to a stronger cortisol response. Studies with a single SET type (e.g., person offering negative social comparisons, performance captured on videotape) produced an effect size of 0.23, while those with two SET types produced an effect size of 0.86. These differences suggest a dose-dependent relationship of sorts, with a higher number of SET stressors being associated with a more substantial effect. Qualitatively, the presence of an evaluative audience and negative

social comparison both yielded higher effect sizes than inclusion of a videotape component; and the presence of an evaluative audience was equivalent to a negative social comparison ( $p=0.20$ ).

Furthermore, data from the Dickerson and Kemeny (2004) study indicated that controllability was strongly related to cortisol responses. Cortisol responses for uncontrollable tasks produced a larger effect size ( $d=0.52$ ) than controllable tasks ( $d=0.16$ ;  $p<0.01$ ). However, there appears to be no progressive increase in effect size with the inclusion of multiple uncontrollable elements, suggesting little if any advantage of adding additional uncontrollable elements ( $p=0.18$ ). SET was not significantly different from uncontrollability ( $p>0.20$ ) for predicting cortisol response, suggesting that these are both significant independent predictors. SET combined with uncontrollability accounted for 26% of variance in cortisol after time of day was controlled for, and represented a significant increase beyond time of day alone ( $p<0.01$ ).

Dickerson and Kemeny (2004) also reported that neither motivated performance tasks (active performance tasks with the potential for evaluation along a self-relevant domain) nor passive performance tasks alone without SET or uncontrollability elicited significant cortisol responses. However, experiments that involved a motivated performance task combined with both SET and uncontrollability yielded the largest effect size ( $d=0.92$ ). Moreover, motivated performance tasks combined with SET and uncontrollability yielded a higher effect size than motivated performance tasks with either of these elements alone. Hence, the combined SET-uncontrollable condition resulted in the highest effect size of any condition. Statistical analyses suggested that SET and

uncontrollability mediated the relationship between stressor task category and cortisol response.

Taken together, this examination of the relative importance of specific factors of laboratory stressors (as related to cortisol secretion) revealed that both the perception of the controllability of the stressor by the participant, as well as the type of stressor administered, are crucial. Moreover, the analysis provided by Dickerson and Kemeny (2004) strongly indicate that combining these factors in the right permutation, such that a stressor is both perceived as uncontrollable and is socially evaluative will produce a cortisol response that is nearly a full standard deviation above the mean resting level. Congealed, these data permit the conclusion that there is evidence of the effectiveness of laboratory stressors in producing strong cortisol responses. Finally, the results of Dickerson and Kemeny's (2004) analysis delineate a prudent route for future research involving induced stress with salivary cortisol as an outcome measure.

In addition to cortisol response, Dickerson and Kemeny (2004) examined the pattern of post-stressor cortisol responses. In aggregate, research indicates that cortisol levels are two times higher during the period of 0-20 minutes post-stressor than 21-40 minutes, and continued to decline at 41-60 minutes post-stressor. Across studies, cortisol responses were significantly higher than baseline during the 0-20 minute and 21-40 minute periods, but not at 41-60 minutes post-stressor.

During the 0-20 minute post-stressor period, performance tasks combined with SET and uncontrollability elicited significant cortisol response ( $d=0.85$ ). Furthermore, performance tasks combined with either SET or uncontrollability alone also elicited a significant cortisol response ( $d=0.25$ ). A significant elevation in cortisol was still present

during the 21-40 minute post-stressor period for performance tasks combined with SET and uncontrollability ( $d=0.74$ ;  $p<0.01$ ). However, performance tasks combined with either SET or uncontrollability alone no longer elicited a significant cortisol response ( $d=0.08$ ). Finally, a significant cortisol elevations remained significant during the 41-60 minute post-stressor period for performance tasks combined with SET and uncontrollability ( $d=0.28$ ;  $p<0.05$ ); and performance tasks combined with either SET or uncontrollability alone remained non-significant ( $d=-0.21$ ).

In sum, the duration of a laboratory stressor does not appear to predict effect sizes of cortisol responses at any of the three intervals. The inclusion of a socially evaluative and uncontrollable component seems to have an impact on the recovery process, as well as on the magnitude of the initial cortisol response. However, the persistence of cortisol elevations after a combination SET uncontrollable task is mostly the result of the greater peak response they produce. In consideration of these data, it may be inferred that those laboratory stressors that yield the most robust initial cortisol responses would include both a social-evaluative component and an uncontrollability component. Furthermore, it may be inferred that there is indeed a relationship between an initial cortisol response and the duration of the cortisol response (i.e., the amount of time required for cortisol to return to a level that is not significantly different from baseline).

There are a number of potential alternative explanations for these results that exist and which must be considered before any firm conclusions may be drawn. As noted by a number of researchers (e.g., Egger, Davey, & Smith; Field, 2003; Thompson & Pocock, 1991), such potential explanations relate to a number of concerns regarding the trustworthiness of meta-analytic procedures. First, it is possible that artifacts of the



studies, or the construct under investigation, contributed to the effects reported. In the case of laboratory-induced stress and its effect on salivary cortisol, sources of artifact may include the subjective distress experienced by the participants in the studies, the reliability of the instrumentation and measurement. Additionally, publication bias, in which meta-analysts select only publications from peer-reviewed journals and exclude dissertation research and studies presented at conferences, may exaggerate effects by precluding the inclusion of research that revealed null results. This bias toward publication of only significant results (i.e., the “file-drawer” problem) exhibited by both researchers (Dickerson, Min, & Meinert, 1992; Rosenthal, 1979) and editors (Hedges, 1984) has been noted for over two decades. Omission of unpublished research may indeed affect the results of aggregated data, as this data may produce effects that are substantially lower than that of published research (Shadish, 1992). The methodology employed for a meta-analysis is another factor that may influence effect sizes. A full review of approaches to meta-analysis is beyond the scope of this critique; however, research indicates that effects may be inflated due to varying control of Type I error rates depending on the number of studies included in the analysis (Field, 2000). It is important that meta-analytical researchers address these concerns statistically, and report them in their results to allow evaluation of the procedural soundness.

These potential confounds were addressed by the authors in their description of the methodology they employed. Additional analyses from the Dickerson and Kemeny (2004) meta-analysis indicated that participants’ distress ratings were not a significant predictor of cortisol response effect size, suggesting that these were independent of social-evaluation, type of task, and controllability factors. Additionally, it is unlikely that

publication bias contributed to these results, given that both peer-reviewed and unpublished dissertations were included in the analyses; and publication status was not a significant predictor of cortisol response effect size. Dickerson and Kemeny (2004) also analyzed the contribution of authorship to the effects due to a large number of studies deriving from a single laboratory. Results revealed that although Kirschbaum and Hellhammer authored 9% of the studies in this meta-analysis, authorship did not predict cortisol response effect size significantly when controlling for time of day and type of stressor.

The authors' description of their selection, instrumentation, and measurement methodology indicates that there was substantial consistency in the sample and methodological characteristics; all studies included in the analysis used "healthy" participants, and all used salivary cortisol as an outcome measure. Studies in the meta-analysis were differentially coded for time of day to allow for ANCOVA to be conducted with this factor as a covariate. Finally, the methodology employed for conducting this meta-analysis has been demonstrated in statistical simulation research (Field, 2000) to be equivalent in its control of the Type I error rate when large samples (100 or more) are used; Dickenson & Kemeny (2004) report an n of 208 studies. Evaluation of the way in which the authors addressed the factors that may mitigate the results of a meta-analysis suggest a sound and empirically-supported scientific methodology was employed based on the best statistical technology currently available. Thus, it is reasonable to conclude that the results of the Dickerson and Kemeny (2004) meta-analysis are valid. The soundness of the meta-analysis as determined by the present methodological evaluation

also speaks to the applicability of these results to research paradigms involving the investigation of salivary cortisol and laboratory stressors.

### The Present Study

There is a substantial corpus of research comprising the NSI and emotion regulation literatures. The emotion-regulation model of NSI is one of the most widely accepted hypotheses; however, this model is supported primarily by self-report anecdotal accounts of the phenomenology of self-injurers. While the nature of this evidence does not discount its validity, a better understanding of the mechanisms underlying the emotional dysregulation experienced and reported by self-injurers is necessary.

Elucidating the psychobiological stress response of self-injurers may provide empirical support that is critically needed for the emotion regulation model of NSI. Much of the phenomenology of NSI and self-injurers does appear to point toward the validity of an emotion regulation hypothesis. Corroborating data from an alternative source is necessary for progress to be made in the further development and refinement of this approach to understanding NSI. Aggregated supportive data for the emotion regulation model may prove to be invaluable to the development of new and more specific psychobiologically-based treatment modalities. A better understanding of the biological aspects of the emotional response system will contribute data that could potentially be applied in both psychotherapeutic and psychopharmacological modalities. To the knowledge of the primary investigator, there is no published research examining the psychobiological effects of acute emotional distress in self-injurers.

An effective investigation of psychobiological factors involved in emotion regulation necessitates that both the requisite theoretical foundation for such research has

been developed based on empirical data, and that the requisite technology be available for such scientific inquiry. The research summarized in the preceding sections has established that both of these criteria have been met by the current body of literature. First, research pertaining to NSI has progressed substantially over the past two decades. A number of explanatory models have been forwarded, ranging from drive-based psychodynamic, to behavioral contingency, and, of course, emotion regulation models of this phenomenon. When considered together, a common theme of regulation, particularly self-regulation, emerges from these models. This theme is echoed by clinical self-reports of many self-injurers as well as empirical research using psychometric measurements (Linehan, 1993a; Nock & Prinstein, 2004; Walsh, 2006).

The second component of the present thesis, emotion regulation, has also received substantial attention in the past two decades (Gross, 1998). Models of emotion regulation have also been developed during this time, and appear to have congealed into a psychobiological information-processing model, which contends that physiological and environmental stimuli inside of and external to the individual are evaluated on an ongoing basis. This model submits that these stimuli and responses are incorporated into a template for satiation of the needs of the individual. Emotional behavior, and its physiological correlates, reflect the modification of the individual's response to such incoming information, and are thereby regulated via this evaluative process. Research from the field of neuroscience, as well as emotion regulation in general, supports the contention that there are psychobiological indices of emotion regulation that are observable in the aforementioned correlates (Kandel et al., 1991; Lovallo & Thomas, 2000). The culmination of such research indicates that the HPA axis and its afferent and

efferent projections are the core of emotional functioning and stress responses in the brain (Gross, 1998; Lovallo & Thomas, 2000).

Furthermore, a prolific quantity of research has amassed documenting the correlation between levels of stress and secretion of glucocorticoids as part of a physiological response to stress initiated by the HPA axis. Research has identified cortisol as the primary glucocorticoid involved in this HPA axis response to stressors (Kirschbaum & Hellhammer, 1989, 1994). This research has also established that the level of unbound cortisol found in saliva is a valid estimate of plasma levels of cortisol (Yao, Moss, & Kirillova, 1998). The data from these lines of research indicate that the technology to evaluate HPA axis functioning via the cortisol response indeed exists and is translatable into clinical research.

With regard to measurability of psychobiological markers of emotion regulation, the stress-response and general psychophysiological research literatures support the contention that effective technology exists. There is ample research suggesting that saliva-based biological samples of multiple hormones (e.g., cortisol, progesterone, testosterone) are accurate estimates of plasma levels of these biochemicals (Kirschbaum & Hellhammer, 1989, 1994). Advances in biological sample assaying methodology have also been developed aggressively over the past 10 years, permitting greater precision in cortisol measurement particularly. The radioimmunoassay procedure has been the dominant technique since the mid 1960s (Deuss, Allolio, Feltes, & Kaulen, 1984; Katz & Shannon, 1964; Walker, Riad-Fahmy, & Llewelyn, 1978); however, newer methods involving nonisotopic techniques (e.g., fluoroimmunoassay) have improved both the sensitivity and convenience of measuring salivary cortisol (Yao et al., 1998). Thus, the

technology for measurement of salivary cortisol has been demonstrated to be available, reliable, and precise.

The present review has evaluated data from multiple related bodies of research. When examined within the framework of a mutual context, these literature bases point toward recent notable progress in developing our understanding of the factors associated with NSI, especially the psychobiological facets of this phenomenon. The present review presents a theoretical foundation for exploring the role of emotion regulation in NSI in a novel way. Furthermore, this review provides ample empirical evidence from the psychobiological research literature suggesting that there are valid, reliable, and measurable biomarkers of emotion regulation. Perhaps most importantly, in bringing together these literatures, the present review has highlighted an overlooked gap in the literature. Specifically, with the exception of one case study examining self-mutilation in a borderline individual, no research has investigated the relationship between psychobiological indices of emotion regulation and NSI. Moreover, although the technological means exist to do so, there has been no experimental investigation of this relationship. This gap represents an important missing component of support for the emotion regulation model of NSI.

The primary purpose of the present study was to evaluate the validity of the emotion regulation model of NSI using cortisol secretion following induced psychosocial stress as an index of HPAA functioning in NSI individuals compared to non-NSI individuals. The primary investigator proposed that this data would diminish the gap in the literature on psychobiological functioning in self-injurers by evaluating hypothesized differences in cortisol responses between these two groups. Aggregated stress-

induction/cortisol research suggests that the most effective laboratory stressors with the strongest effects on cortisol secretion are those that are both (1) perceived as uncontrollable by the participant, and (2) evaluative in nature (Dickerson & Kemeny, 2004). Therefore, the present study employed a social rejection stressor paradigm that has been employed successfully in several previous studies (Blackhart, Eckel, & Tice, 2007; Maner, Dwall, Baumeister, & Schaller, 2007; Nezlek, Kowalski, Leary, Blevins, & Holgate, 1997; Twenge & Campbell, 2003), and is commonly used (Baumeister, Brewer, Tice, & Twenge, 2007) in research, examining biological and psychological stress responses. Furthermore, in the present study cortisol response was conceptualized as the quantity of cortisol secreted at each post-stressor measurement interval.

### *Hypotheses*

#### *Hypothesis 1*

NSI group participants will exhibit significantly greater initial increases in salivary cortisol from baseline measurement than Control group participants. This was determined by the difference in the number of nanomolecules of cortisol per deciliter ( $\mu\text{g/dL}$ ) of saliva following the post-conversational task baseline versus subsequent, post-stressor measurement points.

#### *Hypothesis 2*

NSI group participants will exhibit significantly higher levels of self-reported problems with emotional regulation than Control group participants, as determined by significantly higher scores on all six subscales of the Difficulties in Emotion Regulation Scale (DERS).

#### *Hypothesis 3*

DERS total scores will correlate significantly and convergently with salivary cortisol quantum at measurement points at which the quantum is significantly different from baseline.

*Hypothesis 4*

PANAS positive affect (PA) and negative affect (NA) scores for both groups will correlate significantly at each measurement point with salivary cortisol level differences from baseline, with PA scores correlating inversely, and NA scores will correlating convergently.



## CHAPTER II

### METHODS

#### Participants

A total of 55 participants completed this study, including 26 self-injuring participants and 29 participants who reported having never engaged in any form of self-injury. One control group participant was omitted from analyses because it was discovered (after she had already participated in the study) that she had actually reported a single episode of self-injury during adolescence. Thus, a total of 54 participants were included in the analyses. Although recruitment strategies (as described below) were designed to recruit a diverse sample that included non-student participants, all participants in this study were inevitably derived from the student population at the University of North Dakota.

The entire sample ( $n=54$ ) was comprised of 61.1% ( $n=33$ ) males and 38.9% ( $n=21$ ) females. Participants were predominantly Caucasian (94.4%;  $n=51$ ), with 3.7% ( $n=2$ ) reporting their ethnicity as Asian, and 1.9% ( $n=1$ ) reporting their ethnicity as Native American. Participants' ages ranged from 18-47 years old ( $M=20.69$ ;  $SD=4.65$ ). Most (65.4%) participants in this study reported being in their first two years of college.

When analyzed individually by group, NSI participants ( $n=26$ ) were typically in their early 20's ( $M=20.85$ ;  $SD=5.66$ ) mostly female (57.7%), and mainly Caucasian (96.2%), with 3.8% ( $n=1$ ) reporting Asian ethnicity. Almost three-quarters (72%) were in their first two years of college. Control group participants had a comparable mean age

of 20.54 years ( $SD=3.57$ ), which was not significantly different from NSI participants ( $t(52)=.24; p=.81$ ). Control participants were also typically in their first two years of college (59.2%), predominantly male (78.6%) and Caucasian (92.9%), with 3.6% of control participants reporting Native American ( $n=1$ ) and Asian ( $n=1$ ) ethnicities.

Pearson chi-square analyses were conducted for sex proportions in the full sample and sample subgroups. Confidence intervals ( $CI=99\%$ ) were calculated using an online statistics calculator (GraphPad®) that employs the Wald method, as recommended by Agresti and Coull (1998) for smaller samples. These analyses revealed no significant differences between or within groups for participants' sex, with one exception: chi-square analyses revealed a statistically significant difference in the proportion of males (78.6%;  $CI_{99}= 53.69 - 92.51$ ) versus females (21.4%  $CI_{99}= 7.49 - 46.31$ ) in the Control group ( $\chi^2 [1, 53] = 7.46; p=.006$ ). It is likely that this disparity is related to the exclusion criteria for this study (described below). In short, potential female participants had two additional exclusion criteria that were not applicable to males, specifically pregnancy and oral contraceptive use. It is possible that the restriction of oral contraceptive using women from participating in this study disproportionately biased recruitment of participants for this study, resulting in fewer women than men in the Control group.

Participants were asked to rate their family's socioeconomic status on a scale from 1 ("very poor") to 7 ("extremely wealthy"). NSI group participants ( $M= 4.42; SD=1.03$ ) were not significantly different than Control group participants ( $M= 4.36; SD=.73$ ) on this measure ( $t(52)=.27; p=.79$ ). Additionally, participants were asked to rate the quality of their current friendships on a scale from 1 ("no close friends") to 5 (very strong/close friendships). An independent-samples t-test revealed no statistically

significant difference between NSI ( $M= 4.08$ ;  $SD=.74$ ) and Control participants ( $M= 4.43$ ;  $SD=.63$ ) for this psychosocial measure ( $t(52)= -1.87$ ;  $p =.067$ ).

## Procedures

### *Recruitment Procedure*

Participants were recruited via three methods. First, at the beginning of each of three academic semesters, students in undergraduate psychology courses were given the opportunity to earn extra credit in one of their psychology courses by voluntarily participating in a psychological screening procedure. Informed consent was obtained from all participants prior to administration of assessment protocols. During this screening procedure, all consenting participants were administered a demographic questionnaire (the “About Me” questionnaire), and a modified version of the Deliberate Self-Harm Inventory (DSHI; Gratz, 2001; see “Measures” below). Second, participants were also recruited via scheduled screening sessions in the University of North Dakota (UND) Department of Psychology, during which time participants came to an advertised study location, and volunteered to complete the screening measures described above for extra credit. Data from all screenings were aggregated and analyzed to identify eligible participants.

The study was also advertised on informational posters displayed throughout campus, in a variety of locations (e.g., grocery stores, coffee shops) in the surrounding community, and online on the UND website. These posters and the online posting contained a phone number to call for the information about the study, as well as information regarding some basic aspects of the study, including compensation and approximate duration.

Potential participants were screened for eligibility through a combination of data review and telephone interviews. Screening data were reviewed using frequency analyses of variables that represented study pre-requisites (e.g., NSI history) to identify potential participants who met study criteria. Participants meeting preliminary eligibility criteria (n=372) were contacted for brief telephone interviews to determine their full eligibility. During telephone contact with pre-identified eligible participants, all participants were re-assessed for current oral contraceptive use and mood, anxiety, and eating disorder diagnoses. Participants who had initiated contact via telephone or e-mail in response to an advertisement were also administered the DSHI during the telephone interview to determine their eligibility (n=9).

