ABSTRACT

INTEROCEPTIVE DEFICITS, NON-SUICIDAL SELF-INJURY, AND THE ACQUIRED CAPABILITY FOR SUICIDE AMONG WOMEN WITH EATING DISORDERS

by Dorian R. Dodd

The present study tested the hypothesis that among women with eating disorders (EDs), who are known to have an elevated risk for death by suicide, non-suicidal self-injury (NSSI) and ED behaviors would mediate the relationship between poor interoception and the two components of the acquired capability for suicide (ACS). Adult women with EDs (N = 98) provided self-report data regarding interoceptive deficits (ID), NSSI, ED behaviors, and the two components of ACS: fearlessness about death and pain tolerance. ID significantly predicted NSSI but not ED behaviors. ID was a strong predictor of fearlessness about death, while NSSI was a strong predictor of pain tolerance. The indirect effect of ID on pain tolerance, through NSSI was significant. No ED behaviors significantly predicted either component of ACS. The present results highlight the associations between interoceptive deficits, NSSI and the acquired capability for suicide, among women with EDs.

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Introduction

Eating disorders are serious mental illnesses which can cause significant distress and impairment (Hudson, Hiripi, Pope, & Kessler, 2007; Klump, Bulik, Kaye, Treasure, & Tyson, 2009), as well as severe adverse health outcomes (Dickstein, Franco, Rome, & Auron, 2014; Treasure, Claudino, & Zucker, 2010). Of even greater concern, however, is the fact that eating disorders can ultimately have deadly consequences – having any eating disorder diagnosis is associated with elevated all-cause mortality risk (Franko et al., 2013; Smink, van Hoeken, & Hoek, 2012). While medical complications of eating disorders do contribute to the increased mortality risk in this population (Crow et al., 2009), this elevated risk is also partially explained by another factor: the significantly increased risk for death by suicide found among individuals with eating disorders (Arcelus, Mitchell, Wales, & Nielsen, 2011; Bodell, Joiner, & Keel, 2013; Crow et al., 2009; Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). Well-informed prevention and intervention efforts designed to reduce death by suicide among individuals with eating disorders could have invaluable, life-saving benefits; however, accurately understanding why individuals in this population die by suicide at elevated rates is an essential first step in the development of effective interventions. The Interpersonal Psychological Theory of Suicide (IPTS; Joiner, 2005; Van Orden et al., 2010) provides a cogent theoretical explanation for why people die by suicide. The IPTS suggests that three factors must be present for a fatal or near fatal suicide attempt to occur: low belongingness, high perceived burdensomeness, and an acquired capability to enact lethal self-harm (described in greater detail below). The overarching aim of the present analyses is to understand how the capability to enact lethal self-harm is acquired by individuals with eating disorders.

Eating Disorders

According to the Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5; American Psychiatric Association, 2013) anorexia nervosa (AN) is characterized by dietary restraint, low body weight, and body image disturbances. Bulimia nervosa (BN) is characterized by recurrent episodes of both binge eating (i.e., excessive eating accompanied by a feeling of loss of control over eating) and purging (i.e., inappropriate compensatory behaviors to prevent weight gain – such as self-induced vomiting, over-exercise, laxative use), as well as self-evaluation that is overly influenced by body shape and weight. Unlike those with AN, individuals with BN are not underweight, but instead tend to be normal weight or overweight.

Binge eating disorder (BED) is a newly recognized eating disorder involving recurrent episodes of binge eating without any compensatory behaviors. Individuals with serious eating disturbances who do not meet all diagnostic criteria for AN, BN, or BED are given a diagnosis of other specified or unspecified feeding or eating disorder (OSFED or UFED). The diagnoses of OSFED and UFED have replaced what in previous versions of the DSM (e.g., DSM-IV-TR; American Psychiatric Association, 2000) was known as eating disorder not otherwise specified (EDNOS; which included all diagnosable eating pathology other than AN and BN, including BED). Eating disorders are relatively rare, with lifetime prevalence rates typically in the ranges of 0.5% - 1.0% for AN, 1% - 3% for BN, and 2% to 3.5% for BED (American Psychiatric Association, 2000, 2013; Hudson et al., 2007; Smink et al., 2012). All eating disorders occur more frequently among females than among males: AN and BN occur three to ten times more frequently among females than among males, while BED occurs approximately one and a half to six times more frequently among females than among males (American Psychiatric Association, 2000, 2013; Hudson et al., 2007; Smink et al., 2012).

Acquired Capability for Suicide

As previously mentioned, eating disorders are associated with a significantly elevated risk for death by suicide (Arcelus et al., 2011; Bodell et al., 2013; Crow et al., 2009; Preti et al., 2011). Persons with AN, BN, or EDNOS are as much as 31 times, 7.5 times, and 3.9 times more likely, respectively, to die by suicide than someone without these disorders (Crow et al., 2009; Preti et al., 2011). The IPTS proposes that a combination of low belongingness (i.e., loneliness and an absence of reciprocal care) and high perceived burdensomeness (i.e., feelings of liability and self-hate) will lead to a desire for suicide. However, in order for a fatal or near fatal suicide attempt to occur, a third factor must also be present: the acquired capability for suicide (ACS). In other words, dying by suicide requires a desire for suicide (i.e., low belongingness and high perceived burdensomeness) as well as an *ability* to die by suicide (i.e., ACS). Researchers have found initial support for the two main predictions of the IPTS: first that the interaction of perceived burdensomeness and thwarted belongingness predicts suicidal desire, and secondly that the interaction between perceived burdensomeness, thwarted belongingness, and ACS predicts fatal or near fatal suicide attempts (e.g., Joiner et al., 2009; Van Orden, Witte, Gordon, Bender, & Joiner, 2008).

The acquired capability for suicide (ACS) consists of a lowered fear of death as well as

increased pain tolerance. According to the IPTS (Joiner, 2005; Van Orden et al., 2010), humans should be biologically and evolutionarily predisposed to fear threats to their survival. Thus, individuals who desire to die by suicide would first need to overcome this fear. Indeed, fear of suicide was one reason individuals gave for why they had not engaged in suicidal behavior (Linehan, Goodstein, Nielsen, & Chiles, 1983), and in another study fear of suicide was one factor distinguishing depressed individuals who had not attempted suicide from depressed individuals who had attempted suicide (Malone et al., 2000). The second component of ACS is increased pain tolerance. In addition to being frightening, death by suicide is expected to involve some degree of physical pain. Thus, increased physiological habituation to sensations of pain, and expectations that the pain involved in dying by suicide will be tolerable, should facilitate engagement in suicidal behavior. ACS is theorized to increase over time as a result of experiencing painful and/or provocative events (e.g., combat exposure, previous suicide attempts, childhood maltreatment), which through opponent processes (Solomon & Corbit, 1974), provide an opportunity to habituate to pain and fear of death. Specifically, when applied to the construct of ACS (Joiner, 2005; Van Orden et al., 2010), opponent process theory (Solomon & Corbit, 1974) predicts that a painful and provocative event will elicit a primary response of pain and/or fear, which will be followed by an oppositely valenced response (i.e., opponent process), such as exhilaration, relief, or analgesia. With repetition, the opponent processes (in this case, relief, analgesia, or exhilaration) is thought to strengthen, while the primary process (pain and/or fear) remains the same (Solomon & Corbit, 1974), or weakens (Van Orden et al., 2010), making the valence of the total response (primary + opponent) more positive overall. Thus, repeated experiences with painful and provocative events should lead to greater levels of fearlessness about death and increased pain tolerance (i.e., ACS). Cumulatively, the results of several initial studies (e.g., Bryan, Cukrowicz, West, & Morrow, 2010; Smith, Cukrowicz, Poindexter, Hobson, & Cohen, 2010; Van Orden et al., 2008) support the idea that through painful and provocative events, the capability to enact lethal self-harm is acquired.

In addition to exposure to painful and provocative events, levels of ACS may also be influenced by other factors. For example a study conducted with male twins found that ACS was influenced in part by genetic and trait factors, although specific individual difference factors were not examined (Smith et al., 2012). In one study, men demonstrated greater levels of both components of ACS (fearlessness about death and pain tolerance) than women did, but the

relationship between gender and ACS was indirect: stoicism accounted for the relationship between gender and pain tolerance, while sensation seeking accounted for the relationship between gender and fearlessness about death (Witte, Gordon, Smith, & Van Orden, 2012). Thus, personality traits such as stoicism and sensation seeking seem to influence ACS. While these results show that some individual trait differences are related to ACS, the literature on this subject is emergent, and many potentially relevant individual difference factors have not yet been studied. Therefore, factors with theoretical potential to influence the development of ACS should continue to be tested; the present project will identify and test one such factor – interoception, which is a component of body regard.

Interoceptive Deficits

Body regard (Muehlenkamp, 2012) broadly refers to the relationship an individual has with his or her own body – i.e., how the body is perceived, experienced, and cared for, and the attitudes a person holds toward his or her own body. In the context of self-harming behaviors, it is theorized that how one experiences the body may influence how she treats it; an individual with poor body regard may find it easier to engage in behaviors that harm the body either directly (e.g., non-suicidal self-injury; NSSI) or indirectly (e.g., eating disorder behaviors). One aspect of body regard is interoception (Brausch & Muehlenkamp, 2014; Muehlenkamp, 2012). Interoception refers to an individual's awareness of his or her own physiological functions, and ability to sense internal cues and sensations (e.g., the ability to sense one's heartbeat, to accurately distinguish between emotions, to identify hunger, or to experience emotions and sensations without inappropriate fear or preoccupation). In other words, interoception affects how individuals experience and treat their bodies. Given that ACS also concerns the treatment of the body (specifically the acquisition of the ability to enact lethal self-harm on the body) interoception is of theoretical relevance to the construct of ACS. Individuals with eating disorders typically have greater deficits in interoception (i.e., interoceptive deficits) than healthy controls (Fassino, Pierò, Gramaglia, & Abbate-Daga, 2004; Garner, 2004; Pollatos et al., 2008), and thus if interoceptive deficits influence ACS, this relationship could provide one route by which ACS develops among those with eating disorders. To understand how interoceptive deficits may influence ACS, consider an individual who has interoceptive deficits, and is thus relatively disconnected from her physical body. This disconnection from her own internal sensations may make it easier for her to view her body as an object, rather than a connected piece

of herself, and/or it may make her less aware of physical sensations of discomfort or mild pain. This, in the context of other risk factors (e.g., body dissatisfaction, negative affect), may then facilitate engagement in self-harming behaviors such as NSSI and eating disorder behaviors, which in turn could be considered painful and provocative events likely to increase ACS.