During the telephone screening, participants deemed eligible for the study were given a brief description of the tasks involved in the study, were informed of all requirements of the study and were told they would be compensated for their time. Participants were informed that at the time of their participation, they would be given a choice of being compensated with either four (4) hours of research credit; or \$20. To minimize the effects of extraneous factors known to affect cortisol levels, participants were specifically instructed to abstain from (1) consuming any stimulants (e.g., caffeine, methamphetamines, amphetamine) or alcohol, (2) engaging in any strenuous exercise/physical activity (defined as any activity that leads to an increase in respiration and/or heart rate) 24 hours prior to the experiment; (3) consuming anything but water two hours prior to the experiment; and (4) using any tobacco or nicotine products 1 hour prior to the experiment. Participants were informed that they would be asked about their consumption of all of these items prior to being allowed to participate in the experimental

session, and that failure to comply with these instructions would result in their not being allowed to participate. Participants were also informed that the saliva-hormone assay would detect if they had adhered to the pre-experiment requirements, and that if they were not honest about this information, the extra credit they received from participating in the study would be deducted from their course grade or they would be asked to return the financial compensation they were given. Participants were provided with a telephone number they could call to cancel their participation prior to the experiment, or to ask questions about the experiment or the pre-experiment regimen.

The primary investigator attempted to contact the 372 eligible people for this study via telephone or e-mail. A voicemail message with brief information about the purpose of the call and the study, as well as a contact phone number for the primary investigator, was left for anyone who did not answer their phone and who had a voicemail box. Eleven additional people who called the study hotline were screened via telephone. Several (i.e., at minimum of 5) attempts were made to contact participants who were identified as potentially meeting criteria for the NSI group, whereas only one attempt was made to contact potential control group participants because of the substantial disparity in the number of potential control group versus NSI group members. A total of 135 NSI-eligible participants were contacted (30.3% [n=41] signed up) and 237 controls were contacted (19.4% [n=46] signed up). Of those with whom contact was attempted via telephone, 64 (17.2%) refused to participate either in the study or the screening, including 18 (13.3%) participants from the NSI pool, and 46 (19.4%) participants from the Control pool.

#### *Inclusion and Exclusion Criteria*

Research suggests that both cortisol reactivity and baseline levels of cortisol secretion are altered by pregnancy and oral contraceptive ingestion (Kirschbaum & Hellhammer, 1989). Additionally, a strong body of empirical evidence has documented abnormal patterns of cortisol secretion and HPA activity in: (1) individuals with mood disorders (Birmaher et al., 1996; Board et al., 1956; Young, 2002; 2004) particularly in individuals with Bipolar Disorder (Young, 2004); (2) individuals with anxiety disorders, especially Panic Disorder (Bandelow, Sengos, Wedekind, Huthner, Broocks, Hajak, & Ruether, 1997; Stones, Groome, Perry Huckelbridge, & Evans, 1999) and PTSD, in which cortisol levels may either be higher (Baker et al., 1999; Bremner et al., 1997; Bremner et al., 2003) or lower (Mason, Giller, Kosten, Ostroff, & Podd, 1986; Yehuda, Southwick, Nussbaum, Giller, & Mason, 1991; Yehuda, Teicher, Levengood, Trestman, & Seiver, 1994) than normal depending upon the stressor; and (3) individuals reporting restrained eating (Anderson, Shapiro, Lundgren, Spataro, & Frye, 2002; McLean, Barr, & Prior, 2001).

Participants met criteria for the NSI group if they (1) endorsed at least two episodes of NSI in their lifetime and at least one episode of NSI during the 12-month period preceding the time of screening. Participants met criteria for the Control group if they (1) reported no history of any form of NSI. Due to the preceding evidence presented above and discussed earlier in this paper, participants for both groups were also required to meet the following criteria: (1) deny current pregnancy; (2) report negatively for current use of oral contraceptives; (3) deny a diagnosis of any mood disorder within the past month; (4) deny a history of any anxiety disorder within the past month; (5) deny a

history of a diagnosis of any eating disorder in their lifetime; and (6) deny a history of treatment for any eating disorder in their lifetime.

In total, 27 (7.3%) of those who were screened via telephone ( $n=372$ ) were disqualified because they reported current oral contraceptive use. This included 13 (9.6%) otherwise qualified self-injurers, and 14 (5.9%) otherwise qualified healthy controls. Additionally, two (<1%) of those who were screened via telephone (both from the NSI participant pool) were disqualified because they reported a history being diagnosed with a mood disorder within the past month. Another five (1.3%; also from the NSI pool) of those who were telephone screened were disqualified because they reported a lifetime history of an eating disorder diagnosis or eating disorder treatment. This resulted in a potential NSI participant pool of 115 persons.

Five to ten participants were scheduled to participate in each experimental session. The mean attrition rate (both formal cancellation and no-shows) was 2 participants per session, with an average of 1.5 cancellations and 0.5 no-shows per session. Of the 20 experiments that were scheduled, 8 (40%) were cancelled due to attrition below the required number of participants (<4 participants). On two occasions, a decision was made to use a trained confederate for the group conversational task when a no-show occurred on days when only four participants were scheduled and confirmed. Of those who provided a reason for canceling or no-showing, the most frequently provided reasons were “a family emergency” and schedule conflicts with places of employment. The number of participants actually participating in each experimental group session ranged from four to six ( $M=4.49$ ;  $SD=.69$ ). There was not a significant between-groups

(Rejected vs. Neutral) difference in the number participants participating in the conversational task groups during each experiment ( $t(52) = .57; p = .57$ ).

One participant from the NSI group was dismissed from the study because she had begun taking oral contraceptives between the time she was initially screened and the date of her participation. Nine participants acknowledged that they had consumed products likely to contain caffeine (e.g., sodas, chocolate) and two participants acknowledged alcohol consumption within the 24-hour restricted time period. Two participants acknowledged that they had eaten less than two hours prior to the study rather than abstaining for the two preceding hours as instructed.

All of the aforementioned participants, except the one who was dismissed, were allowed to participate in the experiment to maintain the minimum number of participants needed to run the experiment (i.e., 4 participants). To account for the potential effects of participants' non-adherence to pre-experimental restrictions on factors that can affect cortisol secretion patterns, follow-up repeated measures ANOVAs were performed using only those participants who reported adherence to pre-experimental restrictions.

### *Experimental Procedures*

The timeline for the segments of the experimental procedures used in this study are depicted graphically in Figure 1 at the end of this section below. Research strongly suggests that one of the most crucial factors to consider in experimental designs involving measurement of cortisol is the diurnal variation in secretion (Lovallo & Thomas, 2000), thus all experimental sessions were conducted between 2:00 p.m. and 5:00 p.m. When participants arrived for the experimental session, they were asked to sign an informed consent form describing the purpose, requirements, benefits, and risks



involved in the study (see Appendix I). Participants were also instructed to turn off their pagers and cell phones. After completing the consent form, participants also completed a health questionnaire inquiring about their activity and use and consumption of specific substances in the past 24 hours (Blackhart et al., 2007; see Appendix VI; “Short Health History Form”). Research assistants examined the health form to ensure that all pre-experimental guidelines were followed. Research assistants reminded the participant using specific instructions that they would lose their extra credit or financial compensation if it was determined that they were not honest in the information they provided on the Short Health History Form.

After completing the Short Health History Form, participants were asked to complete a group of questionnaires including the Symptom Checklist-90-Revised (SCL-90-R), Difficulties in Emotional Regulation Scale (DERS), Social Interaction Anxiety Scale, and the Borderline Personality Inventory (BPI) to collect data regarding the participant’s current level of emotional functioning and distress.

The next part of the session consisted of a relaxation segment, lasting approximately 20 minutes. This segment was incorporated into the study to allow for a reduction of any sympathetic nervous system activity that may have been induced by (i.e., habituation to) (1) the novelty of the environment; (2) the novelty of the situation; or (3) distress associated with completing psychometric questionnaires. During this segment, participants were asked to listen to specific selections of classical music chosen for their relaxing tone, tempo, and rhythm. These selections consisted of Antonio Vivaldi’s Concerto N<sup>o</sup> 1 in E Major Largo E Pianissimo Sempre, Allegro Non Molto, Allegro I, and Adagio Molto. Participants were also provided with two magazines, one related to

business in Minnesota and one about arts and crafts (e.g., knitting, crocheting), and instructed to do nothing but read these magazines and listen to the music during this period. No problems were encountered with participants falling asleep during this period. At the end of the relaxation segment, the first saliva sample (T1) was collected and the first Positive and Negative Affect Schedule (PANAS; T1P) was administered.

Next, all participants were brought into a main room of the laboratory and given instructions in a group format. Participants were asked to create a name tag for themselves to wear during this segment using a marker and a self-adhering label. Participants were instructed to engage in a group discussion (referred to from this point forward as the “conversational task”) regarding social activities on and off campus for approximately 15 minutes. Participants were given a printed list of conversation topics and related topical questions to use as a means of initiating or generating conversation. All conversation topics and questions are listed in Appendix VII.

After the 15 minutes had elapsed, the primary investigator or a research assistant interjected in the conversation, and informed participants that they would then be asked to separate into different areas or rooms of the laboratory and select two other people from the group that they would most like to work with during the next segment of the experiment (the “selection task”). The participants were informed that they would get to work with at least one other person, and possibly both that they selected. Once they were separated into their original laboratory rooms, participants were asked to provide a second saliva sample (T2), completed a second PANAS (T2P), and make their selections using a researcher-created form (“Experiment Selection Form”, see Appendix VIII). Approximately three minutes after the selection task was completed, each of the

participants was given one of two sets of feedback based on their condition assignment by the primary investigator or a research assistant. If participants were assigned to the “Neutral” condition, they were told the following by the primary investigator or a research assistant:

*“I need to talk to you about your participation in the next task of the experiment. We accidentally made a mistake and assigned you to the wrong group, so because of our mistake, you’ll have to complete the rest of the experiment on your own.”*

If participants were assigned to the “Rejected” condition, they were told the following by the primary investigator or a research assistant:

*“I need to talk to you about your participation in the next task of the experiment. When people made their selection, no one indicated that they wanted to work with you. This is kind of unusual and it’s never happened before, but consequently you’ll have to complete the rest of the experiment on your own.”*

Approximately three to five minutes after receiving feedback, participants were asked to provide the third saliva sample (T3) and to complete the third PANAS (T3P).

After providing the T3 saliva sample, participants were provided with a pen and a piece of blank white paper and asked to draw a house. Participants were informed that they would be given 10 minutes to complete this task. After the 10-minute period had elapsed, participants were asked to provide the fourth saliva sample (T4) and to complete the fourth PANAS (T4P); five minutes was allotted for this. Participants were subsequently given 10 minutes each to draw a tree, a person, and a car, with five minutes allotted after each drawing task for the collection of T5/T5P, T6/T6P, and T7/T7P, respectively. When participants had provided all saliva samples and completed all

PANAS questionnaires, they were asked to complete a questionnaire designed to function as a manipulation-check (see Appendix III; “My Experience”).

### *Debriefing*

Following collection of the final saliva sample and administration of manipulation check, participants were fully debriefed. The primary investigator, project manager, or a research assistant read the debriefing statement aloud to all participants (see Appendix II; “Debriefing Form”). To maintain experimental control, participants were not permitted to take a copy of the debriefing form with them. An explanation was provided in the debriefing statement for the use of deception, and a verbal apology was given to all participants for misleading them.

Following debriefing, participants were asked to complete a self-affirmations exercise (adapted from Teaster, 2004; see Appendix IV, “Positive Self-Statements”) in effort to alleviate any negative self-thoughts that may have been activated as a result of participation. Participants were then screened for current suicidal and self-injurious ideations using the first five items of the BSS, plus an additional item inquiring about non-suicidal self-injurious ideations structured in the same format of the BSS items. The primary investigator or the project manager carefully reviewed the BSS form to determine the participant’s risk level for suicide or NSI (see “Risk Determination and Management Procedure” below for a description of the risk determination procedure). If participants were determined to not be at risk for NSI or suicide, they were provided with an extra credit slip or financial compensation, and were dismissed from the experiment.

Figure 1. Timeline for Experiment Segments

**2:00 p.m.**

| Consent  
| Screening  
| Psychometric Assessment  
| 20 minute Relaxation  
| Post-Relaxation Saliva Sample  
| "Conversation Task"  
| T1 Sample (baseline)  
| "Selection Task"  
| Participant Feedback/Stressor  
| T2 Sample (3-5 minutes post stressor)  
| Drawing Task 1  
| T3 Sample (20 minutes post stressor)  
| Drawing Task 2  
| T4 Sample (35 minutes post stressor)  
| Drawing Task 3  
| T5 Sample (50 minutes post stressor)  
| Drawing Task 4  
| T6 Sample (65 minutes post stressor)  
| Experimental Manipulation Assessment  
| Debriefing  
| Risk Assessment

**5:00 p.m.**

### *Experimental Manipulation Check*

As noted in the Procedures section, the “My Experiences” questionnaire was administered to participants to determine the effect of the deception in the experimental manipulation. This questionnaire asked three key “Yes/No” questions, and allowed room for an explanation if the answer was “Yes”:

1. Did you believe, at any time, that the experiment dealt with anything other than what the experimenter had described to you?
2. Did this affect your behavior in any way?
3. Were you given any information about the experiment by anyone other than the researchers prior to coming here today?

Across the full sample, 51.9% (n=28) reported that they believed at some point that the experiment dealt with something other than what they had been told. Qualitatively, main themes included things such as “The ways people adapt to new situations and new people” and “How people react when they are stuck in a room for several hours.”

Qualitative review of participant’s free responses to these questions indicated that some of those who reported disbelief may have correctly guessed what the experiment was about, or what the deception had been. Furthermore, 22.2% (n=12) of participants reported that some element of the experiment influenced their behavior in some way. The statements written by participants are depicted in Table 1. Finally, 100% of participants reported that they had *not* been given any information about the experiment by anyone other than the principal investigator.

Participants assigned to both conditions were deceived in the experiment; however, it was most important that participants in the Rejected condition were convinced by the

deception. Examined by condition, 55.2% (n=16) of Rejected participants, and 48% (n=12) of Neutral participants reported believing that the experiment pertained to something other than what they had originally been told. The differences in these proportions were not statistically significant ( $\chi^2 [1, 53] = .28; p = .6$ ). Furthermore, only 6.4% (n=9) of those in the Rejected condition, and 5.6% (n=3) of those in the Neutral condition reported that this had affected their behavior in some way.

In sum, there were some participants in both conditions who believed that they were being deceived, but this percentage of participants was low in both groups and none correctly identified what the deception truly was. Although these qualitative data suggest the deception was not accurately detected, these data suggest that the experimental manipulation may not have been completely effective or believable. The feedback participants were given during the manipulation was a necessary, but not sufficient factor

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Table 1. Explanations of Experiment-Induced Behavior Changes Given by Participants

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*"I got really pissed off. I was tired of spitting in the stupid cup. I don't like to draw."*

*"Increased nervousness and suspicion about the experiment."*

*"Causes headaches probably due to the no caffeine."*

*"Just toward the end it got to be old and repetitive, just got a little warm and bored."*

*"Sure made me hungry..."*

*"More anxious to get out of here."*

*"I became frustrated and bored."*

*"The pre-experiment kept me from eating so as the experiment wore on, I grew hungrier and therefore less patient."*

*"I didn't feel left out or feel lowered self-esteem."*

*"It made me tired and a little lonely, not gonna lie."*

*"I really haven't thought too much about how to make friends and where to make friends. But after today's study, I may now start to think more about how friends are made."*

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for the requisite deception. Effective delivery of the feedback in a believable manner by research personnel was also necessary. However, the only control put in place for this factor was the training of research assistants in how to deliver the experimental manipulation. Thus, the results of this study must be considered in this context.

#### Risk Determination and Management Procedure

The risk determination and management procedure (RDMP) was used to establish each participant's risk level based on their responses to the modified BSS, and provide further assessment and management based on that assessment as needed. First, participant's responses to the BSS screening items were reviewed. If a participant's responses were "1" or "2" on items 3, 4, or 6 of the BSS, or if a participant endorsed a "1" on three out of five of the suicide screening items, then further assessment by the project manager or primary investigator was initiated. Finally, an a priori decision was made to initiate further assessment if a participant exhibited any overt behavioral indications of acute distress (i.e., distress that may be reasonably assumed to impair their functioning). These indications included tearful affect, clearly unsteady gross or fine psychomotor behavior, or distress-oriented verbal expression. When participants met any of these criteria (n=8), the primary investigator took the following steps to ensure the participant's safety:

1. The participant was assessed for acute suicidality, acute self-injuriousness, and risk factors for both of these by the primary investigator, or project manager in consultation with the primary investigator. This assessment was aimed at determining their risk level (see Appendix V; "Risk Screening"), current coping



resources (e.g., social support, currently seeing a trusted psychotherapist) and potential for carrying out any active ideations.

2. *Low Risk Participants.* If the participant is determined to be at a *low* risk for suicide or non-suicidal self-injury, the primary investigator or project manager provided the participant with a referral resource handout, and a recommendation for therapy. Six participants were determined to be at this risk level.
3. *Moderate Risk Participants.* If the participant is determined to be at a *moderate* risk for suicide or non-suicidal self-injury, the primary investigator or project manager gave the participant the option of contacting a support person (e.g., friend, family member) and requesting to get together immediately (preferably the friend will meet the student at the research lab or at Corwin/Larimore). If a support person was not immediately available, the student would have been asked to collaboratively construct a safety plan for remaining safe from self-injury, be provided with mental health resources, and encouraged to seek treatment. All participants determined to be at moderate risk were able to create a safety plan, and contact a support person while in the lab. Two participants were determined to be at this risk level.
4. *High Risk Participants.* Although no participants were determined to be at high risk, if a participant had been determined to be at a *high* risk for suicide or non-suicidal self-injury, the primary investigator or project manager would have determined if the participant had a supportive social contact in their life. If so, the primary investigator or project manager would have requested that the participant contact that person. The lab supervisor, Dr. Jennifer Muehlenkamp, would also

have been contacted while the participant contacted her/his support person and would have come to the lab to conduct a further assessment and ensure the student is connected with support services of some type prior to leaving the lab. The student would not have been permitted to leave the lab alone. Should an immediate support person not be available, the UND crisis team would have been contacted. No participants were determined to be at this risk level.

5. *Imminent Risk Participants.* Although no participants were determined to be at imminent risk, if a participant had been determined to be at a *imminent* risk for suicide or non-suicidal self-injury, the primary investigator or project manager would have requested that the participant remain in the lab, and asked that a research assistant contact Dr. Muehlenkamp. During this time the primary investigator or project manager in consultation with the primary investigator would have contact the UND crisis team and assist the student in speaking with the team. The participant would not have been permitted to leave the lab without speaking with a member of the UND crisis team. No participants were determined to be at this risk level

## Measures

### *Psychometric Instruments*

#### *Deliberate Self-Harm Inventory*

The Deliberate Self-Harm Inventory (DSHI; Gratz, 2001) is a 17-item self-report inventory, which is behaviorally based. It was selected for use in the preset study because of the specificity of its item content and because of the relatively stable psychometric properties reported by its developer (see Gratz, 2001). Each item contains

an initial yes/no question regarding a specific self-damaging behavior, followed by five follow-up questions. This instrument includes multiple response formats, including primary items with dichotomous answer choices (e.g., “Have you ever intentionally (i.e., on purpose) cut your wrist, arms, or other area(s) of your body (without intending to kill yourself)?”), and follow-up items with a free response answer format (e.g., “How old were you when you first did this?”; “How many times have you done this?”). Gratz (2001) reports a high degree of internal consistency ( $\alpha = 0.83$ ).