The Effect of Interoceptive Deficits on ACS, through Eating Disorder Behaviors

In support of the idea that interoceptive deficits facilitate engagement in self-harming behaviors (e.g., eating disorders, NSSI), interoceptive deficits have been shown to be associated with eating disorders (Fassino et al., 2004; Garner, 2004; Pollatos et al., 2008), and among individuals with eating disorders, co-occurring NSSI is associated with even greater interoceptive deficits (Favaro & Santonastaso, 1998; Muehlenkamp, Peat, Claes, & Smits, 2012). Regarding the causal direction of this association, evidence suggests that it is interoceptive deficits which facilitate eating disorder behaviors rather than the reverse. For example, among adolescent girls (N = 852) participating in a 3-year prospective study, interoceptive deficits at Year 2 predicted disordered eating at Year 3 (Leon, Fulkerson, Perry, & Early-Zald, 1995). Similarly, among patients with AN, interoceptive deficits have been found to predict greater severity of symptoms five to ten years later (Bizeul, Sadowsky, & Rigaud, 2001).

In addition to evidence suggesting that interoceptive deficits facilitate engagement in eating disorder behaviors (Bizeul et al., 2001; Leon et al., 1995), several recent lines of research suggest that eating disorder behaviors are positively related to ACS. Recall that ACS is believed to increase through experiencing painful and provocative events; eating disorder behaviors intuitively could be considered painful and provocative. For example, imagine the dull, aching pain that might be experienced by someone who is habitually starving, or the sharp pain of injury that a person driven to over-exercise may experience. Binge eating, and purging behaviors (i.e., self-induced vomiting, and misusing laxatives or diuretics) also seem likely to involve at least some degree of provocation or physical pain. Consequently, according to the IPTS, eating disorder behaviors may be one route by which ACS increases. Selby and colleagues (2010) examined suicidality among individuals with AN, and found initial support for a relationship between eating disorders and ACS. Among those with the restricting subtype of AN, habitual starvation was positively related to suicidal behavior, and among those with the binge-purge subtype of AN, a "provocative behaviors" latent variable which reflected both eating disorder behaviors (self-induced vomiting, fasting, excessive exercise, misuse of laxatives) and non-

eating disordered behaviors (e.g., NSSI, disinhibited or reckless sexual activity) was positively related to suicidal behavior. Additionally, Smith and colleagues (2013) found that in a sample of individuals with BN, over-exercise significantly predicted suicidal gestures and attempts. Although, the two studies just described (Selby et al., 2010; Smith et al., 2013) provide support for a relationship between eating disorder behaviors and suicidal behaviors, it is important to note that neither directly examined ACS among individuals with eating disorders – instead, measures of suicidal behavior were used. Suicidal behavior may be a reasonable proxy measure for ACS (Joiner et al., 2009): if someone is engaging in suicidal behavior it should, theoretically, mean that he or she has acquired the capability to do so. However, empirical support is lacking for a direct relationship between eating disorder behaviors and ACS specifically among individuals with eating disorders. Importantly, initial analyses conducted with the present data (Witte et al., unpublished) do show a significant relationship between dietary restraint and both components of ACS: increased pain tolerance and fearlessness about death; although the relationship between other eating disorder behaviors and ACS among those with eating disorders has yet to be tested.

Non-Suicidal Self-Injury

NSSI, although not a direct symptom of eating disorders, is a common phenomenon among eating disorder patients, and thus may be an additional important route by which ACS develops for these individuals. Svirko and Hawton (2007) suggest that prevalence rates of NSSI among eating disorder patients range from 25.4% to 55.2%, although rates as high as 72% have been reported (Favaro & Santonastaso, 1998). The high rates of co-occurrence between NSSI and eating disorders point to phenomenological similarities between the two, and their co-occurrence may be driven by common proximal and distal risk factors (Claes & Muehlenkamp, 2014). For example, NSSI and eating disorders are both associated with a history of trauma, as well as obsessive-compulsive and perfectionistic tendencies (Bardone-Cone et al., 2007; Glassman, Weierich, Hooley, Deliberto, & Nock, 2007; Hoff & Muehlenkamp, 2009; Johnson, Cohen, Kasen, & Brook, 2002). Furthermore, acts of NSSI and eating disorders are often preceded by strong negative affect, and engaging in these acts leads to a decrease in the intensity of negative emotions (Crosby et al., 2009; Muehlenkamp et al., 2009). Some have argued that NSSI and eating disorder behaviors can all be considered forms of self-harm (Claes & Muehlenkamp, 2014; Favaro & Santonastaso, 1998); however, NSSI is considered a direct form

of self-harm, while eating disorder behaviors are considered an indirect form of self-harm (Hooley & St. Germain, 2014; St Germain & Hooley, 2012, 2013). Indirect forms of self-harm (e.g., eating disorders, substance use, unsafe or risky sexual behavior) are repetitive, and consist of behaviors that could lead to negative physical consequences over time and with repetition. Direct forms of self-harm (e.g., NSSI, suicidal behavior) cause immediate pain and/or injury, and are characterized by the immediacy and directness of the act (Brausch & Muehlenkamp, 2014; Claes & Vandereycken, 2007). Given the high rates of co-occurring NSSI among individuals with eating disorders, as well as the etiological and functional overlap between NSSI and eating disorder behaviors, NSSI is valuable to consider when examining maladaptive behaviors among eating disorder patients.

Episodes of NSSI are specifically hypothesized to increase ACS (Joiner, 2005), and studies have shown a significant association between self-injury and suicidal thoughts and behaviors (e.g. Andover & Gibb, 2010; Nock, Joiner, Gordon, Lloyd-Richardson, & Prinstein, 2006; Whitlock et al., 2013). However, it is important to recognize that these studies did not assess ACS directly either. In a more explicit test of the relationship between self-harm and ACS, St. Germain and Hooley (2013) found an association between self-harm and the pain tolerance component of ACS: individuals who engaged in either NSSI or indirect self-harm (in the form of eating disorder behaviors, or problematic drug or alcohol use) had a significantly higher threshold for pressure pain and were able to endure pressure pain significantly longer than a control group. However, St. Germain and Hooley (2013) did not examine the effects of NSSI on ACS among individuals with eating disorders, and did not examine the effects of self-harm on the second component of ACS – fearlessness about death. Although recent work provides some support for the idea that individuals with eating disorders develop ACS at least partially through NSSI and eating disorder behaviors, direct tests of this relationship are lacking. Aside from dietary restriction (Witte et al., unpublished), the effects of self-harm behaviors prevalent among individuals with eating disorders (e.g., self-induced vomiting, binging, over-exercise, NSSI) on both components of ACS (i.e., pain tolerance and fearlessness about death) have not yet been directly examined among individuals with eating disorders.

The Present Study

The present study tests the hypothesis that among individuals with eating disorders, interoceptive deficits facilitate engagement in self-harming behaviors characteristic of eating

disorders (i.e., dietary restriction, self-induced vomiting, over-exercise, and binging) or commonly associated with eating disorders (i.e., NSSI), and that in turn those behaviors predict levels of ACS. Using a parallel mediation model, I will also test the strength and direction of the direct effect of interoceptive deficits on ACS, and compare the relative strengths of the various indirect paths from interoceptive deficits to ACS through dietary restriction, self-induced vomiting, over-exercise, binging, and NSSI. Given that NSSI is a direct form of self-harm, and therefore creates an immediate, deliberate, and salient link between the behavior and the resulting pain and bodily injury, I hypothesize that NSSI will be a more robust predictor of ACS than eating disorder behaviors. Further, I predict that active eating disorder behaviors (i.e., self-induced vomiting, over-exercise), which also occur in discrete episodes and create an immediate link between the behavior and a physical outcome, will be more strongly predictive of ACS than passive eating disorder behaviors with diffuse outcomes, such as dietary restriction. I will also test whether eating disorder behaviors account for unique variance in ACS, above and beyond NSSI, which, given the hypothesized strength of the relationship between NSSI and ACS will provide a robust test of the effects of eating disorder behaviors on ACS. Through the present analyses I hope to 1) confirm theorized predictions of the IPTS by testing the relationship between specific, relevant predictors (e.g., over-exercise, binging, self-induced vomiting, NSSI) and ACS among those with eating disorders, as well as to examine the relative strength of these predictors and 2) examine how a previously untested and conceptually relevant factor, interoceptive deficits, may contribute to ACS among individuals with eating disorders.

Method

The present study is a secondary data analysis, using data from a larger study. The procedures of the parent study, as well as the procedures of the current analyses, were approved by the relevant Institutional Review Boards. Details of the parent study can be found elsewhere (Witte et al., unpublished); the methods presented below will focus only on those procedures relevant to the present analyses.

Participants

Participants (N = 98) were adult females admitted to an eating disorder treatment center. A total of 100 women originally enrolled in the study; however, two did not complete measures of interoceptive deficits or ACS and were therefore excluded from the present analyses. At the time of admission, 82% of the participants (n = 80) were seeking residential treatment, while the

remainder of participants were seeking treatment in the facility's partial hospitalization program. The sample was predominantly White (*n* = 94; 96%), followed by Black or African American (*n* = 2; 2%), Native Hawaiian or Other Pacific Islander (*n* = 1; 1%), and American Indian/Alaska Native (*n* = 1; 1%). The participants ranged in age from 18 years old to 58 years old (*M* = 26.9 years; *SD* = 7.9 years). Roughly one-third of the sample (*n* = 33; 34%) met DSM-5 (APA, 2013) diagnostic criteria for AN, and roughly one-third of the sample (*n* = 30; 31%) met DSM-5 diagnostic criteria for BN. The remaining third of the sample (*n* = 35; 36%) met DSM-IV-TR (APA, 2000) criteria for EDNOS. Data for the present study were collected prior to the recognition of BED as a formal diagnostic label, and therefore data regarding diagnostic symptoms are lacking the specificity necessary to distinguish between EDNOS and BED. Demographic and eating disorder characteristics of the sample are summarized in Table 1.

Procedures

Women seeking residential treatment or partial hospitalization at an eating disorder treatment facility (which serves only females 18 years old or older) were recruited through an announcement made during their intake session. Interested patients were then asked to read an informed consent form and were given an opportunity to ask questions. Patients who wished to participate indicated their consent by signing the informed consent form; patients who were 18 years old also had to obtain signed permission/assent from a parent or guardian. Some data used for the present study (i.e., diagnostic assessment interview data and the Eating Disorder Inventory) were collected as part of the standard intake procedures for the treatment facility; for those data de-identified versions of the forms were created and put in the participant's research file. All other data in the present study were collected via a battery of self-report questionnaires, within 4 days of admission. The questionnaires were administered on a laptop computer using Qualtrics, an online survey service. Participation was voluntary and unpaid.

Measures

As part of the treatment center's intake procedures, all participants' height and weight were measured. Body mass index (BMI) was calculated for each participant using the following formula: $\text{BMI} = 703 * (Weight\ (lbs)/Height\ (in)^2)$.

The Eating Disorder Inventory-3 (EDI-3; Garner, 2004) is a 91-item self-report questionnaire which assesses behavioral and psychological traits common among individuals with eating disorders. The EDI-3 has strong psychometric properties (Clausen, Rosenvinge,

Friborg, & Rokkedal, 2011) and is appropriate for use among clinical and nonclinical samples, both within the United States and internationally (Cumella, 2006). The EDI-3contains 12 subscales: drive for thinness, bulimia, body dissatisfaction, low self-esteem, personal alienation, interpersonal insecurity, interpersonal alienation, interoceptive deficits, emotional dysregulation, perfectionism, asceticism, and maturity fears. The interoceptive deficits subscale consists of nine items assessing for deficits in the ability to accurately recognize and respond to emotions and physical sensations such as hunger and satiety. Sample items include "I get frightened when my feelings are too strong"; "I get confused as to whether or not I am hungry"; "When I am upset, I don't know if I am sad, frightened, or angry" and "I don't know what's going on inside me." Participants indicate the extent to which each statement is true for themselves, using a scale from 1 (*never*) to 6 (*always*). Higher scores indicate greater interoceptive deficits. Cronbach's alpha[1] for the EDI interoceptive deficits subscale in the present sample was 0.88. The EDI-3 interoceptive deficits subscale will provide the measure of interoceptive deficits for the current study.

The Functional Assessment of Self-Mutilation (FASM; Lloyd, Kelley, & Hope, 1997) is a self-report questionnaire which asks about the frequency and function of NSSI behaviors during the previous year. Eleven specific methods of NSSI are assessed (e.g., "cut or carved on your skin"; "hit yourself on purpose"), and respondents are also asked to specify any other methods of NSSI they have used. For each method of NSSI, respondents indicate whether and how many times they have engaged in that behavior during the past year, and whether medical treatment was received as a result of that behavior. Additionally, the FASM includes six questions pertaining to self-harm experiences more generally (e.g., whether the respondent performed any self-harm behaviors while taking drugs or alcohol, the level of pain experienced during self-harm, and the age when the respondent first engaged in self-harm). The final section of the FASM contains 22 items which assess a respondent's reasons for self-harming (e.g., "to avoid school, work or other activities"; "to relieve feeling 'numb' or empty"). The frequency data from the first section of the FASM will be used in the present analyses as a measure of engagement in NSSI. Cronbach's alpha for the items assessing frequency of various types of self-harm was 0.68 in the present sample.

[1] All Cronbach's alpha scores were calculated using original data, prior to multiple imputation

The Eating Behaviors Painful and Provocative Events Scale (EPPES) is a 40-item self-report questionnaire which asks participants how many times they have had certain experiences related to eating disorders that are thought to be painful or provocative. The EPPES was created by the research group of Dr. Thomas Joiner, developer of the IPTS, and was designed specifically to assess eating disorder experiences that may relate to ACS. For example, the EPPES asks about frequency of experiences such as taking laxatives, self-inducing vomiting, continuing to eat even after feeling painfully full, having esophageal tears or gastric ruptures, or going 24 hours without eating. Consisting of two sections, the EPPES asks respondent's about the frequency of 20 specific experiences over 1) the respondent's lifetime and 2) the past month. For lifetime questions, participants respond using a 10 point scale (coded 0 to 9) which corresponds to the following response options: 0 times, 1 time, 2-3 times, 4-10 times, 11-20 times, 21-40 times, 41-60 times, 61-80 times, 81-100 times, and 101 or more times. Data from the lifetime section of the EPPES will be used in the present analyses to measure engagement in painful and provocative eating disorder behaviors. For each category of eating disorder behaviors relevant to the present study (i.e., purging, over-exercise, binging, and restriction) the EPPES contains at least two relevant items. Therefore, for each eating disorder variable, a composite score will be calculated based on all relevant items from the EPPES. For example, the composite score for over-exercise will be based on the items "…exercised even though you were physically ill (e.g., had the flu)," "…kept on exercising even though you were in a lot of physical pain," and "…exercised hard (i.e., for 2 or more hours)." Cronbach's alphas were as follows: 0.77 for the two restraint items; 0.84 for the three over-exercise items; 0.72 for the six vomiting items; 0.90 for the two binge eating items.

The original version of the *Acquired Capability for Suicide Scale* (ACSS; Ribeiro et al., 2014; Van Orden et al., 2008) is a 20-item self-report questionnaire, also developed by Dr. Thomas Joiner's research group. The ACSS was designed to measure levels of ACS, as the construct was initially conceptualized (i.e., consisting of fearlessness about death and pain tolerance, and increasing through painful and provocative events; (Joiner, 2005). The ACSS has been shown to have good convergent and discriminant validity (Van Orden et al., 2008). For each item on the ACSS, participants are asked to indicate the extent to which they think the statement describes them, using a scale from 1 ("*not at all like me")* to 5 ("*very much like me").* On the original ACSS, seven items assess fearlessness about death (FAD), one item assesses pain

tolerance, and the remaining items ask about painful and provocative events (Ribeiro et al., 2014). However, painful and provocative events are thought to be causally related to ACS, while FAD and pain tolerance are the underlying component parts that make up ACS (Van Orden et al., 2010). Therefore, the outcome measures for the present analyses will be the two components of ACS (i.e., FAD and pain tolerance). The 7-item ACSS Fearlessness about Death subscale (ACSS-FAD) will provide the measure of FAD in the present analyses. The ACSS-FAD has strong psychometric properties (Ribeiro et al., 2014); sample items include "the fact that I am going to die does not affect me"; "I am not at all afraid to die"; and "the prospect of my own death arouses anxiety in me" (reverse scored). Higher scores on the ACSS-FAD indicate greater fearlessness about death. In the present sample, the Cronbach's alpha for the ACSS-FAD was 0.88. The single pain tolerance item on the ACSS ("I can tolerate a lot more pain than most people") will be used in the present study for the second outcome variable of interest: pain tolerance. Higher scores on this item will indicate greater subjective pain tolerance. This is similar to previously used procedures for measuring pain tolerance (e.g., Zuromski, Davis, Witte, Weathers, & Blevins, 2014), and similar self-report items have loaded onto a factor indexing pain and discomfort tolerance (Schmidt, 2006).

Results

Power

The present analyses were conducted with a sample size of $N = 98$. A power analysis conducted with G*Power 3.1 (Faul, Erdfelder, Buchner, & Lang, 2009) revealed that for the following regression analyses this sample size achieves 83% power to detect a medium effect size, $f^2 = 0.15$.

Data Preparation

Data normality.

Skewness and kurtosis were examined for all variables of interest. Skewness and kurtosis were within acceptable ranges for scores on interoceptive deficits, restraint, over-exercise, vomiting, binge eating, fearlessness about death, and pain tolerance (all skewness and kurtosis values < |1|). Age was non-normally distributed (skewness = 1.43, kurtosis = 2.18). NSSI scores (derived from the FASM) were non-normally distributed (skewness = 4.41, kurtosis = 24.81) and were thus transformed using the following formula: Log10(FASM NSSI score + 1).

Transformed NSSI scores[2] were approximately normally distributed (skewness = -0.07; kurtosis = -1.13).

Missing data.

Little's MCAR statistic was calculated for all variables used in the present analyses, and revealed that missing data were consistent with a pattern of missing completely at random χ^2 = 8.73 (*df* = 24, *p* = .99). The percentages of missing data were also examined. Interoceptive deficit scores from the EDI were missing in 2% of the sample (*n* = 2); engagement in NSSI scores, derived from the FASM questionnaire, were missing for 6.1% of the sample (*n* = 6); fearlessness about death subscale scores, and self-report pain tolerance item scores were not missing from any participants. Data derived from the EPPES (i.e., average lifetime ratings of engagement in restraint, over-exercise, vomiting, and binge eating) were each missing for 25.5% of the sample (*n* = 25). These data were missing consecutively from the first 25 participants enrolled in the study, which was a result of changes to the study procedures after enrollment of and data collection from the first 25 participants (at which point this version of the EPPES was added to the protocol). Missing data were handled using multiple imputation procedures (*m* = 40) in SPSS version 21.0 (IBM Corp, 2012). Values were imputed following calculation of subscale scores, and the imputation model included subscale scores rather than individual items; NSSI data were transformed prior to imputation. Pooled estimates are reported when available. For tests on which pooled estimates are not available, scores for each variable of interest were averaged across the 40 imputed datasets, and analyses were conducted on these averaged data. Descriptive statistics for all study variables, in all forms, can be found in Table 2.

Analyses

All of the following correlation and regression analyses were conducted using SPSS, Version 21 (IBM Corp., 2012), and all mediation analyses were conducted using the PROCESS macro for SPSS (Hayes, 2012).

A series of bivariate correlations were calculated in order to examine the relationships between all study variables (Table 3). Given that lifetime engagement in ED behaviors, as well as the acquired capability for suicide (Van Orden et al., 2010) are both theorized to increase over

[2] All analyses were also conducted using the original, pre-transformed NSSI scores as well. Data are not reported for these analyses, as the pattern of results remained consistent. The most notable difference was that the complex contrast of the indirect effect of interoceptive deficits on pain tolerance through NSSI, versus through the eating disorder behaviors, failed to reach significance when using the original NSSI data.

time, combined with the large range of participants' ages (range = 40 years), correlations between age and all study variables were also calculated. Age was found to be significantly negatively correlated with interoceptive deficits (r = -.21, p =.04). Therefore, all subsequent regression analyses control for the effects of participant age.