The version of the DSHI used in this study for both screening and experimental procedures was slightly modified. In this modified version, participants were given categorical choices for indicating the last time that they engaged in any endorsed NSI behavior (i.e. 1=within the past 2 weeks; 2= 3-4 weeks ago; 3=over 1 month but less than 2 months ago; 4=2 months to less than 3 months ago; 5=3 months to less than 4 months ago; 6=4 months to less than 5 months ago; 7=5 months to less than 6 months ago; 7=6 months to less than 9 months ago; 8=9 to 12 months ago; 9=More than 12 months ago), rather than a space for providing a subjectively worded answer. The goal of this modification was to minimize the number of potentially eligible participants who would be lost based solely on either an unintelligible response or a failure to respond to the item altogether. While some screening participants may still have chosen not to respond to this item, the proposed modification was aimed at minimizing the effort required to provide a response (i.e., by only having to circle an answer instead of write one out).

The validation study sample for the original version of the instrument consisted of 159 undergraduate students in Psychology courses at the University of Massachusetts, Boston (Gratz, 2001). Item total correlations for each item in the preliminary validation

study were as follows: Cutting,  $r_b = 0.63$ ; Burning with a cigarette,  $r_b = 0.34$ ; Burning with a lighter or match,  $r_b = 0.49$ ; Carving words into skin,  $r_b = 0.47$ ; Carving pictures into skin,  $r_b = 0.45$ ; severe scratching,  $r_b = 0.51$ ; Biting,  $r_b = 0.54$ ; Rubbing sandpaper on skin,  $r_b = 0.14$ ; Sticking pins, needles, staples into skin,  $r_b = 0.65$ ; rubbing glass into skin,  $r_b = 0.35$ ; Breaking bones,  $r_b = 0.12$ ; Banging head,  $r_b = 0.57$ ; Punching self,  $r_b = 0.44$ ; Interference with wound healing,  $r_b = 0.49$ ; Other forms of NSI,  $r_b = 0.36$ ; Dripping acid on skin,  $r_b < 0.01$ ; Using bleach or oven cleaner to scrub skin,  $r_b < 0.01$ .

Product-moment test-retest correlations were based on a sample of 93 participants who took part in the second administration of the DSHI. Over an intervening period of 2-4 weeks, the DSHI demonstrated a test-retest reliability of .68 ( $p < 0.001$ ), with a concomitant high correlation ( $r = 0.92$ ;  $p < 0.001$ ) between the number of NSI behaviors that were endorsed by participants on the first and second administrations.

With regard to convergent and discriminant validity, the DSHI dichotomous items demonstrated a correlation of .40 ( $p < 0.01$ ) with the Borderline Personality Organization Scale (BPO; Oldham, et al., 1985); the frequency assessment items of the DSHI correlated at 0.48 ( $p < 0.001$ ) with the BPO. The DSHI correlated moderately ( $r = 0.43$ ;  $p < .001$ ) with the NSI item of the DIB-R (Zanarini et al., 1989), and moderately ( $r = 0.35$ ;  $p < 0.001$ ) with the NSI item of the Suicidal Behaviors Questionnaire (SBQ; Linehan, 1981). The DSHI also demonstrated a correlation of 0.49 with a history of mental health service utilization; a history of suicide attempts was correlated 0.20 and 0.21 with the dichotomous and frequency items of the DSHI respectively. Overall, the DSHI has yielded adequately to excellently stable psychometric properties. Thus, its use in the present study was determined to be advantageous. For the present study, reliability

analyses revealed an internal consistency reliability of  $\alpha=.75$ , and an inter-item correlations ranging from  $r = -.25-.69$ .

### *Beck Scale for Suicidal Ideation*

The Beck Scale for Suicidal Ideation (BSS; Beck, Steer, & Ranieri, 1988) is a commonly used 19-item self-report instrument, with 5 preceding screening questions, designed to assess presence and severity of suicidal ideation. The screening items are used to facilitate rapid completion of the scale for nonsuicidal individuals. Items on the BSS are rated on a three-point scale (0, 1, or 2); hence, total scores may range from 0 to 48. Beck and colleagues (1985, 1988) report no specific cut-off scores for this instrument. However, recent research (Cochrane-Brink, Lofchy, & Sakinofsky, 2000) has identified that a cutoff score of 24 may be useful in helping to determine when hospitalization of a suicidal individual is medically necessary. Beck & Steer (1993) report that the BSS addresses five primary factors of suicidality: Intensity of Suicidal Ideation, Active Suicidal Desire, Planning, Passive Suicidal Desire, Concealment. This instrument has demonstrated adequate to good internal consistency, with values for all scales ranging from  $\alpha=0.7$  to 0.84 (Beck & Steer, 1993; Holden & DeLisle, 2005). Recent data also suggest that this instrument has high sensitivity (100%) and specificity (90%) as a suicidality screening tool (Cochrane-Brink et al., 2000). In the present study, items 1-5, 20, and 21 were administered. The first five items (i.e., the critical items) of the BSS were employed as a method of assessing imminent suicide and self-injury risk. An additional item inquiring about non-suicidal self-injurious ideations were added to these items. Items 20 and 21 were used to collect data regarding history of suicidal behavior.

### *Symptom Checklist-90-Revised*

The Symptom Checklist-90-Revised (SCL-90-R; Derogatis, 1994) is a self-report instrument originally developed by Derogatis (1977) for clinical and research settings. As such, it is widely used in both clinical research and clinical practice. This instrument contains 90 items that pertain to different forms and features of psychological functioning. Participants rate the degree to which they have experienced each item during the past seven days, including the current day, on a five-point, Likert-type scale (“0” = Not at all; “4” = Extremely). The SCL-90-R is comprised of nine subscales that reflect nine symptom dimensions (Somatization, Obsessive-Compulsion, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, Psychoticism), and three global scales (Global Severity Index, Problem Severity Distress Index, Problem Symptom Total; Derogatis, 1994).

The SCL-90-R has demonstrated sound psychometric properties. Derogatis (1994) reports test-retest reliability coefficients ranging from .80-.90 based on his validation study. However, an earlier study by Horowitz and colleagues (1988) found test-retest reliability coefficients ranging from .68-.83 over a ten week period. Additionally, internal consistency coefficients reported in the literature have ranged from  $\alpha = .77-.90$  (Derogatis, Rickels, and Rock, 1976; Horowitz, 1988). Furthermore, a wealth of literature has established the convergent, discriminant, concurrent, and predictive validity of the SCL-90-R (Asberg, Kragh-Sorensen, Mindham, & Tuck, 1973; Boleolucky & Horvath, 1974; Derogatis, 1994; Derogatis et al., 1976; Koeter, 1992; Peveler & Fairburn, 1990; Wiznitzer, 1992). Internal consistency for individual SCL-90-R subscales in the present study ranged from moderate to excellent; Depression  $\alpha = .87$ ; Anxiety  $\alpha = .79$ ; Isolation  $\alpha = .92$ ; Somatization  $\alpha = .76$ ; Phobia  $\alpha = .90$ ; Obsessive Compulsion  $\alpha = .84$ ;

Hostility  $\alpha = .73$ ; Psychoticism  $\alpha = .65$ ; Paranoia  $\alpha = .59$ . The SCL-90-R was used in the present study as a method of determining participants' global level of level of psychopathology and distress.

#### *Social Interaction Anxiety Scale*

The Social Interaction Anxiety Scale (SIAS; Mattick & Clarke, 1998) is self-report measure of social anxiety that specifically addresses anxiety that is related to interaction in social situations. This instrument contains 20 items pertaining to various aspects of social interactions in groups. Each item inquires the degree to which a feeling, behavior, or cognition (e.g., "I have difficulty making eye-contact with others.") characterizes the respondent, and is rated on a five-point Likert-type scale ("0" = Not at all; "4" = Extremely). The total score is calculated by summing the value of each item. Higher scores reflect higher levels of social anxiety.

The SIAS has sound psychometric properties. Mattick, Peters, & Clarke (1989) and Mattick & Clarke (1998) reported test retest reliability of .92 for a 4-week interval, and 0.92 for a 12-week interval in a combined sample of undergraduates, community participants, and untreated socially phobic, agoraphobic, and simple phobic individuals. In the same vein, Heimberg, Mueller, Holt, Hope, and Liebowitz (1992) reported good levels of internal consistency ( $\alpha = .88-.83$ ) in Australian samples. Similarly, Osman, Gutierrez, Barrios, Kopper, & Chiros (1998) reported an internal consistency of  $\alpha = .90$  for their sample of 200 undergraduate university students. Additionally, internal consistency for the SIAS is excellent ( $\alpha = .94$ , total sample;  $\alpha = .93$  for individuals with social phobia). The strength of internal consistency in the present study was comparably high ( $\alpha = .92$ ). This scale has also demonstrated high convergent validity ( $r = .73$ ; Peters, 2000) with the

Social Phobia Scale (Mattick & Clarke, 1998), another measure of social anxiety. Furthermore, Brown, Turovsky, Heimberg, Juster, Brown, & Barlow (1997) reported a high level of discriminant validity for the SIAS. In their sample of 165 anxiety disordered patients, Brown et al. (1997) reported that the SIAS correctly classified 86% of socially phobic patients (n=50), thus suggesting this instrument is a reliable measure of social anxiety. In the present study, the SIAS was employed as a method of assessing the effects of social anxiety on cortisol responses given that the stressor will be based on social interactions.

#### *Difficulties in Emotion Regulation Scale*

The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36-item self-report instrument that assesses multiple facets of emotional regulation. Each item requires that the participant indicate what percent of the time they experience each item (e.g., “When I’m upset, I have difficulty controlling my behaviors.”) using a 1-5 Likert-type scale. Response choices range from 1 (“almost never;” 0-10%) to 5 (“almost always;” 91-100%). The DERS is comprised of six scales: Nonacceptance of Emotional Responses, Difficulties Engaging in Goal-Directed Behavior, Impulse Control Difficulties, Lack of Emotional Awareness, Limited Access to Emotion Regulation Strategies, and Lack of Emotional Clarity. The DERS has demonstrated good psychometric properties, with 4-8 week test-retest reliability of .88 for the DERS total score, and coefficients ranging from .57 to .89 for individual subscales. Total scale internal consistency for the DERS is excellent ( $\alpha=.93$ ), as is individual scale reliability ( $\alpha > .80$ ). In the present study, the DERS was included as a method of establishing convergence between biological and self-report measures of emotion regulation, and an



internal consistency alpha of  $\alpha=.95$  was obtained for the total scale with the current sample.

### *Borderline Personality Inventory*

The Borderline Personality Inventory (BPI; Leichsenring, 1999) is a 53-item self-report instrument that assesses features of Borderline Personality Disorder based on Kernberg's (1975) Borderline Personality Organization model. This instrument uses a true/false response format, and is comprised by five subscales: Identity Diffusion, Primitive Defense, Fear of Closeness, Impaired Reality Testing, and Cut-20. The last subscale consists of those items that were found to best discriminate between borderline patients and "neurotic" and schizophrenic patients (Leichsenring, 1999). The BPI yields excellent overall internal consistency ( $\alpha=.91$ ) and one-week test-retest ( $r=.87$ ) reliability. Additionally, all five subscales yield good to excellent internal consistency ( $\alpha=.68-.85$ ) and one-week test-retest ( $\alpha=.73-.89$ ) reliability. Leichsenring (1999) reports levels of diagnostic sensitivity ranging from .85-.89, and diagnostic specificity ranging from .78-.89, thus suggesting the utility of this instrument in classification. This instrument is generally recommended for the purpose of screening participants for BPD or prominent borderline features, and was used for that purpose in the present study. Internal consistency in this sample was  $\alpha=.81$ .

For the present study, the total scale score and the "Cut-20" subscale score were used as indicators of psychopathology. The Cut-20 is the subset of 20 items that best discriminates between BPD patients and "neurotic" and schizophrenic patients. Cut-20 is an atheoretical, empirically derived score comparable to the total score of Diagnostic interview for Borderlines (Gunderson et al., 1989). Liechsenring's (1999) validation data

for the BPI suggests that a score of  $\geq 10$  on Cut-20 reliably discriminates a BPD patient from a clinical non-BPD individual.

### *Positive and Negative Affect Schedule*

The Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) is a 20-item self-report instrument that assesses an individual's current level of both positive and negative affect by asking the individual to rate, using a five-point Likert-type scale (1= Not at All; 5= Extremely), the degree to which each item on the scale describes their current affective state.

The instrument has two subscales: the Positive Affect (PA) subscale and Negative Affect (NA) subscale. The PA subscale assesses the extent to which a participant is currently experiencing positive emotion, and is comprised of ten adjectives describing positive emotional experiences (e.g., enthusiastic, pleasant). Higher scores on the PA scale are theorized to reflect higher levels of positive affect or emotion. Similarly, the NA subscale evaluates the degree to which a respondent is experiencing negative emotion. It is comprised of ten adjectives describing negative emotion (e.g., jittery, afraid). Higher scores on the NA subscale are theorized to reflect higher levels of negative affect or emotion. The PANAS has demonstrated excellent internal consistency, with  $\alpha = .89$  for PA, and  $\alpha = .85$  for NA (Crawford & Henry, 2004).

### *Biological Measures*

#### *Salivary Cortisol*

Salivary cortisol is a valid and consistent measure of serum levels of free cortisol (Kirschbaum & Hellhammer, 1994). Saliva samples were collected using a two milliliter-capacity plastic passive-drool Salivette, obtained from Salimetrics, LLC.

Plastic straws approximately 4 to 5 centimeters in length were used to facilitate movement of saliva into the vials for sampling. Participants were instructed to either chew on a piece of straw, or to make a chewing motion with their mouth, as needed if saliva flow was not readily available; both of these procedures are recommended by the testing company. All saliva samples were stored in a freezer at a temperature of 20° C.

Cortisol levels in saliva samples were determined through a highly sensitive enzyme radioimmunoassay (Salimetrics, PA) conducted by Salimetrics, LLC. This assay uses 25 ul of saliva per determination, has a lower limit of sensitivity of 0.003 ug/dl, standard curve range from 0.012 to 3.0 ug/dl, and average intra-and inter-assay coefficients of variation 3.5 % and 5.1 % respectively. Method accuracy, determined by spike and recovery, and linearity, determined by serial dilution are 100.8 % and 91.7%. Values from matched serum and saliva samples show the expected strong linear relationship,  $r(63) = .89, p < 0.0001$ . As a measure of quality control in the present study, double assays were extracted from 83.3% of the total number samples. Single assays were performed on baseline measurement samples, while 100% of post-baseline samples were double assayed. For those samples that were double assayed, the mean cortisol value ( $\mu\text{g/dL}$ ) was used for statistical analyses.

## CHAPTER III

### RESULTS

#### Data Analyses

An alpha-level of .01 was set for all independent samples *t*-tests and analyses of variance. An alpha-level of .05 was adopted for all correlational analyses. As suggested by Stevens (2002), for analyses in which sphericity could not be assumed, Greenhouse-Geisser corrected values were employed.

#### *Nonsuicidal Self-Injury Characteristics*

Participants in the NSI group reported engaging in a variety of forms of NSI. The total number of types of NSI that participants endorsed ranged from 1 (30.8%; *n*=8) to 11 (3.8%; *n*=1) types ( $M=3.35$  types;  $SD=2.76$ ). This mean, although slightly higher than that found in recent samples of adolescents by Lloyd-Richardson, Perrine, Dierker, and Kelley (2007), is consistent with other prior research indicating a multiplicity of methodology of NSI (e.g., Favazza, 1992; Favazza & Conterio, 1988; Osuch, Noll, & Putnam, 1999). Over one-quarter (26.0%; *n*=7) of the NSI participants endorsed two types of NSI, and almost half (42.3%; *n*=11) endorsed three or more forms of these behaviors. The most common types of NSI behaviors were cutting (66.7%; *n* = 16), severe scratching (43.5%; *n*=10), subcutaneous insertion of sharp objects (43.5%; *n*=10), punching self or objects (40.9%; *n*=9), and burning (34.8%; *n*=8).

NSI group participants reported ages of onset for any NSI behaviors ranging from 11 to 19 years, with a mean age of onset of 14.81 years ( $SD= 2.25$  years). Within

individual types of NSI, age of onset ranged from 11-47 ( $M=15.58$  years;  $SD= 4.38$ ). Participants who reported cutting behavior were most likely to report a history of NSI requiring medical attention (13.6%), although this did not differ significantly from the proportions of NSI participants reporting other forms of NSI that required medical treatment. Frequency of NSI across all types ranged from 2 to 54 or more episodes ( $M=14.36$  episodes;  $SD=13.4$ ), with a multimodal frequency of 4 and 6 episodes.

### *Psychological Variables*

Independent-samples t-tests were conducted to examine potential between group differences on the SCL-90-R subscales and the BPI total score and Cut-20 subscale score (see measures). Subscale scores derived for the SCL-90-R are standardized T-scores based on norms from population subsets (e.g., clinical outpatients, clinical inpatients, nonpatients). Given that some participants may have currently been in outpatient treatment while others may not have been, thus requiring different norms for such groups, raw scores on the SCL-90-R were used in the aforementioned t-tests. Analyses of SCL-90-R raw scores revealed no significant between-group differences on any scales; although a trend toward significance (i.e.,  $.01 < p < .05$ ) was observed for scores on the Anxiety subscale ( $t(52)= 2.19$ ;  $p=.035$ ), with NSI participants scoring higher ( $M=3.42$ ;  $SD=4.29$ ) than control participants ( $M=1.43$ ;  $SD=1.81$ ).

Independent-samples t-tests of BPI subscale scores revealed a significant difference for the 20-item cutoff subscale (Cut-20;  $t(52)=4.05$ ;  $p<.001$ ), with NSI participants ( $M= 4.27$ ;  $SD=3.01$ ) outscoring Control participants ( $M= 1.46$ ;  $SD=2.01$ ). Tests of between-groups differences for BPI Total scores were also significant ( $t(36)=3.34$ ;  $p=.002$ ). NSI participants ( $M= 12.71$ ;  $SD=5.10$ ) scored significantly higher

on BPI Total scores than Control participants ( $M= 7.35$ ;  $SD=4.69$ ). The means were well below the Cut-20 cutoff for both groups, even though they were significantly different. Nonetheless, this finding is consistent with an emotion dysregulation model of NSI considering the wealth of literature establishing BPD as a pervasive disorder of the emotion regulation system and the high prevalence of NSI in individuals diagnosed with BPD (e.g., Linehan, 1993; Skodol et al., 2002a; 2002c), although both groups scored well below clinical cut-off score of 10.

Independent-samples t-tests were also conducted to determine if SIAS scores differed significantly between groups. These analyses revealed no significant differences for the SIAS between NSI and control groups, with mean scores of 18.62 and 17.43 for the NSI and control groups, respectively ( $t(52)= .37$ ;  $p =.71$ ). Furthermore, no significant between-groups difference was found on SIAS scores for participants assigned to different conditions ( $t(52)= -.23$ ;  $p =.82$ ).

Descriptive analyses of the modified version of the BSS revealed that 19.2% ( $n=5$ ) of the NSI group endorsed a current “weak desire” to self-injure themselves, with the remaining 80.8% reporting no desire to self-injure. Broken down by cell, 60% ( $n=3$ ) of those endorsing any current urge to engage in NSI on the BSS at the end of the experiment had been assigned to the Rejected condition, and 40% had been assigned to the Neutral condition. This difference in proportions was not statistically significant ( $\chi^2 [1, 25] = .094$ ;  $p=.76$ ).