Given the significant correlations found between several predictor variables, multicollinearity statistics were computed and examined for predictors in all of the following regression analyses. Variance inflation factors (VIFs) and Tolerance values were all within acceptable ranges (all VIFs > 0.4; all Tolerance values < 2), suggesting that multicollinearity is not a problem in the following analyses.

Hypothesis 1: Interoceptive deficits predicting engagement in NSSI and eating disorder behaviors.

I first tested the hypothesis that interoceptive deficits would predict greater engagement in NSSI and eating disorder behaviors. A series of hierarchical linear regression analyses which controlled for age (entered in the first step) tested whether interoceptive deficits (entered in the second step) predicted engagement in each of the 5 self-harming behaviors: NSSI, restriction, over-exercise, binging, and self-induced vomiting. In support of this hypothesis, when controlling for age, interoceptive deficits significantly predicted engagement in NSSI (B = 0.04, $p < .01$, $R^2\Delta = .15$)[3]. However, contrary to the hypothesis, interoceptive deficits did not significantly predict restriction, over-exercise, binging, or self-induced vomiting ($.10 \leq$ all p values $\geq .46$; see Table 4).

Hypothesis 2: NSSI and eating disorder behaviors predicting levels of FAD and pain tolerance.

I next tested the hypothesis that NSSI and eating disorder behaviors would significantly predict FAD and pain tolerance, and further, that eating disorder behaviors would predict FAD and pain tolerance above and beyond NSSI. In order to test this, two hierarchical multiple regression analyses were conducted, with age and the 5 self-harming behaviors entered in the prediction of FAD and of pain tolerance. For these analyses age was entered as the first step, NSSI was entered in the second step, and the eating disorder behaviors (restriction, binge eating, self-induced vomiting, and over-exercise) were entered in the third step, to determine if eating

[3] B is used throughout this paper to symbolize the unstandardized regression coefficient

disorder behaviors contribute to the prediction of ACS (as measured by FAD and pain tolerance) above and beyond NSSI. Results of these analyses are summarized in Table 5.

In step 1 of the first of these two analyses, age did not significantly predict FAD (B = 0.04, p = 0.67, R^2=0.002). In step 2, NSSI significantly predicted FAD when controlling for age (B = 0.26, p < 0.01, $R^2 \Delta$ = 0.111), as predicted. In step 3, no single eating disorder behavior significantly predicted FAD when controlling for age and NSSI however, the addition of these four eating disorder variables to the model led to a marginally significant increase in the proportion of variance accounted for by the model ($R^2 \Delta$=0.085; $F\Delta$ (4,91) = 2.42, p = 0.054). These results provide marginal support for the hypothesis that eating disorder behaviors predict FAD, above and beyond NSSI. In step 3 of the model, NSSI remained a significant predictor of FAD when controlling for age and the four eating disorder variables (B = 2.24, p < 0.01). When controlling for age, NSSI and the eating disorder behaviors accounted for 19.6% of the variance in FAD.

A model predicting self-reported pain tolerance was examined next. In the first step, age did not significantly predict pain tolerance (B = 0.01, p = 0.58, R^2 = 0.003). In step 2, NSSI significantly predicted pain tolerance when controlling for age (B = 0.54, p < 0.01, $R^2 \Delta$ = 0.191), as hypothesized. In step 3, no eating disorder behavior significantly predicted pain tolerance when controlling for age and NSSI (all p values > 0.17), nor did the addition of these four eating disorder variables to the model significantly increase the proportion of variance accounted for by the model ($R^2 \Delta$ = 0.050, $F \Delta$ (4,91) = 1.52, p = 0.20). However, NSSI remained a significant predictor of pain tolerance when controlling for age and the four eating disorder variables (B = 0.51, p < 0.01). These analyses suggest that contrary to hypotheses, eating disorder behaviors do not contribute to the prediction of pain tolerance above and beyond NSSI.

Hypothesis 3: NSSI and eating disorder behaviors mediating the relationships between interoceptive deficits and FAD, and interoceptive deficits and pain tolerance.

Next, two parallel mediation models were examined in order to 1) test the hypothesis that interoceptive deficits will predict both FAD and pain tolerance, either directly or indirectly through the effects of NSSI and eating disorder behaviors, and 2) to examine and compare the indirect effects through each mediator in a model accounting for all mediators (NSSI, binging, over-exercising, vomiting, and restraint) and the predictor (interoceptive deficits). Both parallel mediation models were tested using the PROCESS macro for SPSS (Hayes, 2013); both models

used interoceptive deficits as the independent variable, and included the following mediators: binging, purging, over-exercise, restriction, and NSSI. The first model used FAD as the dependent variable while the second model examined pain tolerance as the dependent variable. The parallel mediation model predicting FAD accounted for 21.5% of the variance in FAD (R^2 = 0.215, F (6, 91) = 4.16, p < .01). The total effect of interoceptive deficits on FAD was significant (B = 0.26, p = 0.001, 95% CI [0.11 – 0.41]), as was the direct effect of interoceptive deficits on FAD (B = 0.19, p = 0.03, 95% CI [0.03 – 0.36]). The total indirect effect was not significant, nor was the indirect effect through any specific mediator (all 95% CIs spanned 0). Specific contrasts showed that the indirect effect through NSSI was significantly greater than the indirect effect through binge eating (95% CI [0.01 – 0.19]), but no other indirect effects significantly differed. Regression coefficients for each path of the model are shown in Figure 1.

The parallel mediation model predicting pain tolerance accounted for 22.3% of the variance in pain tolerance (R^2 = 0.223, F (6, 91) = 4.36, p < .01). Regression coefficients for each path of the model are shown in Figure 2. The total effect of interoceptive deficits on pain tolerance was not significant (B = 0.02, p = 0.16, 95% CI [-0.01 – 0.04]), nor was the direct effect of interoceptive deficits on pain tolerance (B= -0.01, p = 0.63, 95% CI [-0.03 – 0.02]). However, the total indirect effect of interoceptive deficits on pain tolerance through the mediators was significant, B = 0.03, 95% CI [0.009 – 0.046]. The indirect effect of interoceptive deficits on pain tolerance through NSSI was also significant, B = 0.022, 95% CI [0.01 – 0.05], although the indirect effect of interoceptive deficits on pain tolerance through each eating disorder variable was not significant (all 95% CIs span zero). Specific contrasts revealed that the indirect effect of interoceptive deficits on pain tolerance through NSSI was significantly greater than the indirect effect of interoceptive deficits on pain tolerance through restraint (95% CI [0.01 – 0.04]), binge eating (95% CI [0.01 – 0.04]), over-exercise (95% CI [0.003 – 0.037]), and self-induced vomiting (95% CI [0.01 – 0.05]). No differences were found between the strength of the indirect effects through the eating disorder variables.

Hypothesis 4: The comparative strength of NSSI versus the eating disorder behaviors as predictors of FAD and pain tolerance.

Complex contrasts comparing the combined indirect effects of the eating disorder behaviors against the indirect effect through NSSI were examined by constructing percentile bootstrap confidence intervals for the contrast, following established guidelines using PROCESS

(Hayes, 2014). Specifically, PROCESS was used to generate a file with 10,000 bootstrap values of each regression coefficient. Next a contrast was calculated using the following formula: [(specific indirect effect of interoceptive deficits on FAD through restraint + specific indirect effect of interoceptive deficits on FAD through vomiting + specific indirect effect of interoceptive deficits on FAD through over-exercising + specific indirect effect of interoceptive deficits on FAD through binge eating) – (specific indirect effect of interoceptive deficits on FAD through NSSI)], where the specific indirect effect of interoceptive deficits on FAD through j mediator $= a_j * b_j$. The value of the contrast was calculated for each of the 10,000 sets of regression coefficient bootstrap values, and finally the values demarcating the 2.5^{th} and 97.5^{th} percentiles of the contrast variable were determined. Contrary to hypotheses, there was no significant difference between the indirect effect of interoceptive deficits on FAD through the eating disorder behaviors vs. through NSSI (95% CI [-0.14 – 0.07]). However, in line with hypotheses, the indirect effect of interoceptive deficits on pain tolerance through NSSI was significantly greater than the combined indirect effect of interoceptive deficits on pain tolerance through the four eating disorder variables, 95% CI [-0.04 – -0.002].

Hypothesis 5: The comparative strength of active versus passive eating disorder behaviors as predictors of FAD and pain tolerance.

Lastly, I hypothesized that active eating disorder behaviors (i.e., over-exercise, binge eating, vomiting) would be stronger predictors of FAD than passive eating disorder behaviors (i.e., restraint). The first test of this hypothesis examined eating disorder behaviors without accounting for interoceptive deficits or NSSI. Two regression analyses were conducted using the following predictors: age (entered in step 1), binge eating, over-exercise and vomiting (i.e., active eating disorder behaviors, entered in step 2) and restraint (i.e., passive eating disorder behavior, entered in step 3). In the model predicting FAD, age did not significantly predict FAD in step 1. In step 2, over-exercise was the only significant predictor of FAD ($B = 0.62$, $p = 0.04$), and the addition of these three active eating disorder behaviors contributed significantly to the prediction of FAD ($R^2 \Delta = .132$, $F \Delta (3,93) = 4.74$, $p < .01$). In step 3, restraint was added to the model and did not significantly add to the prediction of FAD ($R^2 \Delta < 0.001$); furthermore, when controlling for the effects of restraint in step 3, over-exercise became only a marginally significant predictor of FAD ($B = 0.62$, $p = 0.052$). See Table 6 for a summary of these results. Given that when controlling for age, the active eating disorder behaviors accounted for 13.2% of

variance in FAD, yet restraint accounted for essentially no variance in FAD above and beyond the active eating disorder behaviors and age, these results provide some support for a distinction between active vs. passive eating disorder behaviors in the prediction of FAD.