#### Notes on Cortisol Analyses

##### *Baseline Measurement*

Fehm-Wolsdorf, Groth, Kaiser, and Hahlweg (1999), note that baseline cortisol samples collected at the beginning of an experiment reflect stressful events occurring prior to the experiment because of the cortisol response curve, which maximizes at 20-30 minutes following a stressful event. It was possible that cortisol levels in samples collected earlier could have been affected by pre-experiment stress; or that the levels in these samples were affected by stress induced before the manipulation (e.g., by answering questions about psychological distress). Thus, for all cortisol analyses in the present study, the post-conversational task sample (the second sample collected overall) was used as the baseline measurement (rather than the post-relaxation baseline) for these analyses for two reasons. First, cortisol response, as conceptualized in the present study, was most accurately characterized by the difference in cortisol volume between the post-stressor (i.e., rejection/neutral feedback) measurement points and post-conversation task measurement point immediately preceding the stressor (i.e. the post-conversational task sample). Second, use of this particular measurement point was aimed at controlling for any potential inflationary effects of social interaction on cortisol levels, and was deemed a better choice than employment of statistical controls, such as including this measurement point in a multiple analysis of covariance. The use of a second, pre-stress sample as a baseline measurement has been employed successfully in previous psychophysiological research involving analyses of cortisol and prolactin responses to 5HT<sub>1A</sub> and 5HT<sub>2A</sub> receptor agonists (Leone et al., 1998); heart rate, skin conductance, and skin temperature in response to alcohol (Newlin & Thomson, 1991); prolactin response following orgasm (Brody & Kruger, 2006); heart rate and blood pressure response to medication (van Stegeren, Everaerd, & Gooren, 2002); and oxygen

consumption and exercise performance during stress tests (Harris, LeMaitre, Mackenzie, Fox, & Denvir, 2003).

### *Outliers*

It is well established in the statistical literature that extreme scores may inflate (or deflate) measures of central tendency, thus rendering inferential conclusions based on these statistics less accurate (Winer, 1971). Box plot analyses conducted for cortisol data in the current study revealed multiple outliers in each of the four cells for most time intervals (see Table 2 below for the exact number of outliers by cell, time interval, and type of outlier [high or low]). Experts have proposed multiple methods for handling such data anomalies, including (1) leaving outlying data points in the data set; (2) removing outlying data points from the data set; and (3) replacing outliers with more representative values derived from the data set. The latter of these (outlier replacement, rather than removal or inaction) was selected for cortisol analyses for three reasons.

First, the present study was a theory-driven pilot study, with no directly comparable data from which to conclude that any outlying data points were anomalies versus representative of the full range of cortisol secretion patterns in self-injurers. Thus omitting the outliers from this data set had the potential to unnecessarily restrict the range, possibly leading to a statistical mischaracterization of the actual data. Second, the cell sizes for the present study were relatively small to begin with, and the potential loss of the number of outliers that would have been omitted was likely to unnecessarily compromise power, given that there were options available for transforming the data, which would preserve cell size and power. Finally, the outlying data points extended far enough beyond the upper and lower bounds of this data set that the probability that this



minority of data points had an effect on the measures of central tendency for the majority of the data was at least moderate. Thus, the principal investigator decided to employ a Winsorization method of data transformation (Winer, 1971) for cortisol analyses, which allowed for retention of the high and low outlying data points via replacing these data with the values of the upper and lower bounds found within the larger sample.

#### Hypothesis 1

Hypothesis 1 was tested using a 2 (GROUP) x 2 (CONDITION) x 5 (TIME) repeated measures ANOVA. Groups in this general linear model included self-injuring and control; conditions included “neutral” and “rejected;” and time was based on six measurement points: pre-stressor baseline (T1), 3-5 minutes post-stressor (T2), 20 minutes post-stressor (T3), 35 minutes post-stressor (T4), 50 minutes post-stressor (T5), and 65 minutes post-stressor (T6). The factor “TIME” was measured in units of mean cortisol level differences from baseline ( $CD_{base}$ ) measurement, with each value equating to the difference between the cortisol level at the respective measurement point and the baseline cortisol level. For example, TIME 1 equals the difference in cortisol level between T1 and T2; and TIME 2 equals the difference in cortisol level between T1 and T3.

An initial series of repeated measures ANOVAs (Series 1) was performed on the full sample. Table 4 (below) depicts mean cortisol levels and standard deviations for NSI and control group participants separated by condition at each measurement point. Figure 2 (also below) depicts this same data in a graphical format, documenting mean changes across time for each group in each condition. A test of requisite statistical assumptions indicated that sphericity had been violated ( $\chi^2 = 94.49$ ;  $p < .001$ ;  $\epsilon_{Greenhouse-Geisser} = .494$ ).

Table 2. Number of Outliers Replaced by Cell, Time Interval, and Outlier Type

	T2-T1 Total (High/Low)	T3-T1 Total (High/Low)	T4-T1 Total (High/Low)	T5-T1 Total (High/Low)	T6-T1 Total (High/Low)
NSI Rejected	3 (3/0)	2 (2/0)	1 (1/0)	1 (1/0)	0 (0/0)
Control Rejected	3 (1/2)	2 (2/0)	2 (2/0)	2 (1/1)	1 (0/1)
NSI Neutral	2 (1/1)	1 (1/0)	2 (2/0)	0 (0/0)	0 (0/0)
Control Neutral	1 (1/0)	2 (2/0)	1 (1/0)	1 (1/0)	1 (1/0)

Therefore, Greenhouse-Geisser corrected  $p$ -values were used for significance tests of within-subjects effects for  $CD_{\text{base}}$  scores.

Results of Series 1 ANOVA revealed no significant between-subjects main effect for Group ( $F(1, 50)=.578$ ;  $p=.45$ ;  $\eta^2=.011$ ). However, as depicted in Table 3, a significant within-subjects main effect was found for Time ( $F(2.18, 109.13)=45.179$ ;  $p<.001$ ;  $\eta^2 = .62$ ). Across both groups and conditions,  $CD_{\text{base}}$  scores for T2-T1 were significantly higher than  $CD_{\text{base}}$  T3-T1 ( $p=.001$ ),  $CD_{\text{base}}$  T4-T1 ( $p<.001$ ),  $CD_{\text{base}}$  T5-T1 ( $p<.001$ ), and  $CD_{\text{base}}$  T6-T1 ( $p<.001$ ).  $CD_{\text{base}}$  scores for T3-T1 were significantly higher than  $CD_{\text{base}}$  T4-T1 ( $p<.001$ ),  $CD_{\text{base}}$  T5-T1 ( $p<.001$ ), and  $CD_{\text{base}}$  T6-T1 ( $p<.001$ ). Additionally,  $CD_{\text{base}}$  scores for T4-T1 were significantly higher than  $CD_{\text{base}}$  T5-T1 ( $p<.005$ ) and  $CD_{\text{base}}$  T6-T1 ( $p<.001$ ). However,  $CD_{\text{base}}$  scores for T5-T1 were not significantly different from  $CD_{\text{base}}$  T6-T1 ( $p=.173$ ).

In sum, the main effect found for Time suggested that regardless of group or condition assignment, participants' change in cortisol levels ( $CD_{\text{base}}$ ) was, on average, both positive and significantly higher at the first post-stressor measurement point than at any of the subsequent measurements points. Results of Series 1 ANOVA also suggested that the subsequent  $CD_{\text{base}}$  scores progressively decreased following the stressor, with each  $CD_{\text{base}}$  being significantly lower than the previous score, with the exception of the T5-T1/T6-T1 comparison, which indicated a nonsignificant difference in these  $CD_{\text{base}}$  scores. Interestingly, all  $CD_{\text{base}}$  scores subsequent to the first post-stressor measurement point were negative, indicating that the mean cortisol levels at these measurement points were below the baseline measurement point. This finding is curious and warrants further exploration.

Table 3. Series 1 Windsorized Mean Cortisol Difference ( $CD_{base}$ ) Scores for NSI and Control Groups by Time Interval Across Groups and Condition

Time Interval	Mean $CD_{base}$ Scores ( $\mu\text{g/dL}$ )	SE
T2-T1	.01332 <sup>bcd</sup>	.0041
T3-T1	-.00117 <sup>cde</sup>	.0036
T4-T1	-.01721 <sup>de</sup>	.0038
T5-T1	-.02613	.0042
T6-T1	-.03228	.0043

Notes: All differences are significant at  $p \leq .001$ ; a=difference is significantly higher than T2-T1; b= difference is significantly higher than T3-T1; c= difference is significantly higher than T4-T1; d= difference is significantly higher than T5-T1; e= difference is significantly higher than T6-T1

Results of Series 1 repeated measures ANOVA also revealed a significant between-subjects main effect for Condition ( $F(1, 50)=10.54; p=.002; \eta^2=.174$ ).

Across all times and groups, the  $CD_{base}$  score for those participants assigned to the Neutral condition ( $M=-.00254 \mu\text{g/dL}; SE=.00459 \mu\text{g/dL}$ ) was significantly greater than that of Rejection condition participants ( $M=-.02285 \mu\text{g/dL}; SE=.00426 \mu\text{g/dL}$ ). Because both of these means are negative, it may be inferred that the robust effect of each condition resulted in a decrease in cortisol across time and group; however, the larger negative mean of the Rejection condition suggests that this group's cortisol levels decreased further on average than those participants in the Neutral condition. Although no formal a priori hypothesis was made regarding this effect, this finding was

counterintuitive, in that it was expected that cortisol levels would increase in those participants who were “rejected.”

A significant 2 (Condition) x 5 (Time) interaction effect was also found ( $F(1.98, 109.13)=7.75; p=.001; \eta^2=.13$ ), such that, for participants in the Rejected condition, T2-T1  $CD_{base}$  scores were significantly greater than  $CD_{base}$  T3-T1,  $CD_{base}$  T4-T1,  $CD_{base}$  T5-T1, and  $CD_{base}$  T6-T1. Additionally, participants assigned to the Neutral condition evidenced significantly greater ( $F(1.98, 109.13)=7.75; p=.001; \eta^2=.13$ )  $CD_{base}$  scores than participants in the Rejected condition at T6-T1. The a priori hypotheses of the present study did not directly speak to this comparison; however, this finding is nonetheless counterintuitive. The cortisol difference scores for both Neutral and Rejection participants were also negative at the T6-T1 juncture, indicating that both groups tended to experience decreases in cortisol levels. Consequently, this latter finding suggests that participants in the Neutral condition tended to have smaller decreases in cortisol than participants in the Rejection condition.

Furthermore, a significant 2 (Group) x 5 (Time) interaction effect was also found ( $F(1.98, 109.13)=5.19; p=.007; \eta^2=.094$ ), such that, for participants in the NSI group, T2-T1  $CD_{base}$  scores were significantly greater than  $CD_{base}$  T5-T1,  $CD_{base}$  scores. For Control participants, T2-T1  $CD_{base}$  scores were significantly greater than T4-T1, T5-T1, and T6-T1  $CD_{base}$  scores.

Of most relevance to Hypothesis 1, Series 1 repeated measures ANOVA revealed no significant 2 (Group) x 2 (Condition) x 5 (Time) interaction effect ( $F(1.98, 109.13)=.86; p=.43; \eta^2=.017$ ). Thus, Hypothesis 1 was not supported by the results of these analyses.

It was possible that extraneous variables such as caffeine or alcohol consumption contributed to a lack of significant findings in these analyses. Therefore, a follow-up repeated measures ANOVA (Series 2) on a reduced data set in which the 11 participants who reported having been non-adherent to pre-experimental dietary restrictions (no participants reported being non-adherent to pre-experimental behavioral restrictions) were removed. Similar to Series 1 ANOVAs, a violation of the sphericity assumption was indicated ( $\chi^2 = 90.04$ ;  $p < .001$ ;  $\epsilon_{\text{Greenhouse-Geisser}} = .432$ ). Thus, Greenhouse-Geisser corrected  $p$ -values were also used for Series 2 significance tests of within-subjects effects for  $CD_{\text{base}}$  scores.

Consistent with the initial ANOVA, the results of Series 2 ANOVAs revealed no significant between-subjects main effect for Group ( $F(1, 39) = .244$ ;  $p = .62$ ;  $\eta^2 = .006$ ). Also in accordance with initial analyses, Series 2 ANOVA revealed a robust, significant within-subjects main effect for Time ( $F(1.94, 75.68) = 39.38$ ;  $p < .001$ ;  $\eta^2 = .502$ ), with a pattern of  $CD_{\text{base}}$  scores that was identical to that of Series 1 in both proportion and degree of significance (all  $p_s \leq .005$ ). As found in Series 1 ANOVA, Series 2 analyses revealed a significant between-subjects main effect for Condition ( $F(1, 39) = 8.04$ ;  $p = .007$ ;  $\eta^2 = .171$ ). Across all times and groups, the  $CD_{\text{base}}$  score for those participants assigned to the Neutral condition ( $M = -.00329 \mu\text{g/dL}$ ;  $SE = .00519 \mu\text{g/dL}$ ) was significantly greater than that of Rejection condition participants ( $M = -.02337 \mu\text{g/dL}$ ;  $SE = .00482 \mu\text{g/dL}$ ). Once again, both of these means are negative, indicating that the effect of each condition resulted in a decrease in cortisol across time and group.

Also consistent with Series 1 ANOVA, Series 2 ANOVA revealed a significant 2 (Condition) x 5 (Time) interaction effect ( $F(1.73, 75.67) = 9.88$ ;  $p < .001$ ;  $\eta^2 = .202$ ). The

pattern of  $CD_{base}$  score differences were identical to that of Series 1 ANOVA. Similarly, “neutral” participants demonstrated significantly greater ( $p \leq .01$ )  $CD_{base}$  scores than “rejected” participants for T6-T1. However, in contrast to Series 1, no significant 2 (Group) x 5 (Time) interaction effect ( $F(1.73, 75.67)=3.94; p=.03; \eta^2=.092$ ) was found.

Finally, tests of within-subjects interaction effects revealed no significant 2 (Group) x 2 (Condition) x 5 (Time) interaction effect ( $F(1.73, 75.67)=1.11; p=.33; \eta^2=.028$ ).

Thus, even with the potential effects of caffeine and alcohol on cortisol controlled for, these data were not supportive of Hypothesis 1. It is also important to note that controlling for these factors involved omitting a subset ( $n=11$ ) equal to about one-fifth of the total sample ( $n=54$ ) from statistical analyses, which further lowered the power of this design.

Due to the potential effects of covariance from external factors on cortisol responses, a 2 (Group) x 2 (Condition) x 5 (Time) repeated measures ANCOVA for  $CD_{base}$  scores was also conducted using SCL-90-R Anxiety scores and Gender as covariates. It was hypothesized that holding both of these factors constant would result in the significant differences hypothesized in Hypothesis 1. A  $p$ -value of .05 was set for this test.

Results of this ANCOVA indicated that neither SCL-90-R Anxiety scores ( $F(2.20, 105.81)=2.34; p=.11; \eta^2=.046$ ), nor Gender ( $F(2.20, 105.81)=1.38; p=.26; \eta^2=.028$ ) was significantly related to cortisol responses. This analysis also revealed a significant between-subjects main effect for Condition ( $F(1, 48)=10.50; p=.002; \eta^2=.18$ ). Across all times and groups, the  $CD_{base}$  score for those participants assigned to the Neutral condition ( $M=-.023 \mu\text{g/dL}; SE=.00464 \mu\text{g/dL}$ ) was significantly greater than that

of Rejection condition participants ( $M = -.00233 \mu\text{g/dL}$ ;  $SE = .00431 \mu\text{g/dL}$ ).

Furthermore, a significant 2 (Condition) x 5 (Time) interaction effect was also found ( $F(2.20, 105.81) = 9.19$ ;  $p < .001$ ;  $\eta^2 = .16$ ), such that Rejected participants ( $M = -.05246 \mu\text{g/dL}$ ;  $SE = .00585 \mu\text{g/dL}$ ) had significantly lower  $CD_{\text{base}}$  scores at T6-T1 than Neutral participants ( $M = -.01208 \mu\text{g/dL}$ ;  $SE = .0063 \mu\text{g/dL}$ ) at T6-T1 ( $F(1, 48) = 10.50$ ;  $p = .002$ ;  $\eta^2 = .18$ ).

Finally, identical to Series 1 and 2 ANOVAs, repeated measures ANCOVA revealed no significant 2 (Group) x 2 (Condition) x 5 (Time) interaction effect ( $F(2.20, 105.81) = .63$ ;  $p = .55$ ;  $\eta = .013$ ). Thus, even when potential covariates were held constant, Hypothesis 1 was not supported by the results of these analyses.

#### Hypothesis 2

Between-groups differences in DERS scores were tested using MANOVA with Bonferroni correction to control for Type I error rates, which are known to be inflated when between-groups differences are analyzed for multiple potentially related dependent variables. A  $p$ -value criterion of .01 was set for all analyses. NSI participants scored significantly higher than control group participants on Nonacceptance of Emotional Responses ( $F(1, 52) = 8.63$ ;  $p = .005$ ), Lack of Emotional Awareness ( $F(1, 52) = 9.43$ ;  $p = .003$ ), Limited Access to Emotion Regulation Strategies ( $F(1, 52) = 8.75$ ;  $p = .005$ ), and Lack of Emotional Clarity ( $F(1, 52) = 9.82$ ;  $p = .003$ ). The between-groups difference for Impulse Control Difficulties indicated a strong trend toward significance ( $F(1, 52) = 4.21$ ;  $p = .045$ ), with NSI participants scoring higher than control group participants. The between groups difference for the Difficulties Engaging in Goal-Directed Behavior subscale was not significant ( $F(1, 52) = 1.31$ ;  $p = .26$ ). Mean scores and standard



deviations of DERS subscales, and effect sizes for between-groups differences are displayed in Table 4.

### Hypothesis 3

Hypothesis 3 was contingent on the results of Hypothesis 1. Because there was not a significant interaction effect between Group, Time, and Condition, Hypothesis 3 could not be tested as stated. Thus, a post hoc revision of Hypothesis 3 (Hypothesis 3a), proposing that DERS subscale scores would correlate significantly and positively with  $CD_{base}$  scores was forwarded. An additional post hoc hypothesis (Hypothesis 3b) proposed that, because cortisol secretion is an index of HPA axis functioning, which is in turn an index of emotional regulation/dysregulation, raw cortisol levels would correlate significantly and positively with DERS in the full sample and sub-samples. Hypotheses 3a and 3b were tested using Pearson bivariate product-moment correlations.

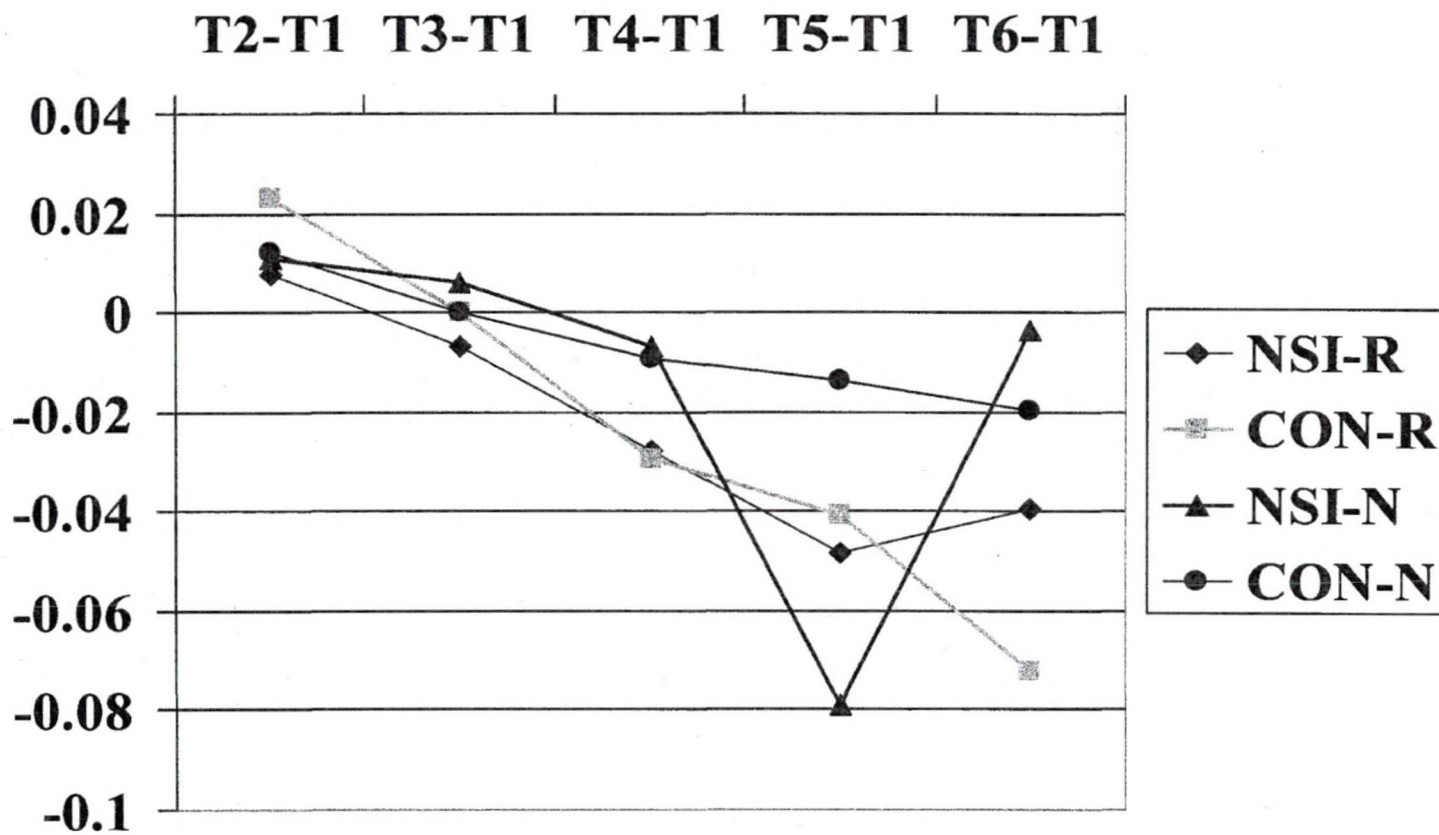
### Hypothesis 3a

Results of correlational analyses for the full sample revealed no significant correlations between any of the DERS subscales and any  $CD_{base}$  scores:  $CD_{base}$  T2-T1 (all  $ps \geq .10$ );  $CD_{base}$  T3-T1 (all  $ps \geq .08$ );  $CD_{base}$  T4-T1 (all  $ps \geq .39$ );  $CD_{base}$  T5-T1 (all  $ps \geq .28$ ); and  $CD_{base}$  T6-T1 (all  $ps \geq .13$ ). Similarly, no significant correlations were found between any of the DERS subscales and any  $CD_{base}$  scores in the Control group:  $CD_{base}$  T2-T1 (all  $ps \geq .12$ );  $CD_{base}$  T3-T1 (all  $ps \geq .17$ );  $CD_{base}$  T4-T1 (all  $ps \geq .28$ );  $CD_{base}$  T5-T1 (all  $ps \geq .12$ ); and  $CD_{base}$  T6-T1 (all  $ps \geq .21$ ).

Table 4. Series 1 Windsorized Mean Cortisol Difference ( $CD_{base}$ ) Scores ( $\mu\text{g/dL}$ ) for NSI and Control Groups by Condition

	T2-T1	T3-T1	T4-T1	T5-T1	T6-T1
NSI Rejected (SD)	.0077 (.0258)	-.0067 (.0215)	-.0276 (.0251)	-.0484 (.0306)	-.0394 (.0240)
Control Rejected (SD)	.0229 (.0423)	-.0001 (.0305)	-.0294 (.0339)	-.0407 (.0401)	-.0720 (.0326)
NSI Neutral (SD)	.0109 (.0084)	.0060 (.0199)	-.0067 (.0243)	-.0789 (.0295)	-.0036 (.0415)
Control Neutral (SD)	.0121 (.0303)	-.0001 (.0294)	-.0092 (.0293)	-.0136 (.0201)	-.0199 (.0267)

Figure 2. Series 1 Windsorized Mean Cortisol Difference Scores ( $\mu\text{g/dL}$ ) for NSI and Control Groups by Condition Across Time



Notes: NSI-R= NSI Rejected; CON-R= Control Rejected; NSI-N= NSI Neutral; CON-N= Control Neutral

Table 5. Mean Scores and Standard Deviations for Difficulties in Emotion Regulation Subscales

	NSI (SD)	Control (SD)	<i>D</i>
NONACCEPTANCE	12.31** (6.49)	8.43 (2.5)	.79
STRATEGIES	14.58** (6.47)	10.57 (2.97)	.79
AWARENESS	16.35** (5.7)	12.18 (4.21)	.83
CLARITY	10.81** (4.14)	7.96 (2.35)	.85
IMPULSE	9.35* (4.99)	7.29 (1.76)	.55
GOALS	13.92 (5.37)	12.18 (5.79)	.31

Notes: \* $p < .05$ ; \*\*  $p < .01$

Correlational analyses of the NSI sample revealed a significant correlation ( $r = .427$ ;  $p = .03$ ) between the DERS Difficulties Engaging in Goal-Directed Behavior subscale and  $CD_{base}$  T3-T1. All other correlations in this subsample analysis were nonsignificant:  $CD_{base}$  T2-T1 (all  $ps \geq .23$ );  $CD_{base}$  T4-T1 (all  $ps \geq .07$ );  $CD_{base}$  T5-T1 (all  $ps \geq .31$ ); and  $CD_{base}$  T6-T1 (all  $ps \geq .21$ ).

#### Hypothesis 3b

Correlational analyses of mean raw cortisol levels and mean DERS subscale scores in the full sample revealed no significant relationships among these variables: T1

(all  $ps \geq .07$ ); T2 (all  $ps \geq .07$ ); T3 (all  $ps \geq .08$ ); T4 (all  $ps \geq .23$ ); T5 (all  $ps \geq .06$ ); and T6 (all  $ps \geq .26$ ). Analyses of Control group participants revealed a significant correlation ( $r = -.394$ ;  $p < .038$ ) between the DERS Difficulties Engaging in Goal-Directed Behavior subscale and raw cortisol level at T3, suggesting that greater difficulties with goal-directed behavior were associated with lower cortisol levels 35 minutes post-stressor for Control group participants. All other correlations in this subsample analysis were nonsignificant: T1 (all  $ps \geq .08$ ); T2 (all  $ps \geq .10$ ); T4 (all  $ps \geq .18$ ); T5 (all  $ps \geq .14$ ); and T6 (all  $ps \geq .12$ ). Finally, bivariate analyses of the NSI sample revealed no significant relationships between raw cortisol data and DERS subscales: T1 (all  $ps \geq .06$ ); T2 (all  $ps \geq .14$ ); T3 (all  $ps \geq .11$ ); T4 (all  $ps \geq .11$ ); T5 (all  $ps \geq .22$ ); and T6 (all  $ps \geq .15$ ).

#### Hypothesis 4

Hypothesis 4 was tested using a series of Pearson bivariate product-moment correlations to evaluate the strength of the relationship between mean PANAS positive affect (PA) and negative affect (NA) scores and respective salivary cortisol level differences ( $CD_{base}$ ) at each of the five post-stressor measurement points in both groups and both conditions. Individual correlational analyses were not significant for NSI Rejected (all  $ps \geq .067$ ); NSI Neutral (all  $ps \geq .086$ ); Control Rejected (all  $ps \geq .135$ ); or Control Neutral (all  $ps \geq .131$ ). Thus, Hypothesis 4 was not supported by these analyses, suggesting that there was not a unique relationship between post-baseline differences in cortisol secretion and PA or NA in any of the cells in this study.

#### Post-hoc Hypotheses and Analyses

In addition to the above hypotheses, two post-hoc hypotheses were tested. First, a 2 (Group) x 2 (Condition) x 5 (Time) interaction effect for PANAS NA and PA scores

was hypothesized. Specifically, it was hypothesized that NSI Rejected Participants would have significantly higher mean differences from baseline in negative affect and significantly lower mean differences from baseline in positive affect than participants in other cells at each post-baseline measurement point. Second, a 2 (Group) x 2 (Condition) x 2 (Gender) x 5 (Time) interaction effect for  $CD_{base}$  scores was hypothesized and tested.

#### *Positive Affect*

Repeated measures ANOVAs were performed on the full sample. The results of these ANOVAs are presented below in text and in Table 5 and Figure 3. Similar to ANOVAs for cortisol data, a test of requisite statistical assumptions indicated that sphericity had been violated ( $\chi^2 = 35.51$ ;  $p < .001$ ;  $\epsilon_{Greenhouse-Geisser} = .732$ ). Therefore, Greenhouse-Geisser corrected  $p$ -values were once again used for significance tests of within-subjects effects for PA score differences from baseline ( $PAD_{base}$  scores).

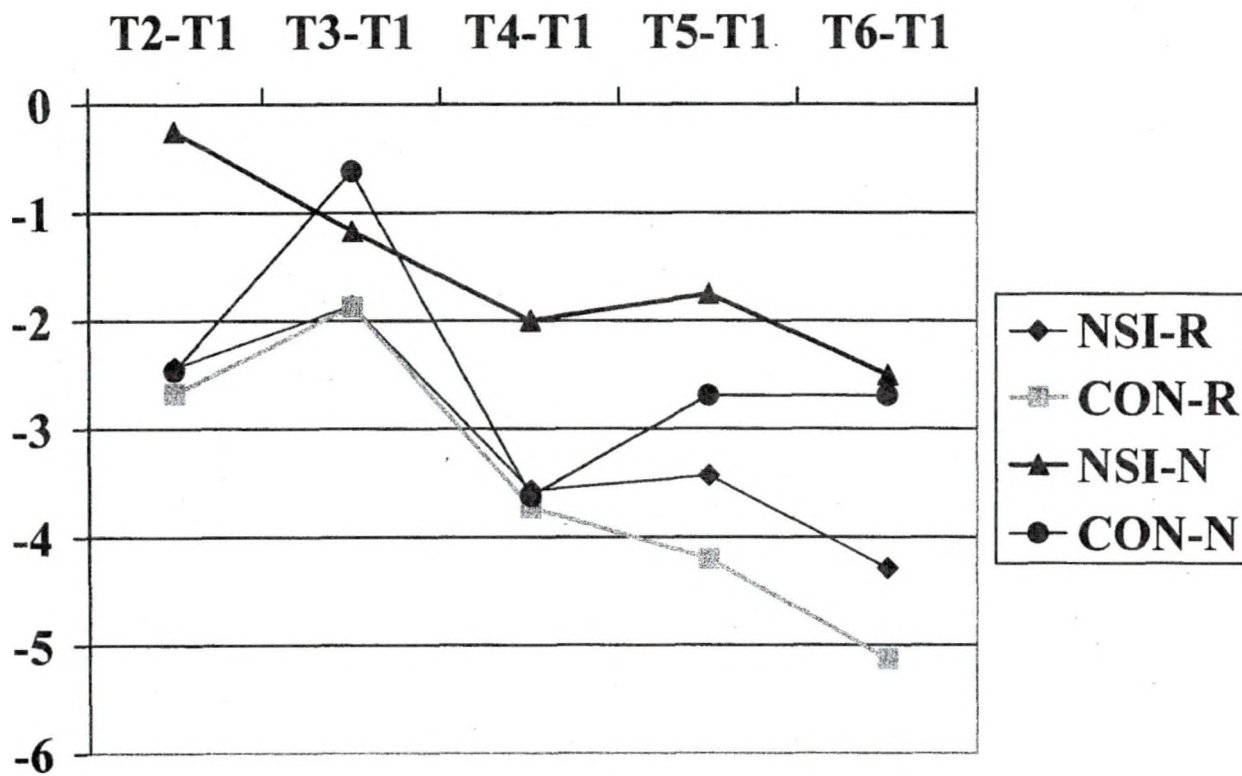
Results of ANOVA for PANAS  $PAD_{base}$  scores revealed no significant between-subjects main effect for Group ( $F(1, 50) = .64$ ;  $p = .43$ ;  $\eta^2 = .013$ ) or Condition ( $F(1, 50) = 1.91$ ;  $p = .17$ ;  $\eta^2 = .037$ ). Conversely, a significant within-subjects main effect was found for Time ( $F(3.31, 165.82) = 5.74$ ;  $p = .001$ ;  $\eta^2 = .103$ ). However, none of the inter-interval differences was significant at a priori levels (all  $ps \geq .012$ ).

Results of this ANOVA also revealed no significant interaction effects for 2 (Group) x 2 (Condition) ( $F(1, 50) = .13$ ;  $p = .72$ ;  $\eta^2 = .003$ ); 2 (Group) x 5 (Time) ( $F(2.93, 146.40) = .61$ ;  $p = .60$ ;  $\eta^2 = .012$ ); 2 (Condition) x 5 (Time) ( $F(2.93, 146.40) = .38$ ;  $p = .76$ ;  $\eta^2 = .008$ ); 2 (Group) x 2 (Condition) x 5 (Time) ( $F(2.93, 146.40) = .54$ ;  $p = .65$ ;  $\eta^2 = .011$ ) comparisons.

Table 6. Mean PANAS Positive Affect Score Differences ( $PAD_{base}$ ) for NSI and Control Groups by Condition

	T2-T1	T3-T1	T4-T1	T5-T1	T6-T1
NSI Rejected (SD)	-2.43 (4.69)	-1.86 (4.11)	-3.57 (4.40)	-3.43 (4.01)	-4.29 (4.56)
Control Rejected (SD)	-2.67 (3.20)	-1.87 (5.28)	-3.73 (4.32)	-4.20 (5.97)	-5.13 (6.50)
NSI Neutral (SD)	-.25 (1.91)	-1.17 (2.76)	-2.0 (2.63)	-1.75 (2.70)	-2.50 (3.63)
Control Neutral (SD)	-2.46 (3.20)	-.62 (3.45)	-3.62 (4.21)	-2.69 (.0201)	-2.69 (4.85)

Figure 3. Mean PANAS Positive Affect Score Differences ( $PAD_{base}$ ) for NSI and Control Groups by Condition



Notes: NSI-R= NSI Rejected; CON-R= Control Rejected; NSI-N= NSI Neutral; CON-N= Control Neutral



### *Negative Affect*

Repeated measures ANOVAs were also performed on the full sample for PANAS NA data. The results of these ANOVAs are presented below in text and in Table 6 and Figure 4. Here too, the requisite statistical assumptions indicated that sphericity had been violated ( $\chi^2 = 90.77$ ;  $p < .001$ ;  $\epsilon_{\text{Greenhouse-Geisser}} = .602$ ). Therefore, Greenhouse-Geisser corrected  $p$ -values were used for significance tests of within-subjects effects for NA score differences from baseline ( $\text{NAD}_{\text{base}}$  scores). Results of the ANOVA for PANAS  $\text{NAD}_{\text{base}}$  scores revealed no significant between-subjects main effect for Group ( $F(1, 50) = .103$ ;  $p = .75$ ;  $\eta^2 = .033$ ), or Condition ( $F(1, 50) = 19$ ;  $p = .67$ ;  $\eta^2 = .004$ ). The test of the within-subjects main effect of Time was also nonsignificant ( $F(2.41, 120.38) = 3.31$ ;  $p = .032$ ;  $\eta^2 = .062$ ). Results of this ANOVA also revealed no significant 2 (Group)  $\times$  2 (Condition) ( $F(1, 50) = .59$ ;  $p = .45$ ;  $\eta^2 = .012$ ); 2 (Group)  $\times$  5 (Time) ( $F(2.41, 120.38) = 1.69$ ;  $p = .18$ ;  $\eta^2 = .033$ ); 2 (Condition)  $\times$  5 (Time) ( $F(2.41, 120.38) = 1.69$ ;  $p = .18$ ;  $\eta^2 = .022$ ); or 2 (Group)  $\times$  2 (Condition)  $\times$  5 (Time) ( $F(2.41, 120.38) = .522$ ;  $p = .63$ ;  $\eta^2 = .01$ ) interaction effects.

### *Gender Interactions*

A repeated measures ANOVA was also performed on the full sample with gender included as a between-subjects factor (in addition to Group and Condition) to test for main and interaction effects of this variable on  $\text{CD}_{\text{base}}$  scores, which may not have been detected in the ANCOVA discussed earlier. As with all previously described ANOVAs, the sphericity assumption was violated ( $\chi^2 = 72.55$ ;  $p < .001$ ;  $\epsilon_{\text{Greenhouse-Geisser}} = .554$ ), and Greenhouse-Geisser corrected  $p$ -values were used for significance tests of within-subjects effects for  $\text{CD}_{\text{base}}$  score differences (see Figure 5 below for Gender comparisons).

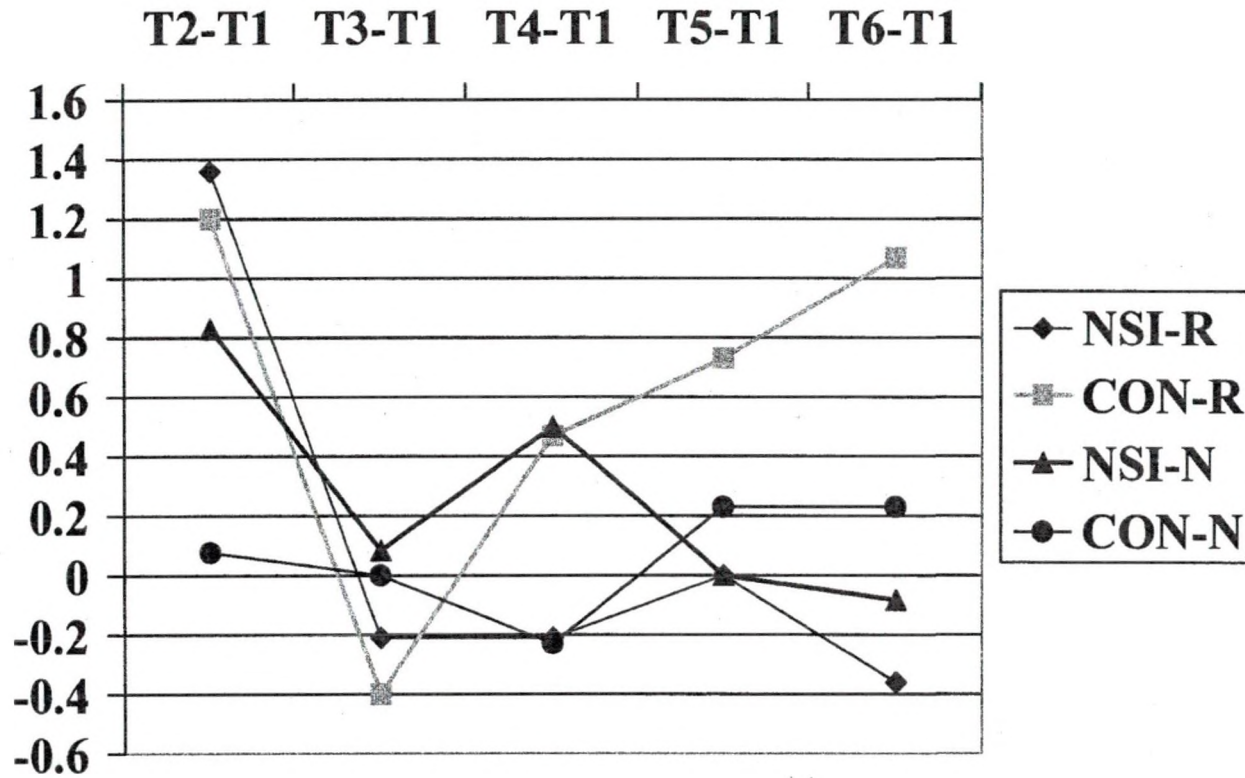
Results of this post hoc ANOVA for  $CD_{base}$  scores revealed no significant between-subjects main effect of Gender ( $F(1, 46)=2.02; p=.16; \eta^2=.042$ ). Additionally, no interaction effects were found for Gender x Condition ( $F(1, 46)=.22; p=.65; \eta^2=.006$ ), or Gender x Time ( $F(2.22, 101.99)=.73; p=.50; \eta^2=.016$ ) comparisons. However, a significant three-way interaction effect was found for Gender x Group x Time ( $F(2.22, 101.99)=5.43; p=.004; \eta^2=.11$ ). This interaction indicated that: (1) mean  $CD_{base}$  scores for NSI males were significantly higher at T2-T1 than T4-T1, T5-T1, and T6-T1  $CD_{base}$  scores; (2) mean  $CD_{base}$  scores for Control males were significantly higher at T2-T1 than T4-T1, T5-T1, and T6-T1  $CD_{base}$  scores; and (3) mean  $CD_{base}$  scores for Control females were significantly higher T2-T1 than T6-T1. This comparison also indicated that mean  $CD_{base}$  scores for NSI females were significantly higher than mean  $CD_{base}$  scores for Control females at T6-T1; and that mean  $CD_{base}$  scores for Control males were significantly higher at T3-T1 than T6-T1.