In the second model, age did not significantly predict pain tolerance in step 1. In step 2, over-exercise was the only significant predictor of pain tolerance (B = 0.10, p =.04), and the addition of all three active eating disorder behaviors contributed significantly to the prediction of pain tolerance ($R^2 \Delta$ = 0.083, $F \Delta$ (3,93) = 2.83, p = .04). Restraint was added to the model in step 3, and did not contribute significantly to the prediction of pain tolerance, above and beyond age and the active eating disorder behaviors ($R^2 \Delta$ = 0.008). When controlling for restraint in step 3, over-exercise was no longer a significant predictor of pain tolerance (see Table 6). In the prediction of pain tolerance, active eating disorder behaviors accounted for 8.3% of variance (when controlling for age), yet restraint accounted for only 0.8% of variance, above and beyond the active eating disorder behaviors and age, supporting the hypothesis that active eating disorder behaviors more strongly predict pain tolerance than the passive eating disorder behavior.

Lastly, the effects of the active eating disorder behaviors were compared to the effects of the passive eating disorder behavior in modified versions of each parallel mediation model. For these analyses, NSSI was not included as a mediator, so that the effects of the eating disorder behaviors could be compared directly, without potentially being obscured by the effect of NSSI. A complex contrast was calculated to compare the indirect effects of interoceptive deficits on FAD through over-exercise, binge-eating, and vomiting, versus through restraint, and contrary to hypotheses a percentile bootstrap confidence interval showed no significant difference between the indirect effects through active vs. passive eating disorder variables (95% CI [-.04 – .17]). Likewise, no significant difference was found between the specific indirect effects of interoceptive deficits on pain tolerance through the active eating disorder behaviors versus through the passive behavior (95% CI [-.02 – .02]). Thus, no significant differences were found between the indirect effects of interoceptive deficits through active vs. passive eating disorder behaviors on either FAD or pain tolerance.

Exploratory Analyses (as suggested by committee).

Regression analyses conducted thus far have not examined or controlled for the effects of interoceptive deficits; this variable has been examined only in the parallel mediation models. To further deconstruct the relationships between study variables, and to examine the effect of the

mediators on both FAD and pain tolerance when accounting for the independent variable (as recommended in traditional mediation analyses; Baron & Kenney 1986), two further regression analyses were conducted. In both analyses, age was entered in step 1, interoceptive deficits was entered in step 2, NSSI was entered in step 3, and eating disorder behaviors were entered in step 4.

In the prediction of FAD, age was not a significant predictor in step 1, but when controlling for age, interoceptive deficits was a significant predictor in step 2 ($B = 0.27$, $p = 0.02$, $R^2\Delta = 0.116$, $F\Delta\ (1,95) = 12.46$, $p < .01$). In step 3, both NSSI ($B = 1.84$, $p = 0.04$) and interoceptive deficits ($B = 0.20$, $p = 0.02$) significantly predicted FAD, and the addition of NSSI significantly contributed to the model ($R^2\Delta = 0.046$, $F\Delta\ (1,94) = 5.21$, $p = .03$). In the final step of the model, the eating disorder behaviors were added, and only interoceptive deficits was a significant predictor of FAD ($B = 0.22$, $p = 0.01$), although the addition of the eating disorder behaviors did significantly contribute to the model ($R^2\Delta = 0.089$, $F\Delta\ (4,90) = 2.69$, $p = .036$). See Table 7 for a summary of these analyses.

In the prediction of pain tolerance, age was not a significant predictor in step 1. In step 2, neither age nor interoceptive deficits emerged as significant predictors of pain tolerance. NSSI was added in step 3, and was found to be a significant predictor of pain tolerance when controlling for age and interoceptive deficits ($B = 0.55$, $p < .01$). The addition of NSSI to the model significantly improved the proportion of variance in pain tolerance accounted for ($R^2\Delta = 0.167$, $F\Delta\ (1,94) = 19.44$, $p < .01$). Finally, in the fourth step all eating disorder behaviors were added to the model. NSSI remained a significant predictor of pain tolerance ($B = 0.52$, $p < 0.01$), but no other predictors were significant, and the combined addition of the four eating disorder variables did not add significantly to the model ($R^2\Delta = 0.051$, $F\Delta\ (4,90) = 1.51$, $p = .21$). See Table 7 for a summary of these analyses.

Diagnostic status.

To examine the relationships of study variables to diagnostic status, a series of one-way ANOVAs were conducted.[4] No significant differences were found between diagnostic groups on interoceptive deficits, NSSI, restraint, FAD, or pain tolerance ($.08 \leq$ all p values $\leq .53$).

[4] Analyses were conducted with original, non-imputed and untransformed data (n values range 73 – 98) as well as on transformed, averaged values across multiply imputed datasets. Patterns of results were similar; only the latter are reported.

However, significant differences were found across diagnostic groups on over-exercise, vomiting, binge eating, BMI, and age. For significant omnibus ANOVAs for which Levene's test indicated homogeneity of variances (over-exercise, vomiting), Tukey's post-hoc analyses were conducted. For significant omnibus ANOVAs for which Levene's test indicated significant differences in variances (binging, BMI, age), Dunnett's T3 post-hoc analyses were conducted (Wilcox, 2013). Results of post-hoc analyses are presented in Figure 3.

Next, all regression analyses were conducted within each diagnostic group, using pooled values from the multiply imputed dataset, and results were compared; some differences were found in the relationship between study variables based on diagnosis. When controlling for age, interoceptive deficits was a significant predictor of restraint for the AN group ($p = .033$) but not for the BN or EDNOS group; interoceptive deficits significantly predicted NSSI for the BN group ($p < .001$), but not for AN or EDNOS. Interoceptive deficits did not significantly predict over-exercise, vomiting, or binge eating for any group.

Group differences across diagnoses were also found in a model predicting FAD from age, NSSI, and all ED behaviors: for the AN group, age ($p = .04$), over-exercise ($p = .01$), and vomiting ($p = .04$) were significant predictors of FAD, whereas for the BN group no variables were significant predictors of FAD, and for the EDNOS group only NSSI was a significant predictor ($p = .01$) of FAD. When predicting pain tolerance from age, NSSI, and all eating disorder behaviors, NSSI was a marginally significant predictor of pain tolerance for the AN group ($p = .07$) and BN group ($p = .08$) but was a significant predictor for the EDNOS group ($p = .01$); no other variables significantly predicted pain tolerance in this model.

When examining the prediction of FAD and pain tolerance from the eating disorder behaviors and age (i.e., *excluding* NSSI), over-exercise ($p = .01$) and vomiting ($p = .04$) were significant predictors of FAD for the AN group, but no variables significantly predicted FAD for the BN or EDNOS group. In the prediction of pain tolerance, no variables were significant for any diagnostic group.

Finally, when age, interoceptive deficits, NSSI, and all ED behaviors were entered in the prediction of FAD, several variables significantly predicted FAD for the AN group (age, $p = .02$; over-exercise, $p < .01$, and vomiting, $p = .02$), no variables significantly predicted FAD for the BN group, and only NSSI significantly predicted FAD for the EDNOS group ($p = .03$). When age, interoceptive deficits, NSSI, and all ED behaviors were entered in the prediction of pain

tolerance, NSSI trended towards significance in the prediction of pain tolerance for the AN group ($p = .09$), and was a significant predictor of pain tolerance for the EDNOS group ($p < .01$), but did not significantly predict pain tolerance for the BN group. No other variables significantly predicted pain tolerance for any of the diagnostic groups.

These results suggest that ED diagnosis may moderate some relationships between study variables; however, a full accounting of all moderation effects in the present models is beyond the scope of the present study.

Discussion

The present study was designed to test the hypotheses that among women with eating disorders, interoceptive deficits would predict engagement in eating disorder behaviors and NSSI. Further, I hypothesized that directly, or indirectly through its effects on eating disorder behaviors and NSSI, interoceptive deficits would predict greater fearlessness about death and self-reported pain tolerance, which are the two components of ACS. Lastly, I predicted that among women with eating disorders, NSSI would be a stronger predictor of FAD and pain tolerance than eating disorder behaviors would be, and that active eating disorder behaviors (i.e., self-induced vomiting, binge eating, and over-exercise) would be stronger predictors of FAD and pain tolerance than a passive eating disorder behavior (i.e., restraint).

In partial support of these hypotheses, when controlling for age, interoceptive deficits was a significant predictor of NSSI, but not of any eating disorder behaviors, and interoceptive deficits significantly predicted FAD but not self-reported pain tolerance. When controlling for age, NSSI, and eating disorder behaviors, interoceptive deficits remained a significant predictor of FAD. Additionally, when controlling for the effects of age and eating disorder behaviors, NSSI was a significant predictor of both FAD and pain tolerance; however, when accounting for the effects of interoceptive deficits in addition to age and eating disorder behaviors, NSSI significantly predicted pain tolerance, but was no longer a significant predictor of FAD. Results indicated that NSSI was a stronger predictor of both FAD and pain tolerance than any of the eating disorder behaviors were, and in a robust test of the influence of the eating disorder behaviors on FAD and pain tolerance, the combined influence of the eating disorder behaviors was found to be marginally significant in predicting FAD above and beyond age and NSSI. However, the eating disorder behaviors did not add significantly to the prediction of pain tolerance, above and beyond age and NSSI.

Parallel mediation analyses revealed that interoceptive deficits significantly predict FAD, when controlling for NSSI and eating disorder behaviors. However, the indirect effect of interoceptive deficits through NSSI and eating disorder behaviors was not significant (i.e., the effect through each individual mediator was not significant, nor was the total indirect effect through all mediators). Contrary to hypotheses, the indirect effect of interoceptive deficits on FAD through NSSI was not significantly greater than the combined indirect effect of interoceptive deficits on FAD through the eating disorder behaviors. Regarding self-reported pain tolerance, counter to hypotheses, interoceptive deficits did not significantly predict pain tolerance. Consistent with hypotheses though, NSSI emerged as a strong predictor of pain tolerance in this model; the indirect effect of interoceptive deficits on pain tolerance through NSSI was significant, as was the total indirect effect of interoceptive deficits on pain tolerance through all mediators combined. The indirect effect through any individual eating disorder behavior was not significant. Furthermore, as hypothesized the indirect effect of interoceptive deficits on pain tolerance through NSSI was significantly stronger than the indirect effect through each individual eating disorder behavior; the indirect effect through NSSI was also significantly stronger than the indirect effect through all eating disorder behaviors combined.

Only limited support was found for a distinction between passive and active eating disorder behaviors based on their ability to predict ACS. Specifically, when not accounting for interoceptive deficits or NSSI, active eating disorder behaviors significantly contributed to the prediction of both FAD and pain tolerance, above and beyond age, and over-exercise was a significant individual predictor of both FAD and pain tolerance. When adding the passive eating disorder behavior, restraint as a predictor (i.e., controlling for the effects of age and the active eating disorder behaviors), restraint did not significantly contribute to the prediction of either FAD or pain tolerance. In combination the active eating disorder behaviors seemed to account for proportionally more variance in both FAD and pain tolerance than the passive eating disorder behavior. However, no individual eating disorder behavior significantly predicted pain tolerance when controlling for the effects of age and all other eating disorder behaviors, and over-exercise was only a marginally significant predictor of FAD when controlling for age and all other eating disorder behaviors (with no other eating disorder behaviors approaching significance). Further, in a parallel mediation model with eating disorder behaviors as mediators (and without NSSI) the indirect effect of interoceptive deficits on FAD through active eating disorder behaviors was not

significantly different than the indirect effect through restraint (the passive eating disorder behavior). The same was true for the indirect effects on self-reported pain tolerance.

Individuals with eating disorders are at an elevated risk for death by suicide (Arcelus et al., 2011; Bodell et al., 2013; Crow et al., 2009; Preti et al., 2011), which through the framework of the IPTS (Joiner, 2005; Van Orden et al., 2010) would indicate an elevated risk for ACS. Thus, better understanding factors which contribute to ACS (comprised of FAD and pain tolerance) among those with eating disorders could have important treatment and suicide prevention implications for this population. Results of the present study indicate strong associations between interoceptive deficits and FAD, as well as between NSSI and pain tolerance among indviduals with eating disorders. Surprisingly, NSSI and interoceptive deficits are two risk factors neither unique to or specifically characteristic of EDs, but which are prevalent among those with eating disorders (Fassino et al., 2004; Garner, 2004; Pollatos et al., 2008; Svirko & Hawton, 2007) and appear to contribute to ACS in this population. Additionally, while in combination, eating disorder behaviors contributed to the prediction of FAD, no single eating disorder behavior emerged as a significant predictor when controlling for the effects of the others. Thus, total eating disorder pathology and key non-eating disorder variables (e.g., interoceptive deficits, NSSI) may be a better indicator for suicide risk than any specific eating disorder behavior, among those with eating disorders.

The present pattern of results suggests that risk factors commonly co-occuring with eating disorders may contribute to the acquired capability for suicidality more than eating disorder symptoms themselves. This is consistent with research by Bodell, Joiner, and Keel (2013) who found that only BN, but not AN or EDNOS, conferred additional risk for suicide above and beyond the effect of comorbid Axis I disorders. However, given that rates of suicide are elevated for those with eating disorders even in comparison to populations with other psychiatric disorders (Harris & Barraclough, 1997) it seems unlikely that co-occurring risk factors could account entirely for this elevated suicide risk. Research suggests that some eating disorder symptoms (e.g., restraint) may be related to perceived burdensomeness and thwarted belongingness (Dodd, Smith, & Bodell, 2014), so it is possible that symptoms of eating disorders elevate suicidal ideation (i.e., by increasing thwarted belongingness and perceived burdensomeness) while co-occurring factors such as NSSI, interoceptive deficits, and co-occurring disorders contribute to ACS. Additional research is needed to establish which factors,

including eating disorder symptoms as well as co-occurring risk factors, drive the elevated risk for suicide among those with eating disorders.

Interoception is a theorized key component of body regard (Brausch & Muehlenkamp, 2014; Muehlenkamp, 2012) and by definition influences how one experiences the body and it's internal sensations. The present results indicate that interoceptive deficits facilitate engagement in NSSI, yet they have little impact on engagement in eating disorder behaviors. Future research should investigate potential explanations for this pattern of results. For example, perhaps expectations of NSSI and eating disorder behaviors differ: if one expects direct and immediate pain or injury resulting from NSSI, yet does not anticipate as great a degree of pain or injury from eating disorder behaviors, then disconnection from the experiences and sensations of the body may facilitate the former to a greater extent than the latter. If applicable, the idea of body regard – i.e., how one experiences and pereceives the body influences how she treats it, including the ability to harm it through damaging behaviors (Muehlenkamp, 2012) – could offer a simple, straightforward explanatory model for engagement in eating disorder behaviors; to the extent that one experiences the body as an object, is dissatisfed with or disconnected from the body, does not have protective attitudes toward the body, and does not appreciate the body's abilities and capacity for health, engaging in behavior that hurts or weakens the body, such as eating disorder behaviors, should be easier. Future research should examine whether the idea of body regard might still apply to eating disorder behaviors, by determining if other components of body regard (e.g., body objectification, body satisfaction) are stronger predictors of engagement in eating disorder behaviors than interoception.

The present study is the first, to my knowledge, to investigate the effects of interoceptive deficits on the components of ACS, FAD and pain tolerance. In the present sample, interoceptive deficits were significantly related to FAD but not pain tolerance. Interoceptive deficits indicate a difficulty with accurately identifying and responding to internal sensations (including emotions), and thus may make it hard for a person to accurately identify fear about death and dying, leading to higher reported levels of FAD. However, the same logic would theoretically apply to the effects of interoceptive deficits on pain tolerance (i.e., that by creating disconnect from the body, interoceptive deficits would relate to higher perceived pain tolerance), yet no link was found between interoceptive deficits and pain tolerance in the present study. It is possible that pain is a more salient and easily identifiable state than fear, making pain tolerance

less susceptible to influence from interoceptive deficits than FAD; alternatively, it is possible that the self-report measure of interoceptive deficits used in the present study, which had a large proportion of items asking about emotional feelings, may have been conceptually more related to the construct of FAD than pain tolerance. However, additional research would be needed in order to determine why interoceptive deficits significantly predicted FAD but not pain tolerance, and whether other, perhaps more objective or emobodied, methods of measuring interoception (i.e., heartbeat perception task; Schandry, 1981; Pollatos, et al., 2008) may show a relationship between interoceptive deficits and pain tolerance.

Strategies to improve an individual's interoception have recently begun to be studied and have shown some promise. For example, providing participants with cardio-visual feedback (Aspell et al., 2013) and asking participants to direct their attention to a picture of themselves or to self-relevant words (Ainley, Maister, Brokfeld, Farmer, & Tsakiris, 2013) have been shown to improve interoceptive awareness. Additional strategies to improve interoceptive awarness, such as mindfulness training (i.e., increasing one's ability to be aware of thoughts and feelings in the present moment) should be examined, and further, given the association found in the present study between interoceptive deficits and FAD, future research should examine whether strategies to improve interoception lead to subsequent decreases in FAD. It is theorized that ACS is a relatively stable construct, increasing over time (Van Orden, et al., 2010). However, longitudinal investigatsions of ACS have not been conducted, so future research is needed to increase understanding of how ACS changes over time, whether it can decrease generally, and whether strategies to mitigate the effects of specific factors that contribute to it would work to decrease ACS (such as improving interoception, which predicts higher FAD). Understanding whether and how ACS could be diminished, through attempts to improve interoception for example, could inform the development of more effective suicide prevention interventions, particularly among populations at high risk for suicide such as those with eating disorders. However, until it is known whether efforts to decrease ACS can be successful, attempts to reduce suicide risk in clinical settings should focus on intervening at the level of the two other components of the IPTS (i.e., decreasing burdensomeness and increasing belongingness) which are believed to be more immediately ammenable to change.

The results of the present study also point to the importance of assessing NSSI as a risk factor for suicide among those with eating disorders. In the present study, NSSI emerged as the

strongest predictor of pain tolerance. Thus although NSSI is not related to eating disorder pathology, it may be an important target for early intervention in order to decrease suicide risk among individuals seeking eating disorder treatment who also engage in NSSI.

The present study focused on the effects of specific risk factors on the components of ACS, rather than examining the effects of disorders or pre-determined constellations of symptoms. However, exploratory analyses revealed that diagnostic status may moderate the relationships between study variables. Given that self-reported lifetime engagement in behaviors most characteristic of each diagnosis were examined (i.e., restraint, vomiting, binge eating, over-exercise) it is unclear what role diagnostic status plays in impacting the relationships between variables. Future research is needed comparing factors that influence ACS using larger sized samples of participants with each eating disorder diagnosis, so that a more thorough comparison across diagnoses can be conducted, and the role of diagnosis as a moderator of relationships between variables which influence ACS can be more fully understood.

In combination, eating disorder behaviors added to the prediction of FAD above and beyond NSSI and interoceptive deficits. Consistent with previous research (Smith et al., 2013), over-exercise was a marginally significant predictor of FAD when controlling for the other eating disorder behaviors. Thus over-exercise may be a particularly important target for early intervention among those seeking treatment for an eating disorder. However, no individual eating disorder behavior emerged as a significant predictor of pain tolerance, and no predictors reached significance at the .05 level for FAD, suggesting that eating disorder pathology as a whole may be a better predictor of ACS than any one eating disorder symptom. Subthreshold disordered eating behaviors are more prevalent than full syndrome DSM eating disorders (Stice, Marti, Shaw, & Jaconis, 2009), and given that ACS is believed to develop over time, through repeated exposure to painful and provocative events (Joiner, 2005; Van Orden et al., 2010), interventions which address eating pathology and NSSI soon after their onset, regardless of disorder status, could be beneficial. Such interventions could prevent continued repetition of eating disorder behaviors and NSSI, and thus prevent the development of ACS through the repetition of NSSI or eating disorder behaviors. Earlier interventions for behaviors which contribute to ACS, particularly among those at risk for eating disorders, may be one way to decrease risk of suicide among those with eating disorders.