Although a three-way Group x Condition x Time was not found ( $F(2.22, 101.99)=1.26; p=.29; \eta^2=.027$ ), a four-way interaction effect was found for the Gender x Group x Condition x Time comparison ( $F(2.22, 101.99)=5.61; p=.004; \eta^2=.11$ ). This interaction revealed that: (1) mean  $CD_{base}$  scores for NSI Rejected females were significantly higher than Control Rejected females at T6-T1; and (2) mean  $CD_{base}$  scores for Control Neutral males were significantly higher than Control Rejected females at T4-T1, T5-T1, and T6-T1.

Table 7. Mean PANAS Negative Affect Score Differences (NAD<sub>base</sub>) for NSI and Control Groups by Condition

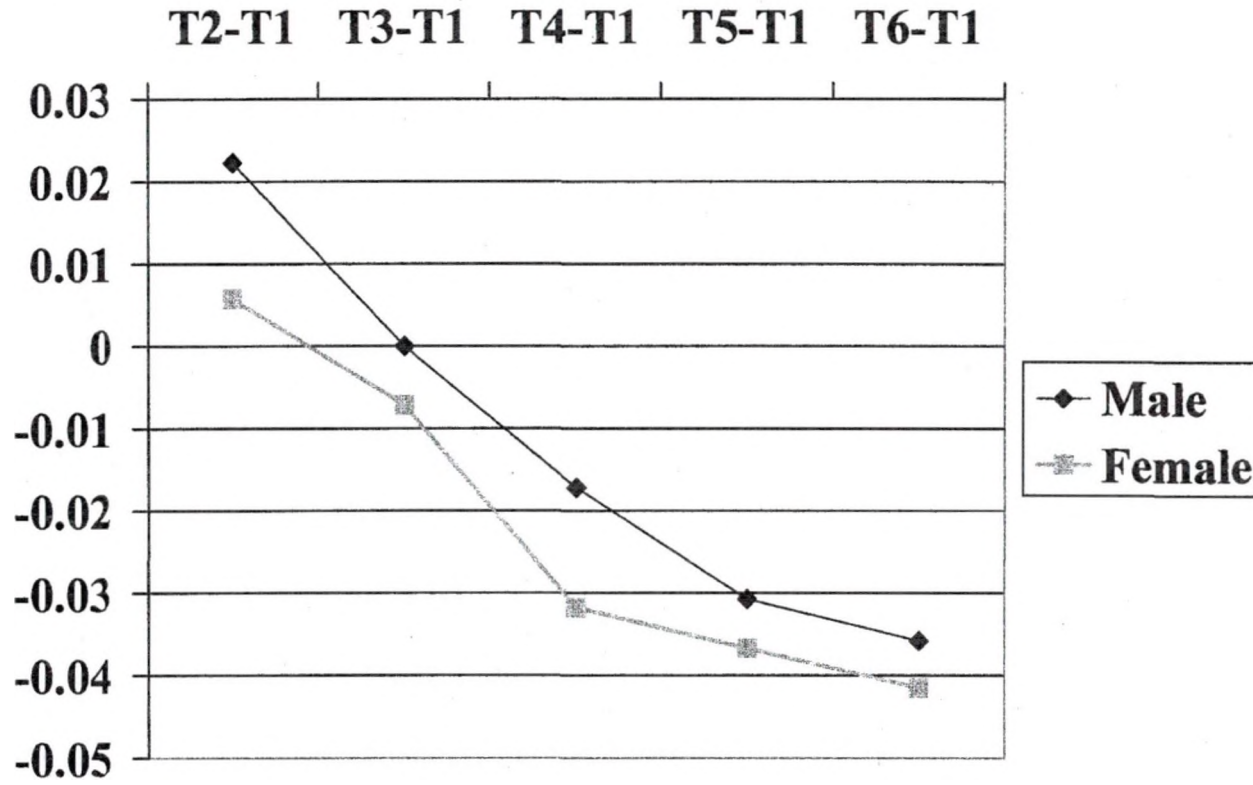
	T2-T1	T3-T1	T4-T1	T5-T1	T6-T1
NSI Rejected (SD)	1.36 (2.37)	-.21 (1.97)	-.21 (1.72)	<.0001 (1.88)	-.36 (2.24)
Control Rejected (SD)	1.20 (1.90)	-.40 (1.99)	.47 (1.77)	.73 (2.34)	1.07 (3.08)
NSI Neutral (SD)	.83 (3.46)	.0833 (3.20)	.50 (3.40)	<.0001 (1.35)	-.0833 (2.68)
Control Neutral (SD)	.0769 (.95)	<.0001 (.71)	-.23 (.73)	.23 (.73)	.23 (1.01)

Figure 4. Mean PANAS Negative Affect Score Differences ( $NAD_{base}$ ) for NSI and Control Groups by Condition



Notes: NSI-R= NSI Rejected; CON-R= Control Rejected; NSI-N= NSI Neutral; CON-N= Control Neutral

Figure 5. Mean CD<sub>base</sub> Scores ( $\mu\text{g}/\text{dL}$ ) by Gender by Time Across Condition



## CHAPTER IV

### DISCUSSION

Non-suicidal self-injury, in all of its various forms, is a dangerous and poorly understood behavior. Despite the myriad explanations that have been forwarded, the exact etiology and functions of NSI remain debatable. The most comprehensive, and arguably most viable, explanatory theory in the study of this clinical phenomenon has historically been the biopsychosocial model. Although some peripheral evidence has been found to support the biological component of this model (e.g., Cassano, Latanzi, Pini, Osso, Battistini, & Cassano, 2001; Chengappa et al., 1999), this aspect of the theorem has nonetheless remained without direct and systematic empirical support.

The present study aimed to examine one potential avenue of biological emotional dysregulation in self-injurers by assessing HPA axis functioning using analysis of cortisol secretion as a proxy measure. The guiding principle of this research was that, if a biological difference in stress response was observed in individuals who engage in NSI compared to healthy individuals who do not, this would contribute to a more complete understanding of NSI by providing evidence of a specific biological factor that may mitigate this behavioral pathology. This research was aimed at providing evidence of the convergence of self-report and biological indices of emotional regulation, so that the relationship between emotional dysregulation and NSI could be better understood.

The data reported in this paper were evidentiary of support for some hypotheses, but not for others. The primary hypothesis of this study was that, when exposed to an

uncontrollable, socially evaluative interpersonal situation in which they were rejected, individuals who engage in NSI would exhibit significantly higher levels of cortisol from baseline than psychologically healthy individuals experiencing the same rejection. The ANOVAs conducted to evaluate this hypothesis found no supporting evidence of such an effect. This remained true even when accounting for the potential effects of alcohol and caffeine consumption via removal of potentially contaminated samples.

Although none of the hypothesized quantitative differences in cortisol were significant a number of interesting qualitative patterns were observed in these data. First, examination of Figure 2 indicates a somewhat contra-hypothetical finding. As hypothesized, there were initial increases in cortisol levels for participants in each cell following the stressor. However, the highest increases were found in the Control Rejected cell, and the lowest increases were found for the NSI Rejected cell. It was proposed that NSI Rejected participants would exhibit the highest initial cortisol secretions. Conversely, these results seem to indicate that this cell scored just slightly lower on average than even those participants in both groups assigned to the Neutral condition.

Although it was comprised of non-significant between-groups differences, the above pattern merits exploration. These differences are not likely to be accounted for by differences in interpersonal sensitivity or social anxiety, as NSI participants actually reported slightly, but not significantly ( $p=.71$ ), higher scores on the SIAS. The NSI Rejected cell evidenced lower  $CD_{base}$  scores at T2-T1 than all other cells, including Control Rejected participants, however the difference was not significant. Another intriguing, though not significant, pattern was observable in the data in Figure 2. NSI

participants (irrespective of condition) tended to exhibit progressive decreases in  $CD_{base}$  scores across time up to 50 minutes post-baseline, followed by a slight increase in cortisol response at 65 minutes post-baseline; whereas Control participants (regardless of condition) exhibited a progressive decrease in  $CD_{base}$  scores that continued for the duration of the experiment. Furthermore, the largest decreases in  $CD_{base}$  scores were also in the NSI group, with the NSI Neutral cell evidencing the largest drop at  $CD_{base}$  T5-T1. This is a complex pattern of data that does not easily lend itself to a simple explanation.

Examination of the data suggests two primary possibilities for the pattern of results described above. One possibility is that the normal cortisol response to stress was inhibited or blunted in these self-injurers. Indeed, HPA dysfunction may be characterized by either a hyper- or hypocortisolemic response. Another potential explanation is that the cortisol response in self-injurers is delayed. This would be supported by the gradual rise that was observed across NSI participants in both conditions. In this vein, it's possible that the measurement simply failed to capture a difference in cortisol response because the timing of collections ended at 65 minutes post-stressor and the cortisol response window is far more protracted for self-injurers. Nonetheless, any inferences in this regard are attenuated by a lack of significance and remain strictly hypothetical.

The cortisol data in the present study may interface with the emotion regulation-biopsychosocial model in a unique way, and theoretically portend other biological mechanisms for NSI to function. Although cortisol secretion patterns are proximally reflective of emotion regulation vis-à-vis HPA functioning, and theoretically should be different in self-injurers than psychologically healthy controls, other biological systems



may be dysregulated in those who engage in NSI. In fact, while the HPA axis is a central mechanism for emotional regulation, it is certainly not the only tract for this process. For example, emotion regulation also involves the orbital-frontal cortex, anterior cingulate cortex, and the amygdala; disruption in any of these circuits has been associated with impulsivity and violence in some prior research (Davidson, Putnam, & Larson, 2000). Previous pharmacological research has also documented that the mechanism of action of some anticonvulsant compounds in Bipolar disordered patients operates through reduction of amygdala reactivity to emotional stimuli (e.g., Drevets et al., 2002; Krystal et al., 2002) and limitation of electrical activity in the anterior cingulate cortex. At the molecular level, anticonvulsants appear to work by potentiating the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) and stabilizing the neuronal membrane at the sodium ion channel through inhibiting the release of aspartate and glutamate (Blumberg et al., 2000; Chengappa et al., 1999; Krystal et al., 2002; Theoharides, Dessain, & Shuster, 1992). This research suggests that any of these neurochemicals may offer an alternative to cortisol as biological mechanisms (or biomarkers) of emotional dysregulation in individuals who self-injure.

Furthermore, there is evidence to suggest that an association exists between beginning anticonvulsant treatment and decreases in the frequency of NSI in certain patient populations (Cassano, Latanzi, Pini, Osso, Battistini, & Cassano, 2001; Chengappa et al., 1999). For example, in Chengappa and colleagues' study of Topiramate (an anticonvulsant) for the treatment of mania, the authors reported an ancillary finding of "near abolition of self-mutilation" (Chengappa et al., 1999, p.5) in two of their Bipolar Disordered participants who were diagnosed with comorbid BPD.

Similarly, Cassano et al. (2001) reported that administration of Topiramate was associated with three-month cessation of NSI in a BPD/Bipolar Disorder woman after two weeks of medication, and a subsequent nine-month cessation of NSI following resumption of this anticonvulsant. Interestingly, the NSI cessation occurred in absence of any change in depressive symptoms. This is contrasted with work by Colman, Newman, Schopflocher, Bland, and Dyck (2004) that found depression to be a primary predictor of “repeat parasuicide,” although this latter study employed an International Classification of Diseases-9 definition that also includes suicide attempts. Nonetheless, this research suggests a biological component to emotion dysregulation may be operating in NSI, but it also suggests that other systems may be more integral to the relationship between this behavior and emotion regulation than the HPAA. For example, dysregulation (excesses or deficiencies) of electrical activity in the orbital-frontal or cingulate cortexes during distress could disrupt executive functioning, and lead to increased impulsive behaviors and reduced inclinations toward self-preserving behaviors. In this vein, future research examining activation of these neural regions using either fMRI or PET imaging of NSI individuals during tasks inducing frustration or negative affect may be useful in furthering the understanding the neural correlates of emotion regulation/dysregulation in NSI.

Alternatively, research has also indicated that chemical stimulation of the central nucleus of the amygdala by glutamate, the secretion of which is inhibited by anticonvulsant compounds, is associated with pronounced cardiovascular reactivity and gastroenterological activity (e.g., ulceration, gastric acid production). These effects of glutaminergic stimulation are associated with poor regulation of (i.e. chronic) anxiety and

fear. Indeed, the dense array of corticotropin releasing hormone (CRH) receptors and nerve tracts in the amygdala make this structure vulnerable to influxes of CRH (DeSouza, Insel, Perrin, Rivier, Vale, & Kuhar, 1985; Uryu, Okumura, Shibasaki, & Sakanaka, 1992). Such influxes are associated with anxiogenic effects. Substantial research indicates that fear responses are attenuated in many species by the introduction locally of GABA or GABA agonists, opiatergic agonists, benzodiazepines, and glutamate antagonists, among others (e.g., Gallagher, Kapp, McNall, & Pascoe, 1981; Gallagher, Kapp, & Pascoe, 1982; Helmsetter, 1993; Roozendaal, Wiersma, Driscoll, Koolhaas, & Bohus, 1992; Shibata, Kataoka, Yamashita, & Ueki, 1986; Sullivan, Henke, Ray, Herbert, and Trimper, 1989; Takao, Nagatani, Kasahara, Hashimoto, 1992). Thus, a failure to inhibit glutamate may result in hyperstimulation of key areas of the amygdala involved in emotion regulation, in turn resulting in decreased stability of neuronal activity in this structure. Decreased stability of neuronal activity may result in decreased overall regulation of emotional behaviors.

As the above discussion of neurobiology pertains to emotion dysregulation and NSI etiology, it is possible that a lower threshold for emotional dysregulation in self-injurers exists. If this is the case, such a lower threshold may be related to higher densities of CRH receptors in the amygdala, more frequent pulses of CRH released into the amygdala, poor inhibition of glutamate, or a combination of these factors. To date, no known research has addressed any of these questions in self-injurers.

It is also important to note that, while the aim of this study was to provide evidence of convergence between physiological and self-report modalities of emotional regulation measurement, there is ample stress-induction literature to suggest that similar

discrepancies are commonly found in this type of research design. Linden's (2004) extensive treatment of the stress research literature accurately notes that it is generally rare for biomarkers of stress to be synchronous with self-report measures. Indeed, the majority of studies find a correlation between subjective ratings and physiological measures of  $r \leq .3$ , with subjective stress ratings accounting only infrequently for over 10% of variance in physiology (Linden, 1987). Earlier work by Pennebaker (1982) found substantial differences in research participants' willingness and capacity to identify and disclose changes in their physiological states. The subjective-objective index discrepancies are likely related to a combination of such reluctance with people's reliance on situational cues for information about their internal state, and the fact that there are very few connections between consciousness and the functions of the central nervous system (Linden, 2004). The aggregate findings from the stress response literature fit with the biopsychosocial model of NSI in that they portend interactive, yet differentially weighted, effects of biology/physiology, social dynamics, and psychological processes. In short, stress research indicates that there are several biological, psychological, and contextual factors that, in concert with each other, determine an individual's stress response; and the relative importance of these factors is likely to be idiographic.

Alternatively, some models of the stress response (Cox & McKay, 1978), or stress regulation, posit that a stress response to a "demand situation" is mediated by one's cognitive appraisal (i.e. perception) of the demand-to-coping resources ratio. This is also amenable to a biopsychosocial approach in that the same three components are represented, required, and interactive in such a model. Here, an imbalance in this ratio in which there are more demands than resources to deal with those demands results in

stress. Thus, “demand” is comprised of actual demand characteristics of the environment plus one’s learning history and idiographic differences of the individual; resources for coping are comprised of a similar combination of factors (i.e. actual resources plus learning and personal history differences). This model is akin to information-processing models of stress responses (Hamilton, 1980), where stress is defined based on the individual’s evaluation of the personal significance of the stress-inducing stimulus and learning history with available coping strategies (e.g., have the resources been sufficient in the past?).

Accordingly, the stressor in the present study may have been judged to be insignificant or meaningless by participants for multiple reasons. First, the stressor was brief and singular, rather than protracted and chronic. The duration of this stressor may have thus been insufficient to engender salience for participants, resulting in a cognitive appraisal of the stressor as non-threatening, or simply not stressful. Additionally, the stressor may have lacked direct personal relevance because the rejection did not originate from a personal acquaintance or a friend. Another consideration here is that almost 50% of the sample suspected that they had been deceived in some way, which had the potential to impact these participants’ stress response.

Alternatively, this stressor may have been consonant with the expectations of self-injurers who were rejected for typical outcomes in social scenarios. Essentially, it is possible that rejected self-injurers have either simply habituated to rejection or have such low self-esteem that they anticipate such rejection. This possibility is supported theoretically by Linehan’s (1993a) concept of the invalidating environment (which is often characterized by rejection) as a spawning pool for self-destructive behaviors. The

possibility that poor self-regard played a role in the cortisol secretion changes would be supported peripherally in work by Scarpa and Luscher (2001), which revealed that self-esteem mediated the relationship between depression and cortisol reactivity. In this study, Scarpa and Luscher found that low self-esteem was associated with decreases in cortisol response to an uncontrollable laboratory stressor in depressed participants, while the reverse was found for high self-esteem. Applied to these results, it is possible that lower self-esteem in NSI participants affected cortisol reactivity by producing decreases in cortisol secretion in response to stress. However this would not account for the same pattern being observed in Control participants. Future research involving similar stress-induction may benefit from having a longer-lasting, more personally relevant, and more effectively deceptive stressor; however, this must be balanced with ethical demands.

Despite the lack of support for Hypothesis 1, partial support was found for other a priori predictions. Among those hypotheses that were at least partially supported was the hypothesis that NSI participants would report significantly more difficulties with emotion regulation. This hypothesis was partially supported in that between-groups differences were significant for most DERS subscales. Specifically, participants in the NSI group reported significantly greater difficulties with emotional clarity, accessing emotion regulation strategies, acceptance of emotional responses, and emotional awareness than Control participants. These results suggest that self-injuring participants in this study perceived themselves as struggling with these aspects of emotion regulation more so than non-self-injurers did, and are accordant with the psychological aspect of the biopsychosocial model. This feature may suggest a psychological substrate for NSI. If individuals who engage in NSI believe they have fewer coping skills in general (or that

they lack the requisite coping skills for a given situation), then they may be less likely attempt to use adaptive coping strategies that are not self-destructive. Again, from an informational processing perspective, if the cognitive appraisal of the demand:resources ratio suggests that one's skills are insufficient for the situation, then distress and self-destruction may ensue. Over time, a general lack of self-efficacy for coping with distress may develop and create a perpetually self-fulfilling prophecy (i.e. "I couldn't cope with X before, I still don't have what it takes to get through X now, so why try anything different when nothing will change.").