Several methodological limitations of the present study should be considered when interpreting the results. Data were cross-sectional, and thus causal directions of the relationshionships between constructs were theoretically derived and were not demonstrated empirically across time. Further, the present sample was made up exclusively of treatment-seeking females, the large majority of whome were Caucasian; this limits generalizability of the present results to other populations. Regarding measures used in the present study, the EPPES is a relatively new and unused instrument, and although it demonstrated acceptable internal reliability in the present sample, its broader psychometric properties are unknown. Finally, pain tolerance was determined via a single self-report item. Although assessing pain tolerance by taking multiple physiological measurements (e.g. with a pressure algometer) may have provided a more objective measure of pain tolerance, the subjective single-item measure did offer valuable information. According to the IPTS, the pain tolerance component of ACS requires that in order to enact near-fatal or fatal self-harm, a person believe that he or she will be capable of tolerating the pain of death by suicide, which relates to a person's subjective assessment of personal pain tolerance. Thus subjective (i.e., self-report) assessments of pain tolerance may offer useful and unique information; future research should aim to determine whether subjectove or objective measures of pain tolerance are more accurately predictive of suicidal behavior.

Despite these limitations, the present study provides an important contribution to the existing literature on suicide risk among those with eating disorders. The use of a clinical sample allowed me to examine and compare the effects of four eating disorder behaviors and NSSI on the components of ACS, among a highly relevant population shown to have elevated risk for suicide. Understanding factors that contribute to ACS, and thus according to the IPTS contribute to overall risk of death by suicide, should aid in the development of more effective suicide prevention strategies for this population. However, the utility of understanding what factors contribute to ACS hinges on the accuracy of the assumption that ACS is predictive of suicidal behavior among those with eating disorders, as would be expected based on the IPTS. Although preliminary support for the hypotheses of the IPTS has been found (Joiner et al., 2009; Van Orden, et al., 2008), additional research is needed to confirm the theorized role of ACS in increasing one's likelihood of making fatal or near-fatal suicide attempts.

Results of the present analyses suggest that among women seeking treatment for eating disorders, interoceptive deficits are a strong predictor of FAD, while engagement in NSSI is a

strong predictor of pain tolerance; and although in combination eating disorder behaviors contribute to FAD, no individual eating disorder behavior emerged as a strong predictor by itself. Thus, two risk factors which appear to elevate ACS among those with eating disorders are neither unique to or characteristic of eating disorders. Separate interventions to address interoceptive deficits and NSSI in addition to eating pathology may help reduce suicide risk among those with eating disorders.

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Table 1

Demographic and Eating Disorder Characteristics of the Sample (N = 98)

Diagnosis			%	*n*
AN			34.7	34
BN			27.6	27
EDNOS			37.8	37
Race			%	*n*
American Indian / Alaska Native			1	1
Native Hawaiian or Other Pacific Islander			1	1
Black or African American			2	2
White			95.9	94
Ethnicity			%	*n*
Hispanic/Latino			2	2
Not Hispanic/Latino			98	96
Treatment Program			%	*n*
Residential			81.6	80
Partial Hospitalization			18.4	18
	M	*SD*	Min	Max
Age	26.92	7.86	18	58
BMI	20.36	4.74	12.88	43.65

Table 2

Descriptive Statistics for all Study Variables, in all Forms

Dataset	Variable	*N*	*M*	*SD*	Min	Max
Original	ID	96	33.20	8.91	11.00	53.00
	NSSI, original	92	91.61	204.95	0.00	1467.00
	NSSI, transformed	92	1.22	0.90	0.00	3.17
	restraint	73	4.90	2.68	0.00	9.00
	over-exercise	73	4.43	2.50	0.00	9.00
	vomiting	73	2.39	1.55	0.00	5.67
	binge eating	73	5.08	3.45	0.00	9.00
	FAD	98	13.96	7.07	0.00	28.00
	pain tolerance	98	2.30	1.11	0.00	4.00
Pooled values across 40 multiply imputed datasets	ID	98	33.16	-	-	-
	NSSI, original	-	-	-	-	-
	NSSI, transformed	98	1.21	-	-	-
	restraint	98	4.96	-	-	-
	over exercise	98	4.49	-	-	-
	vomiting	98	2.43	-	-	-
	binge eating	98	5.15	-	-	-
	FAD	98	13.96	-	-	-
	pain tolerance	98	2.30	-	-	-
Averaged value across all imputed values	ID	98	33.16	8.82	11.00	53.00
	NSSI, original	-	-	-	-	-
	NSSI, transformed	98	1.21	0.87	0.00	3.17
	restraint	98	4.96	2.39	0.00	9.00
	over exercise	98	4.49	2.26	0.00	9.00
	vomiting	98	2.43	1.42	0.00	5.67
	binge eating	98	5.15	3.09	0.00	9.35
	FAD	98	13.96	7.07	0.00	28.00
	pain tolerance	98	2.30	1.11	0.00	4.00

Note: ID: interoceptive deficits; NSSI: non-suicidal self-injury; FAD: fearlessness about death

Table 3

Intercorrelations Between all Study Variables

	Age	ID	FAD	Pain Tolerance	NSSI	Restraint	Over-exercise	Vomiting	Binge eating
Age	1.00	–	–	–	–	–	–	–	–
ID	-.21*	1.00	–	–	–	–	–	–	–
FAD	.04	.32**	1.00	–	–	–	–	–	–
Pain Tolerance	.06	.14	.33**	1.00	–	–	–	–	–
NSSI	-.18	.41**	.31**	.41**	1.00	–	–	–	–
Restraint	-.13	.22	.15	.15	.22	1.00	–	–	–
Over-exercise	-.20	.13	.25*	.22	.21	.41**	1.00	–	–
Vomiting	-.19	.16	.20	.01	.20	.38**	.22	1.00	–
Binge Eating	-.10	.21	.05	.03	.14	.13	.10	.52**	1.00

Note. All correlations computed with pooled results of multiply imputed data (m = 40)
ID: interoceptive deficits; NSSI: non-suicidal self-injury; FAD: fearlessness about death
*Correlation is significant at the .05 level
**Correlation is significant at the .01 level

Table 4

Prediction of NSSI and Eating Disorder Behaviors from Interoceptive Deficits, Controlling for Age

Dependent Variable	Step	Variables Entered	Regression Coefficients*					Model Summary†	
			B	SE	t	p	sr^2	R^2 Δ	Change Statistics
Restraint	1	(Constant)	6.25	1.43	4.36	.000		.025	F Δ (1,96) = 2.46, p = .120
		age	-0.05	0.05	-0.95	.344	.018		
	2	(Constant)	3.70	2.20	1.68	.094		.056	F Δ (1,95) = 5.80, p = .018
		age	-0.03	0.05	-0.63	.527	.008		
		ID	0.06	0.04	1.66	.098	.040		
Over-Exercise	1	(Constant)	6.28	1.20	5.22	.000		.054	F Δ (1,96) = 5.43, p = .022
		age	-0.07	0.04	-1.55	.122	.038		
	2	(Constant)	5.20	1.96	2.65	.009		.012	F Δ (1,95) = 1.22, p = .273
		age	-0.06	0.04	-1.35	.177	.030		
		ID	0.03	0.04	0.75	.456	.008		
Vomiting	1	(Constant)	3.50	0.71	4.95	.000		.049	F Δ (1,96) = 4.92, p = .029
		age	-0.04	0.03	-1.55	.122	.036		
	2	(Constant)	2.60	1.13	2.29	.022		.021	F Δ (1,95) = 2.11, p = .150
		age	-0.03	0.03	-1.31	.192	.026		
		ID	0.02	0.02	1.06	.288	.015		
Binge Eating	1	(Constant)	6.43	1.69	3.81	.000		.015	F Δ (1,96) = 1.43, p = .235
		age	-0.05	0.06	-0.80	.423	.011		
	2	(Constant)	3.34	2.54	1.32	.189		.050	F Δ (1,95) = 5.11, p = .026
		age	-0.03	0.06	-0.49	.626	.004		
		ID	0.08	0.05	1.66	.098	.035		
NSSI	1	(Constant)	1.76	0.33	5.37	.000		.034	F Δ (1,96) = 3.37, p = .069
		age	-0.02	0.01	-1.74	.082	.032		
	2	(Constant)	0.23	0.49	0.47	.638		.153	F Δ (1,95) = 17.82, p < .001
		age	-0.01	0.01	-1.02	.309	.010		
		ID	***0.04***	***0.01***	***3.98***	***.000***	***.143***		

Note: ID: Interoceptive deficits; NSSI: Non-Suicidal Self-Injury; *Regression coefficients calculated using pooled variances of multiply imputed data (m = 40); † Model summary statistics calculated using averaged values across 40 imputed datasets

Table 5

FAD and Pain Tolerance Hierarchically Regressed on Age, NSSI and Eating Disorder Behaviors

Dependent Variable	Step	Variables Entered	Regression Coefficients*					Model Summary†	
			B	SE	t	p	sr^2	$R^2\Delta$	Change Statistics
Fearlessness about Death	1	(Constant)	12.92	2.57	5.02	.000		.002	F Δ (1,96) = 0.18, p = .674
		age	0.04	0.09	0.42	.673	.002		
	2	(Constant)	8.32	2.83	2.94	.003			
		age	0.09	0.09	1.03	.302	.010	***.111***	***F Δ (1,95) = 11.85, p = .001***
		NSSI	***2.61***	***0.82***	***3.19***	***.001***	***.106***		
	3	(Constant)	4.13	3.57	1.16	.247			
		age	0.15	0.09	1.58	.113	.023		
		NSSI	***2.24***	***0.84***	***2.65***	***.008***	***.071***		
		restraint	-0.07	0.34	-0.21	.830	.001	***.085***	***F Δ (4,91) = 2.42, p = .054***
		over-exercise	0.53	0.31	1.70	.090	.031		
		vomiting	0.81	0.59	1.39	.166	.022		
		binge eating	-0.16	0.26	-0.60	.546	.005		
Pain Tolerance	1	(Constant)	2.08	0.40	5.13	.000		.003	F Δ (1,96) = 0.31, p = .578
		age	0.01	0.01	0.56	.576	.003		
	2	(Constant)	1.13	0.43	2.64	.008			
		age	0.02	0.01	1.42	.154	.018	***.191***	***F Δ (1,95) = 22.56, p < .001***
		NSSI	***0.54***	***0.12***	***4.50***	***.000***	***.182***		
	3	(Constant)	0.83	0.55	1.50	.134			
		age	0.02	0.01	1.54	.125	.021		
		NSSI	***0.51***	***0.13***	***4.07***	***.000***	***.151***		F Δ (4,91) = 1.52, p = .204
		restraint	0.02	0.05	0.38	.706	.002	.050	
		over-exercise	0.07	0.05	1.39	.166	.022		
		vomiting	-0.07	0.10	-0.73	.463	.007		
		binge eating	0.01	0.04	0.15	.878	.000		