Conversely, NSI participants did not report significantly more difficulties with engaging in goal-directed behavior. Given that such behaviors can be instrumental to emotion regulation, it is unclear why this may be. One potential explanation for a lack of a significant difference in difficulties with goal-directed behaviors is that NSI may be viewed by the self-injurer as a goal-directed behavior in and of itself. So, for example, when someone who self-injures endorses "When I'm upset, I have difficulty thinking about anything else," as applying to them 0-10% of the time, this may be because they know that they have their self-injury to focus on. This is supported by both emerging quantitative research (e.g., Himber, 1994; Whitlock, Muehlenkamp, & Eckenrode, in press) and more qualitative, literary depictions of NSI (Miskec & McGee, 2007; Strong, 1998) suggesting that this behavior may be ritualistic, involving extensive planning for at least some people who self-injure. From the quantitative perspective, Whitlock et al. (in press) report that, among college self-injurers, a subset (16.4%) of more severe injurers reported having a regular self-injury routine. About one-third (31.6%) of this same subset also reported NSI characterized by multiple phases, further suggesting a

systematic approach to NSI, even if only inadvertently. However, such an explanation is only one extrapolation borne out of the behavioral-functional models of NSI (e.g., Nock & Prinstein, 2004, 2005), and is based on speculation rather than any extant empirical evidence.

Another possible explanation for a lack of a significant difference in goal-directed behaviors is that self-injurers may lack acceptance (or even acknowledgement) of their emotional states and emotional awareness, both of which were significantly more difficult for NSI participants in this study. In this vein, someone who engages in NSI may not see themselves as having difficulty with “getting things done” when they are “upset” because they are not attending to the “upset” emotions in the first place. Thus, emotional nonacceptance and lack of emotional awareness may theoretically mediate self-reported difficulties with goal-directed behavior or a perceived lack thereof. This would also be supported by the recent work of Whitlock et al. (in press) which found that, at most, less than half of those who engage in NSI may see it as life-interfering behavior. Further research will be needed to establish the validity of any of these possible hypotheses.

One interesting aspect of the DERS data presented here is that difficulty with impulse control was not clearly significantly different ( $p=.045$ ) in NSI participants, which is discordant with some models of NSI and consonant with others (Klonsky, 2007). Some researchers (e.g., Favazza, 1995; New et al., 1995; Welch & Linhan, 2002) have proposed that NSI is a behavior primarily based on problems with controlling one’s impulses (i.e. to self-injure), while others have posited that NSI (especially in BPD) is a “manipulative” strategy, primarily aimed at extracting what is needed/desired from the



environment and those in it (e.g., Adams et al., 2001). Some empirical support exists for the former of these positions, although no clear evidence has been found for the latter. Simeon et al. (1992), for example, found that all participants in their sample of self-injuring and non-self-injuring personality disordered patients had elevated levels of impulsivity, with self-injurers reporting more aggression than non-self-injurers. Other researchers have found that individuals with one impulsivity-based form of psychopathology (e.g., substance abuse, eating disorders) are more likely to meet criteria for other forms of psychopathology that have impulsivity features (Evans & Lacey, 1992; Fitcher et al., 1994). Other work examining clinical correlates of different subtypes of NSI, has recently emerged to show high levels of impulsivity as a characteristic of self-injurers (St. Germain & Hooley, 2008). The current data seem to suggest a modest role at best ( $d=.55$ ) for difficulties with impulse control in NSI. It is possible that this role is mediated by other domains of emotion regulation, and further research will be required to more adequately address this question.

The analyses indicated that Hypotheses 3a and 3b were mostly not supported. However, a modest significant relationship was found between difficulties engaging in goal-directed behavior and cortisol level differences from baseline at 35 minutes post-stressor in NSI participants. Although this relationship was only modest ( $r=.427$ ;  $p=.03$ ), this relationship was not found in the Control group. Interestingly, analyses of correlations between raw cortisol data and DERS scores indicated a modest, but significant inverse relationship ( $r=-.394$ ;  $p=.038$ ) between goal-directed behavior problems and T3 cortisol levels in the Control group, indicating that as cortisol rose in Controls, these difficulties decreased, and vice versa. This finding is also ironic in that

this domain was the only DERS subscale that did not evidence significant between group differences.

Why the relationship between cortisol differences and goal-directed behaviors was significant 35 minutes post-stressor, but not at any other point, and not for Controls, is unclear. Moreover, it is unclear why T3 cortisol levels were significantly related to this same emotion regulation difficulty but not others, not at other measurement points, and not in the NSI group. Nonetheless, these data suggest a relationship between biological and psychological indices of emotion dysregulation in self-injurers that may be mitigated by time. It is possible that following activation of the HPAA, an absence of directed or engaging activity (as was part of this study's design) is associated with further increases in HPAA activity. If one assumes that being shut in a relatively small (in some cases windowless) room for two and a half hours is distressing (as is qualitatively supported by the comments noted earlier), then it is possible that this stress interacted with a potentially lower threshold for distress in NSI participants, which in turn activated the HPAA in absence of access to the persons typical method of coping using NSI. The time at which this occurred may represent a critical juncture at which NSI is more likely to occur, meaning that there is perhaps a vulnerability window, within which NSI is more/most likely to occur. Future research must seek to replicate this finding, as doing so may prove to be valuable in translating a phenomenological understanding of NSI into treatments for this behavior.

Finally, the lack of support for Hypothesis 4 indicates that no relationship was present between self-report measures of negative and positive affect (i.e. PANAS scores) and cortisol responses in any of the cells in this study. Considered in the context of the

other findings (or lack thereof) in the present study, this finding adds a further complexity to a pattern of discrepant data. In aggregate, these results suggest that self-injurers in this study were more likely than Controls to report global difficulties with emotional regulation; however, these problems were not related to ecological momentary assessment of affect in either group in either condition. This is consistent with previously discussed research, which has indicated that such a discrepancy is common for a variety of reasons. Post-hoc ANOVAs of PANAS PA and NA difference scores nearly mirrored cortisol level difference ANOVA results, in that no interaction effects were found; NA initially increased following exposure to the stressor, and subsequently decreased throughout the rest of the study; and PA progressively decreased across all measurement intervals. The pattern of results was indeed convergent, i.e. NA changes converged with cortisol changes, only not in the predicted direction and not to a statistically significant degree. It is possible that researcher demand effects generated the initial differences in cortisol and affect, but that these effects were short-lived, thus resulting in an inconsistent pattern of relationships among physiological and psychological variables. Regardless, evidence for an unreported extant effect of the stressor was not found, and it seems likely that no relationship in this regard exists among this sample of self-injurers.

#### Study Limitations and Strengths

##### *Limitations*

Although the results of the present study were not supportive of the main hypotheses and revealed no significant differences in HPA function between self-injurers versus controls exposed to psychological stress, several potentially mitigating limitations of the study must be acknowledged. First, despite designed and purposeful

efforts to recruit a heterogeneous sample, the sample for the present study was comprised entirely of college students, many of whom were enrolled in undergraduate psychology courses, which is a psychosocially idiosyncratic subset of a larger unique subset (i.e., college students) of the population (King et al., 2004). There is a strong possibility that different results may be found in clinical samples of self-injurers (e.g., inpatient or outpatient), because more severe psychiatric impairment is associated with more distress (Coyne & Schwenk, 1997) and could hypothetically be associated with a greater degree of biological emotional dysregulation than evidenced in this study. Thus, these results will likely not be generalizable to populations outside of a university setting.. As Foot and Sanford (2004) note, it is questionable at best to assume that college student samples are representative of the general population.

On the other hand, it is reasonable to assume that college students, regardless of major, are somewhat higher functioning than individuals of a similar age who have never been college students. Thus, it is likely that this study examined a relatively higher functioning population than that which is typically seen in outpatient and inpatient facilities. Nonetheless, 57.7% (n=15) of the NSI participants reported a history of “counseling or psychotherapy,” compared to only 7.1% (n=2) of controls ( $\chi^2 [1, 53]=15.97; p < .001$ ). This dynamic of the sample seems to suggest that, although the sample was likely to be higher functioning in general, self-injurers may be more likely to have psychological difficulties requiring treatment than psychologically healthy individuals.

Another limitation of this study is that the sample size was relatively small. This was a pilot study, and a priori power analysis suggested that moderate effect sizes could be detected if present. It is possible that there were small effects not detected due to the

power restrictions based on the current sample size. Nonetheless, additional participants may have strengthened these results by adding further power with which to draw conclusions. Future studies must seek to extend these findings by employing larger samples.

Connected to the above sample limitation was this study's lack of comparably sized gender group subsamples. While the inclusion of male self-injurers is a strength, the gender imbalance in the Control group limits the strength of any conclusions regarding the relationship of gender to NSI and emotion regulation. Although this imbalance occurred inadvertently, and in a design aimed at randomly sampling the population from which it was drawn, it is clear that matching Control and NSI groups on this variable would have strengthened the power of the study's design. Some of the weak but significant interaction effects found for gender may have been strengthened by larger cell sizes.

Also related to the limitations of the sample was the definition of study groups. Participants met criteria if they had engaged in NSI at least two times in their life with one episode occurring in the past 12 months. This means that someone could have reported a single episode of NSI at age 12 and then a second episode 11 months before the screening and have met criteria for the study just as easily as someone who reported cutting themselves daily for the past four years would have. Therefore, the heterogeneity of the severity of NSI in the sample is also a potential limitation. Someone who received intensive treatment for their NSI and was abstinent for several months could still have participated in the study as a NSI participant, despite the fact that they may have been more likely to have developed better emotional regulation skills through treatment than

others. Qualitatively, four NSI participants reported taking psychotropic medications, while none of the control participants did. Thus, there may be some indication in these data that treatment of some form had a mitigating effect on emotional dysregulation.

Additionally, participants were not excluded based on mood or anxiety disorder history as long as the diagnosis had not occurred in the past month, which allowed for the possibility that people meeting full mood disorder criteria could have been included and that people in remission from a mood or anxiety disorder could have been included. Either of these would be supported by the fact that four participants reported currently taking either selective serotonin reuptake inhibitors or selective norepinephrine reuptake inhibitors. Simultaneously, this exclusion criterion also limited some otherwise eligible participants who had reported a more severe history of NSI (i.e. more methods, higher frequency). Thus the inclusion/exclusion criteria may have contributed to a restriction in the full range of self-injurers available within this population because people in the acute stages of mood symptomatology were not allowed to participate. Designers of future research in this area may consider foregoing such restrictions in favor of a better representative sample, despite the risk of confounds to cortisol data. After all, the emotion dysregulation model of NSI directly implies impairment in emotion regulation, which can manifest in a variety of forms of psychopathology.

A further limitation of the study is the inherent variability of cortisol. The sensitivity of cortisol to stress induction and emotion made it an excellent candidate as a biomarker of HPA axis functioning, but this glucocorticoid is affected by a variety of factors, including the time of day, physical activity, brain activity, pregnancy, mood, environmental factors, and even posture (Hennig Friebe, Ryl, Kramer, Bottcher, &

Netter, 2000). The design of this study was aimed at maintaining as much control of the quality of the cortisol data as possible through restricting use of certain substances and participation in activities that are known to affect cortisol secretion. However, these measures relied almost exclusively on “the honor system” of self-report, and adherence cannot be determined definitively. Qualitatively, a number of the participants contacted the principal investigator to inquire about whether certain behaviors or substances would disqualify them from the study, or to provide notification of a potential lapse in adherence. Nonetheless, the individual variation in cortisol secretion patterns and the number of potential confounds for this data limits the tenacity with which conclusions may be drawn.

This study was also limited by its reliance on self-report NSI data, which is known to be somewhat unreliable (Klonsky, 2007). While the two week test-retest reliability data for the DSHI ( $\alpha=.68$ ;  $r=.92$ ) are strong, they are also indicative of variability in reporting the same behaviors across a short period of time. A variety of factors can influence whether or not a participant accurately reports their NSI history, including perceived experimenter demands and prior learning history related to disclosure of NSI. In the present experiment, for example, the compensation component of the study design also provided an incentive for participants recruited via advertisements to embellish or even fabricate their history of NSI to receive financial compensation that they otherwise could not obtain. While there is neither quantitative nor qualitative data generated in the present study to support such a contention, this possibility highlights the concerns inherent in relying on self-reported NSI data, and beckons for more innovative and accurate approaches to NSI assessment.

A final limitation of this study was the technique used for the experimental manipulation. This technique has been previously used effectively in several studies to experimentally produce a cortisol/stress response (cf. Blackhart et al., 2007), and it is founded on a large body of literature (see Dickerson & Kemeny, 2004). Nonetheless, the effectiveness of this technique is contingent upon several factors in its implementation. First, the appropriate wording of feedback must be used. This study used feedback that was based on work by Blackhart et al. (2007) in their study of cortisol response in social anxiety. One modification was made to the Rejected feedback to increase the harshness and impact of the wording, which included adding the statement. "This is kind of unusual, and it's never happened before." It is possible that this variation actually decreased the believability of the feedback. Use of the original, unaltered feedback script for future research may be advisable to reduce the risk of such iatrogeny.

Second, the delivery of feedback must be effective, involving minimizing emotive behavior and changes in facial affect (i.e. "keeping a straight face"). Although some research personnel associated with this project reported and exhibited initial difficulty with delivery of feedback effectively, delivery improved substantially during the training process. Nevertheless, it is possible that behavioral drift occurred over the course of the experiment, resulting in a diluted impact of this stress-induction technique. It will behoove future researchers employing a similar design to conduct regular ongoing training of research personnel throughout the duration of the study.

Third, environmental factors within the experiment must be conducive to the believability of this feedback. Most experiment groups consisted of four participants. Although participants were told that they would get to work with "at least one of the



other participants” during “the second half of the experiment” depending on whom picked whom to work with, it is possible that a group number in which pairs could be matched up evenly made this less believable (i.e., participants perhaps “did the math” and disregarded the “at least one other person” statement). Moreover, although all participants remained in their respective lab rooms before and after the conversational task and had no other potential opportunities for interaction with other participants, the only noise of people moving around outside of the lab rooms was likely to be easily identifiable as the research personnel. Anecdotally, during debriefing one participant commented, “I knew something was up- everyone went back to the same rooms, and I never heard anyone else walking around or getting together in a group.” The lab rooms used in this experiment were also poorly insulated for sound and were close together, in some cases adjacent. Therefore, it is possible that participants overheard some interactions between research personnel and other participants, thus contributing to disbelief (among Rejected participants) that other participants were gathered together working in a group. Future studies involving social rejection stressors must control such environmental factors that potentially degrade the believability of experimental deception.

Finally, the deception used for participants in the Neutral condition may also have had an iatrogenic effect, especially for the nearly half (48%) who suspected deception. Instead of having a null effect as anticipated, it is possible that this deception resulted in annoyance or other negative emotion, which in turn produced a cortisol response comparable or higher than that of Rejected participants. This is also consistent with qualitative data from the experimental manipulation check, in which some participants

appeared not to have believed this deception either, as denoted by one Neutral condition participant's response, "You made 'a mistake?' Yeah, right." Future research using this stressor must be designed to control for this possibility.

### *Strengths*

While the aforementioned limitations must be considered, the present study was also characterized by several methodological strengths. First, this study was the first known research to employ both biological and self-report measures of emotional duress in the study of emotional regulation in NSI. Cortisol is a well-known proxy measure of HPA functioning, which in turn is a well-established neurological component of emotional regulation. However, studies of NSI have typically used only paper-and-pencil assessments for mood, affect, or emotional state. This study is among the first to employ biological measures in the study of NSI, and no known studies have used cortisol as a measure of emotional dysregulation following stress induction in this population.

A second key strength of this study that merits discussion is the demographic composition of the sample; specifically, in the NSI group, males were included, and nearly equally represented. Older individuals were also included in the NSI group. This feature of the present research is a movement away from the long-held, and misconceived stereotype of the mid- to late adolescent (or very young adult) female self-injurer (e.g., Strong, 1998). Recent work by Gratz (2001) and a variety of others (e.g., Nijman, Dautzeberg, Merckelbach, Juang, Wessel, & Campo, 1999; Nock & Prinstein, 2005; Stanley et al., 2001) indicates that NSI is a problem in males, but that this behavior may develop for different reasons and may have different manifestations (e.g., different methods, different body parts; Whitlock et al. [in press]). Such work has established that

the aforementioned stereotype is passé, has resulted primarily from sampling bias, and has served mainly to hinder progress in the study of NSI. While NSI is becoming more widely recognized and documented as an equivalent problem in males (Gratz, 2001; Klonsky, 2007), self-injuring men remain an understudied population. This study makes a purposeful step forward in this regard, especially by its inclusion of males.

The setting for this study is also one of its strengths. Research in both Europe (Lehtinen et al., 2003) and North America (Mueser, Essock, Drake, Wolfe, & Frisman, 2001) suggests that there may be differences in the prevalence of psychopathology across different types of national regions. Clearly, there are differing sociocultural caveats and dynamics in different regions of North America that potentially alter reporting rates and manifestations of behaviors such as NSI. Whereas prior research on NSI in college students has been primarily conducted at larger, urban-based universities in western or eastern North America, the present study examined a sample drawn from a medium-sized, rurally based university in the North American Midwest. Thus, the present research provides NSI data on an understudied subset of the general North American population. Thus, the generalizability of these results to similar populations may have been higher than prior research in different regions.

Some aspects of the experimental design employed in this study may also be considered strengths. First, the stressor that was used in this research was based on decades of research examining the biology of stress responses in human. The characteristics of this stressor (uncontrollability and social evaluation) have been demonstrated to produce the strongest cortisol response in research participants when combined together as they were in this experiment. Thus, there is strong research support

to indicate that this method was highly likely to produce a strong cortisol response (Dickerson & Kemeny, 2004). Additionally, the stressor used was social in nature, and relied on social exclusion to produce stress. It is likely that this stressor presented a more realistic context than an abstract stressor task (e.g., performing mental calculations out loud for a mock or real audience) because actual or perceived social exclusion is something that people, self-injurers included, are likely to experience. This stressor was also designed to appear as a non-routine and unexpected part of the participant's experience during the study, thus making it more difficult for participants to actively cope with any resulting stress by attributing the manipulation to meaningless or arbitrary research demands.

#### Future Directions

This research represents an important initial step toward examination of potential underlying biological components of NSI. While the limitations of this study do not inherently portend invalidity of the results, and must be balanced with the strengths of this research, they do merit some caution when extrapolating these findings. Future research must seek to replicate these results before any firm conclusions may be drawn or applied from them. It is incumbent upon future researchers to examine other facets of emotion regulation in self-injurers.

The lack of significant findings in the present study both furthers and frustrates the advancement of our understanding of NSI etiology. The pattern of results points toward some potentially fruitful lines of inquiry that may be addressed in future studies. First, other indices of emotion regulation in the brain must be evaluated in self-injurers under stressed and non-stressed conditions. Investigations of receptor density in the

amygdala may provide support for a hypo-threshold model of emotion dysregulation in NSI, whereby higher densities of CRH in the central nucleus of the amygdala could theoretically (1) lower the threshold for emotional reactivity; (2) increase the amount of stimulation of the amygdala; and/or (3) protract the chronicity of negatively valenced emotional responses in self-injurers, such as fear.

Second, subsequent investigations in this area must evaluate the role of other potential mitigating factors such as severity of NSI pathology (e.g., frequency, duration). Regardless of the model that is used to explain NSI, higher frequency, higher dangerousness, and chronicity are likely to suggest disruption in the person's life at some level. It is possible that these factors bare some unique relationship to the function of NSI in a self-injurer's life. As suggested by Klonsky (2007), it is possible that the function of NSI changes over time for people, and these changes may be associated with lesser or greater levels of risk for other forms of psychopathology, as well as greater or lesser applicability of psychotherapeutic interventions. For example, evaluation of different durations of NSI history, ages of onset, and different severities of NSI as predictors of initial readiness-to-change may facilitate a better understanding of the chronic nature of much NSI. However, it is the *determination* of the role of these factors that is the first step in this direction.

Third, in lieu of the null results of this study, future research must turn attention toward the expansion of alternative extant models of NSI. The behavioral model proposed by Nock and Prinstein (2004) was indicative of emotion or affect regulation as a function of NSI in adolescent self-injurers. However, it is also quite possible that NSI develops primarily through a process of chaining, paired association, and contingencies

of reinforcement inadvertently initiated by either the individual who engages in NSI or his/her environment. Although self-damaging behavior would seem to be a “failed mutation” in an evolutionary sense, it is possible that behavioral principles combined with a conducive environment make this behavior more functional for the self-injurer than is commonly assumed by either the pedestrian general public or the seasoned clinician.

Fourth and finally, it is clear from this study that the relationship between gender and NSI has yet to be elucidated. Clarification of the way in which one’s interpersonal and intrapersonal experience of gender is needed. It is imperative for future research designs to incorporate balanced gender subsamples, in an effort to better understand this relationship. Future research examining emotion regulation and NSI especially must seek to address questions pertaining to the role of gender in these psychological phenomena. Doing so will ideally develop an empirical background against which new, targeted treatments for NSI may be developed.

These proposed directions are but a few of the myriad avenues yet to be pursued in NSI research, many of which are beyond the scope of this discussion. In general, research on this behavior remains relatively nascent at this time, given our limited understanding of it. Substantially more work is required for a useful and comprehensive model of NSI to be advanced. The findings of this study are merely a small step in a long road of empirical discovery yet to be traveled by behavioral science.

The pattern of differences presented in this paper is important and amenable to a biopsychosocial understanding of NSI. The primary hypothesis regarding cortisol was not supported, but self-injurers in this study essentially stated that they have significant

problems with emotional regulation, thus supporting the psychological component of this model. Although it appeared that NSI participants' bodies told a different story biologically, it is possible that what was uncovered in this experiment is a unique neurobiology requiring further exploration with more sophisticated techniques in future research. As Carl Sagan (1994) once noted, "Absence of evidence, is not evidence of absence." Indeed, the discrepancy between biological and psychological measures of emotional regulation seems to point toward some important, yet unexplored, avenues of empirical inquiry. It is imperative for future researchers in this area to continue to advance our understanding of the biological component of the biopsychosocial model of NSI, in an effort to provide a more complete understanding of and more effective treatments for this behavior.

## APPENDICES



## APPENDIX I

### Informed Consent Form

This study is being conducted by Patrick Kerr. I am a graduate student in the psychology department and am coordinating this research project. My advisors for this project, are Dr. Jennifer Muehlenkamp, and assistant professor in the Department of Psychology, and Dr. Alan King, an associate professor in the Department of Psychology, both of whom are supervising this research. The psychology department supports the practice of the protection for this project of human subjects in experimental research, in accordance with the Ethical Principles for Psychologists put forth by the American Psychological Association.

The purpose of this research is to study certain biological factors associated with non-suicidal self-injury (NSSI). Both individuals with and without a history of NSSI have been invited to participate in this study. The experimenter conducting the study with you today has not been informed about which group you are in.

This experiment has been approved by the University of North Dakota Institutional Review Board. Your participation in this research is entirely voluntary. You were invited to participate in this study based on your responses on the questionnaires administered in the group testing session at the beginning of this semester, in which you participated. The following information is provided so that you may decide whether or not you wish to participate in this study. You are free at any time during the experiment to withdraw your participation for any reason. Also, if you decide not to participate in this study, your decision will not affect your course grade or your relationships with psychology faculty members. You will receive four (4) hours of extra credit to be used toward a psychology course, or \$20 for participating today. If you discontinue your participation early, you will be compensated at a rate of 0.5 hours of extra credit and \$2 for every half-hour or part thereof that you participate.

In this study, you will be asked to engage in conversational tasks with other participants. These other participants are also unaware of what group you are in. These tasks will require you to discuss certain topics indicated by the experimenter. The time required for any conversational task that you participate in will not exceed 15 minutes. Prior to the conversational tasks, you will be asked to complete a questionnaire pertaining to your recent activities. The questions on this questionnaire will determine your eligibility to participate, and will inquire about the following activities:

- 1) Caffeine intake
- 2) Drug use
- 3) Prescription medication use
- 4) Food consumption
- 5) Physical activity and exercise

Prior to the conversational tasks, the experimenter will ask you to engage in a relaxation activity involving listening to music and reading through some magazines. This will last for 20 minutes. Following this, the experimenter will ask that you provide samples of saliva at designated times. The experimenter will provide you with the necessary materials to do this. You will then engage in a second task involving drawing with selected members of the group. After all conversational tasks have been completed, the experimenter will ask that you provide additional samples of saliva at designated times. The experimenter will provide you with the necessary materials to do this at this time as well. Each time you provide a sample of saliva, you will be asked to complete a brief questionnaire. At the conclusion of the experiment, you will be asked to complete two more questionnaires. One questionnaire will inquire about your

thoughts on this experiment, and the other will inquire about thoughts related to self-injury. Finally, you will be asked to copy some statements on a page provided by the experimenter.

**The criteria for participating in this study are that you are at least 18 years of age, that you are not currently pregnant, that you are not currently taking oral contraceptives, that you have complied with the requirements for pre-experimental food and drug intake and physical activity, that you have not been diagnosed with any form of mood disorder (i.e., Major Depressive Disorder/Clinical Depression, Bipolar Disorder, Dysthymia, Cyclothymia) or anxiety disorder (i.e., Generalized Anxiety Disorder, obsessive-Compulsive Disorder, Posttraumatic Stress Disorder, Panic Disorder) within the past month, and that you have not been diagnosed with an eating disorder at any time in your lifetime. Verification of this compliance will be made when the samples are analyzed.**

Your course grade will not be affected by a decision not to answer any item on these questionnaires. If you have not complied with the pre-experiment instructions regarding food and drug intake, and physical activity, you may not participate in this study research today; however, you may elect to reschedule your participation in this study for another day, with no consequence to you.

This experiment should last no more than two and a half hours, for which you will receive a total of four hours of extra course credit, or \$20. If you discontinue your participation prior to completion of the experimental session, you will still be compensated based on the time you have contributed. Specifically, you will be provided with ten dollars or two hours of research participation credit for every full hour that you have participated (e.g., for 1 or 1 ½ hours of time spent, you would receive \$10 or 2 hours of extra credit participation).

All data collected in all experimental testing sessions will remain confidential and will be used for research purposes only. Random identification numbers will be assigned to each participant so that your responses and data (including salivary sample data) will not be identified by either your name or your student number. This random number will not be your social security number or your student number, but rather an unassociated random number. All biological samples will be analyzed by Salimetrics, Inc.; however, your identity will not be linked with your individual samples in any way or at any time. This consent form will be stored separately from the data collected in the experimental sessions, meaning that your data will not be connected with any identifying information at any time. However, we will include your random number on this consent form in case you have questions about this research or would like to discuss your responses with us. The data from this experiment, which includes the questionnaires you complete and the biological analysis reports generated by Salimetrics, Inc., as well as the consent forms will be stored in locked cabinets for a period of three years following the completion of this study. After three years, all data will be destroyed using a paper shredder.

There may be no benefits for participating in this study beyond gaining experience in scientific research. On a larger scale, it is expected that this study will benefit the larger field of clinical psychology because it will yield data that will be important in understanding new aspects of certain behaviors in humans. It is expected that the tasks used in this procedure will provide useful information that can one day be used to develop new psychological treatment approaches for certain behaviors.

Potential risks to individuals who participate in this study may include emotional discomfort or distress due to certain components of the experimental procedure, and discomfort in rating behaviors pertaining to situations that may elicit fear or worry in some individuals. If you experience such discomfort, please feel free to contact me to discuss your experience in the study. At the conclusion of your participation in this study, you may be provided with, or may request, a list of community and campus resources where you can receive psychological services either at no cost (e.g., University Counseling Center) or on a sliding fee scale (e.g., Psychological Services Center).

Another potential risk in this study is providing data that might be linked with your name. As stated above, a number of steps will be taken to maintain confidentiality. Moreover, all of the personnel associated with this study have gone through a confidentiality workshop, which includes a review of the ethical principles published by the American Psychological Association. Only the researcher (Patrick Kerr), the research advisors (Jennifer Muehlenkamp, Ph.D. and Alan King, Ph.D.), and people who audit IRB procedures will have access to the data. However, please be aware that if at any time during the experiment you express any desire or intent to harm yourself in any way, the experimenter will be obligated to breach your confidentiality as a research participant and take appropriate measures to ensure your safety. This may include contacting one of the clinical psychologists supervising this study (Jennifer Muehlenkamp, Ph.D. and Alan King, Ph.D.), the University of North Dakota Crisis Response Team, or other emergency personnel.

You will be given a thorough debriefing of the rationale behind the study, expected results, and the manner in which this study might benefit individuals with certain behavioral tendencies at the end of the experimental session. It is expected that results from this study will be presented at conferences and published in a peer-reviewed journal. All data will be presented as means and standard deviations, and no individual data set will be published.

If you have any questions or concerns about this research, please do not hesitate to call me at (701) 777-4348. You can also contact Dr. Jennifer Muehlenkamp at (701) 777-4496 or Dr. Alan King at (701) 777-3644. If you have other questions or concerns, you may call UND's Office of Research and Program Development at 777-4279. The experimenter will provide you with a copy of this form to keep for your own records. Your signature below indicates that you have thoroughly read this consent form and voluntarily agree to participate. Thank you.

---

Participant Signature

---

Date

## APPENDIX II

### Debriefing Statement

Now that you have completed your participation in this study, we would like to explain its purpose. The main purpose of this study is to study the biological responses of individuals who engage in non-suicidal self-injury to emotional distress. It is theorized that individuals who engage in non-suicidal self-injury do so for a variety of reasons, one of which may be to help moderate negative feelings and emotions. The feedback that you were given by the researcher today was pre-selected prior to the experiment based on your random assignment to either the "neutral group" (if you were told we made a mistake and were assigned to the wrong group) or the "stressed group" (if you were told that no one chose to work with you). Therefore, any feedback you were given by the experimenter regarding whether or not you were selected by someone else to work with during the second part of the experiment was fictional. In fact, it is likely that some of the other participants in today's study were given the same feedback that you were. This is referred to as a "social rejection technique." It was part of the experiment, and was aimed at eliciting an emotional response from you which could be measured using biological indicators in your saliva. This was a deceptive technique and we sincerely apologize for using it. Unfortunately, this is one of the most consistent techniques used in research for eliciting emotional responses in research participants. By studying the way in which self-injurers and those who do not engage in self-injury respond biologically to stress, we can better understand both the reasons that people engage in self-injury and how to treat this behavior more effectively.

It is possible and may even be expected that you felt uncomfortable while engaging in the conversational task, and when given feedback about who had supposedly selected to work with you. The feelings that you felt during this experiment may have ranged from completely neutral to sad, angry, anxious, or disturbed. Any and all of the feelings you experienced are normal reactions to this procedure and are shared by many others who go through this experiment. Moreover, there is ample research suggesting that these are very typical responses to this procedure. The discomfort associated with this experiment is expected to be temporary; however, the exact duration is unknown. If, for any reason, you would like to discuss these feelings with the primary investigator of this experiment or my supervisors, we will be available to speak with you. All office numbers, telephone numbers, and email are listed below. If these feelings persist, it is also strongly suggested that you speak to a mental health professional.

As mentioned at the beginning of this study, all of your responses and all of your data will be kept strictly and completely confidential. None of the data that you have personally submitted will be used on an individual basis in any way, and all of the results gathered from this study will be compiled, presented, and reported as group statistical averages. Likewise, while it is understandable and appropriate that you may want to discuss your experiences today with a mental health professional or other supportive person, we request that you do not discuss this experiment with other undergraduate students so as to avoid biasing responses obtained from future participants.

We sincerely appreciate your participation and cooperation with our study!

Patrick Kerr, Primary Investigator  
Office: 113 Corwin-Larimore  
Phone: 701.777.4348  
[patrick.kerr@und.nodak.edu](mailto:patrick.kerr@und.nodak.edu)

Jennifer Muehlenkamp, Ph.D., Research Supervisor  
Office: 337 Corwin-Larimore  
Phone: 701.777.4496  
[jennifer.muehlenkamp@und.nodak.edu](mailto:jennifer.muehlenkamp@und.nodak.edu)

Alan King, Ph.D., Research Supervisor  
Office: 339 Corwin-Larimore  
Phone: 701.777.3644  
[alan\\_king@und.nodak.edu](mailto:alan_king@und.nodak.edu)

APPENDIX III

**My Experiences**

In your own words, what was the present study about?

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Did you believe, at any time, that the experiment dealt with anything other than what the experimenter had described to you (circle one)?

Yes                  No

If yes, what?

---

---

Did this affect your behavior in any way (circle one)?    Yes                  No

If yes, how?

---

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---

---

Were you given any information about the experiment by anyone other than the researchers prior to coming here today (circle one)?    Yes                  No

If yes, what?

---

---

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## APPENDIX IV

### Positive Self-Statements

**Directions:** Please read each of the following statements to yourself now, and copy each statement on the line below it. This page is yours to keep, so you may take it with you if you choose and use it as often as necessary.

**"I am an intelligent person"**

---

**"I can succeed at anything I set my mind to"**

---

**"I have special talents and abilities"**

---

**"It's okay to just be me"**

---

**"My opinion is just as important as the next person's"**

---

**"I am not stupid"**

---

**"I am smart and can figure things out just as easily as others"**

---

**"It's okay not to be perfect"**

---

**"I am competent at many things"**

---

**"I am a strong person"**

---

"I can achieve anything I set my mind to"

---

"I have many strengths"

---

"I have much potential"

---

"I am worthy of a happy and fulfilling life"

---

"I am a person of value"

---

"I am a likeable person"

---

"It's okay to take care of myself"

---

"I am important"

---

"I am just as important as the next person"

---

"I am worthy of being loved"

---

"I am okay"

---



## APPENDIX V

### Risk Screening

- A. Express concern regarding some of the responses. Let the participant know you just want to speak briefly to see how they are doing. Briefly establish some rapport.
- B. Assess risk for risk factors:
  - i. Recent loss or frustration/failure
  - ii. Mood state or current distress (depression, anxiety, agitation); have them rate depressed mood on a scale from 0-10.
  - iii. Assess degree of hopelessness
  - iv. Review BSS for history of suicide attempts and level of desire to die
- C. Assess suicide and NSSI factors (PIMP)
  - i. **Plan:** "Have you thought about what you might do (to self-injure, end your life)?"
  - ii. **Intent:** "How upset would you say you are right now?"  
"How strong is your desire to hurt yourself right now?"
  - iii. **Means:** "Do you have what you would need to \_\_\_\_\_ (plan)?"  
"Are you thinking about how to get what you need?"
  - iv. **Past:** "Have you attempted suicide/self-injured in the past?"
    - a. "When was that?" (if within past month consider HIGH RISK.
- D. Assess resources
  - i. Treatment: "Are you seeing anyone for treatment or therapy?"
    - a. If yes, "Do they know how you've been feeling?"
  - ii. Supports: "Do you live with anyone?"  
"What are you doing next?"  
"Is there someone you can go hang out with?"
  - iii. RFL: "What keeps you going right now?"
- E. Determine Level of Risk and Required Action
  - i. LOW: No past attempt or recent/current SIB, low ideation w/o plan.
    - 1. **Required Action:** validate participant's feelings and provide referral/recommendation for therapy
  - ii. MODERATE: Past attempt OR recent/current SIB, low ideation w/o plan

1. **Required Action:** help participant articulate a brief safety plan (i.e., what to do if thoughts/urges increase, distraction, call friends); If the client is unable to identify a plan to remain safe, contact the UND crisis response team.
- iii. HIGH: Recent attempt, current suicidal or SIB ideation w/ plan and no intent or access to lethal or injurious method
    1. **Required Action:** encourage participant to immediately contact support system via telephone while you're in the room; request that another RA contact Dr. Muehlenkamp while participant does this
- iv. IMMEDIATE: Current suicidal or SIB ideation, access to method, some intent
    1. **Required Action:** Call/find/track down Dr. Muehlenkamp
      - a. ask participant to remain in lab, send another RA to get Dr. Muehlenkamp
      - b. help participant contact support system to inform of risk; enlist help of support system in getting participant to a clinician
      - c. DO NOT let participant leave lab alone; have friend, family member meet them OR walk participant to counseling center or PSC.

APPENDIX VI

**Short Health History Form**

At what time did you last eat/drink? \_\_\_\_\_ a.m./p.m.

What did you eat/drink? \_\_\_\_\_

Have you consumed any alcohol and/or caffeine within the last 24 hours?      Yes    No

If yes, please list below day and time, what was consumed, and how much:

\_\_\_\_\_

Have you engaged in any physical activity/exercise that made your heart beat faster and/or your breathing rate increase for 20 minutes or more within the last 24 hours?      Yes    No

If yes, please list below day and time, what physical activity you engaged in, and duration:

\_\_\_\_\_

Do you smoke cigarettes or regularly use other tobacco/nicotine products?      Yes  
No

If yes, at what time did you last smoke or use another tobacco product? \_\_\_\_\_ a.m./p.m.

Have you experienced any illness within the last 48 hours?      Yes  
No

If yes, what were your symptoms? \_\_\_\_\_

Please list any prescription medications or over-the-counter medications you routinely take or have taken in the last 5 days (including birth control, cold or allergy medication, migraine/headache medications, antibiotics, etc.).

\_\_\_\_\_

Have you been diagnosed with any of the following within the past month (please check all that apply)?

- Major Depressive Disorder       Bipolar Disorder     Cyclothymia  
 Dysthymia  
 Generalized Anxiety Disorder     Obsessive-Compulsive Disorder       Panic Disorder  
 Posttraumatic Stress Disorder

Have you been diagnosed with *or* received treatment for any of the following in your lifetime (please check all that apply)?

Anorexia Nervosa     Bulimia Nervosa     Binge Eating Disorder

Females Only:

Are you currently taking birth control?

Yes    No

Are you currently pregnant?

Yes    No

## APPENDIX VII

### Conversation Task Topics

#### **The best ways that people meet new people and new friends on campus**

1. How do students at UND meet new people on campus and make new friends?
2. What are some of the ways that you have met new people on campus and/or made new friends?
3. What is the best way to meet new people and make new friends at UND?

#### **The best places in town to spend free time**

1. What are some places in town to spend leisure/free time?
2. What are the best places in town to spend leisure/free time?
3. What is the best place in town to spend leisure/free time?

#### **The most interesting classes available on campus**

1. What are some interesting classes that are available to students at UND?
2. What classes at UND do most students seem to like more than others?
3. What is the most interesting class that any student can take here at UND?

APPENDIX VIII

**Experiment Selection Form**

In the spaces below, please list the two individuals that you would most like to work with during the second part of the experiment. Remember that these are not *rankings* (e.g., "I want to work with Sally most, so I will list her first!"), so it does not matter who you list first or second.

Person 1. \_\_\_\_\_

Person 2. \_\_\_\_\_

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