Note: NSSI: Non-Suicidal Self-Injury; *Regression coefficients calculated using pooled variances of multiply imputed data (m = 40); † Model summary statistics calculated using averaged values across 40 imputed datasets

Table 6

FAD and Pain Tolerance Hierarchically Regerssed on Age, Active and Passive Eating Disorder Behaviors

Dependent Variable	Step	Variables Entered	Regression Coefficients*					Model Summary†	
			B	SE	t	p	sr^2	$R^2\Delta$	Change Statistics
Fearlessness about Death	1	(Constant)	12.92	2.57	5.02	.000		.002	F Δ (1,96) = 0.18, p = .674
		age	0.04	0.09	0.42	.673	.002		
	2	(Constant)	6.58	3.45	1.90	.057		***.132***	***F Δ (3,93) = 4.74, p = .004***
		age	0.11	0.09	1.19	.233	.014		
		over-exercise	***0.62***	***0.29***	***2.11***	***.035***	***.050***		
		vomiting	0.91	0.57	1.59	.111	.031		
		binge eating	-0.12	0.26	-0.48	.635	.003		
	3	(Constant)	6.49	3.60	1.80	.072		.000	F Δ (1,92) = 0.01, p = .931
		age	0.11	0.10	1.20	.230	.015		
		over-exercise	***0.62***	***0.32***	***1.95***	***.052***	***.043***		
		vomiting	0.90	0.60	1.51	.132	.027		
		binge eating	-0.12	0.26	-0.47	.642	.003		
		restraint	0.01	0.33	0.03	.973	.000		
Pain Tolerance	1	(Constant)	2.08	0.40	5.13	.000		.003	F Δ (1,96) = 0.31, p = .578
		age	0.01	0.01	0.56	.576	.003		
	2	(Constant)	1.46	0.56	2.60	.009		***.083***	***F Δ (3,93) = 2.83, p = .043***
		age	0.01	0.02	0.94	.345	.009		
		over-exercise	***0.10***	***0.05***	***2.05***	***.041***	***.056***		
		vomiting	-0.03	0.10	-0.28	.779	.001		
		binge eating	0.01	0.04	0.27	.786	.001		
	3	(Constant)	1.37	0.58	2.38	.017		.008	F Δ (1,92) = 0.85, p = .358
		age	0.01	0.02	0.95	.343	.009		
		over-exercise	0.09	0.05	1.67	.096	.036		
		vomiting	-0.05	0.11	-0.48	.630	.003		
		binge eating	0.01	0.04	0.33	.742	.002		
		restraint	0.04	0.05	0.71	.479	.007		

Note: *Regression coefficients calculated using pooled variances of multiply imputed data (m = 40); † Model summary statistics calculated using averaged values across 40 imputed datasets

Table 7

FAD and Pain Tolerance Hierarchically Regressed on Age, Interoceptive Deficits, NSSI, and Eating Disorder Behaviors

Dependent Variable	Step	Variables Entered	Regression Coefficients*					Model Summary†	
			B	SE	t	p	sr^2	$R^2\Delta$	Change Statistics
Fearlessness about Death	1	(Constant)	12.92	2.57	5.02	.000		.002	FΔ (1,96) = 0.18, p = .674
		age	0.04	0.09	0.42	.673	.043		
	2	(Constant)	2.12	3.94	0.54	.591		***.116***	***FΔ (1,95) = 12.46, p = .001***
		age	0.10	0.09	1.15	.248	.111		
		ID	***0.27***	***0.08***	***3.48***	***.001***	***.337***		
	3	(Constant)	1.70	3.86	0.44	.660		***.046***	***FΔ (1,94) = 5.21, p = .025***
		age	0.12	0.09	1.41	.160	.133		
		ID	***0.20***	***0.08***	***2.41***	***.016***	***.230***		
		NSSI	***1.84***	***0.88***	***2.10***	***.036***	***.212***		
	4	(Constant)	-2.37	4.26	-0.56	.578		***.089***	***FΔ (4,90) = 2.69, p = .036***
		age	0.18	0.09	1.97	.049	.185		
		ID	***0.22***	***0.09***	***2.53***	***.011***	***.240***		
		NSSI	1.49	0.88	1.69	.090	.166		
		restraint	-0.17	0.35	-0.50	.614	-.058		
		over-exercise	0.56	0.31	1.78	.076	.186		
		vomiting	0.89	0.58	1.52	.130	.161		
		binge eating	-0.24	0.26	-0.94	.350	-.105		
Pain Tolerance	1	(Constant)	2.08	0.40	5.13	.000		.003	FΔ (1,96) = 0.31, p = .578
		age	0.01	0.01	0.56	.576	.057		
	2	(Constant)	1.29	0.65	1.98	.048		.025	FΔ (1,95) = 2.45, p = .121
		age	0.01	0.01	0.87	.384	.088		
		ID	0.02	0.01	1.54	.122	.157		
	3	(Constant)	1.16	0.60	1.93	.053		***.167***	***FΔ (1,94) = 19.44, p < .001***
		age	0.02	0.01	1.39	.165	.130		
		ID	0.00	0.01	-0.08	.932	-.008		
		NSSI	***0.55***	***0.13***	***4.14***	***.000***	***.397***		
	4	(Constant)	0.90	0.67	1.34	.180		.051	FΔ (4,90) = 1.51, p = .205
		age	0.02	0.01	1.49	.137	.141		
		ID	0.00	0.01	-0.19	.849	-.018		
		NSSI	***0.52***	***0.14***	***3.87***	***.000***	***.370***		
		restraint	0.02	0.05	0.39	.694	.042		
		over-exercise	0.07	0.05	1.37	.170	.146		
		vomiting	-0.07	0.10	-0.74	.462	-.084		
		binge eating	0.01	0.04	0.18	.861	.019		

Note: ID: Interoceptive Deficits; NSSI: Non-Suicidal Self-Injury; *Regression coefficients calculated using pooled variances of multiply imputed data; † Model summary statistics calculated using averaged values across 40 imputed datasets

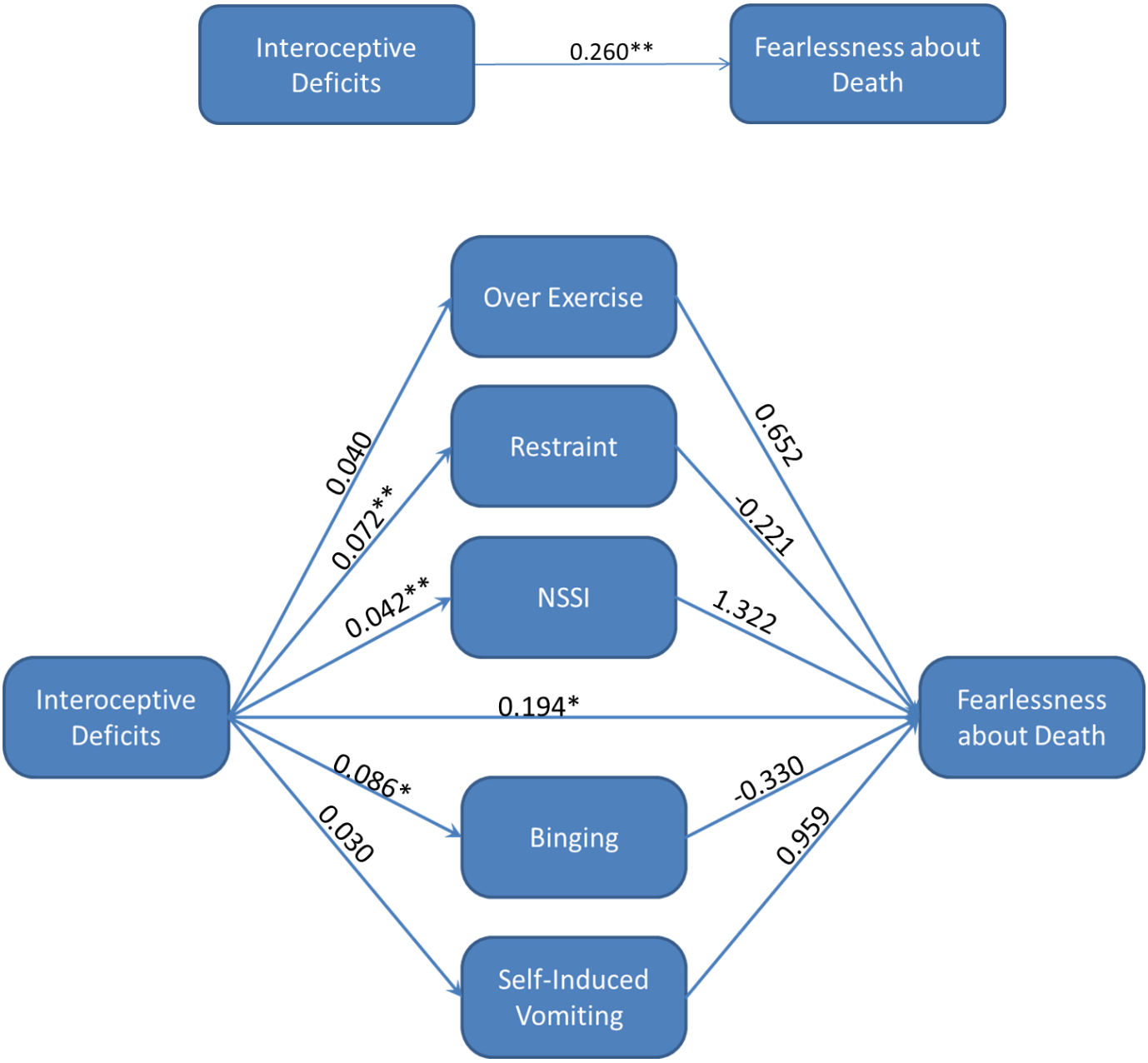

Figure 1: Unstandardized coefficients for the total effect of interoceptive deficits on fearlessness about death (top), and for each path in the parallel mediation model (bottom)
Note: *denotes significance at the .05 level; **denotes significance at the .01 level
The total indirect effect (not depicted here) was not significant (95% CI [-0.04 – 0.19])

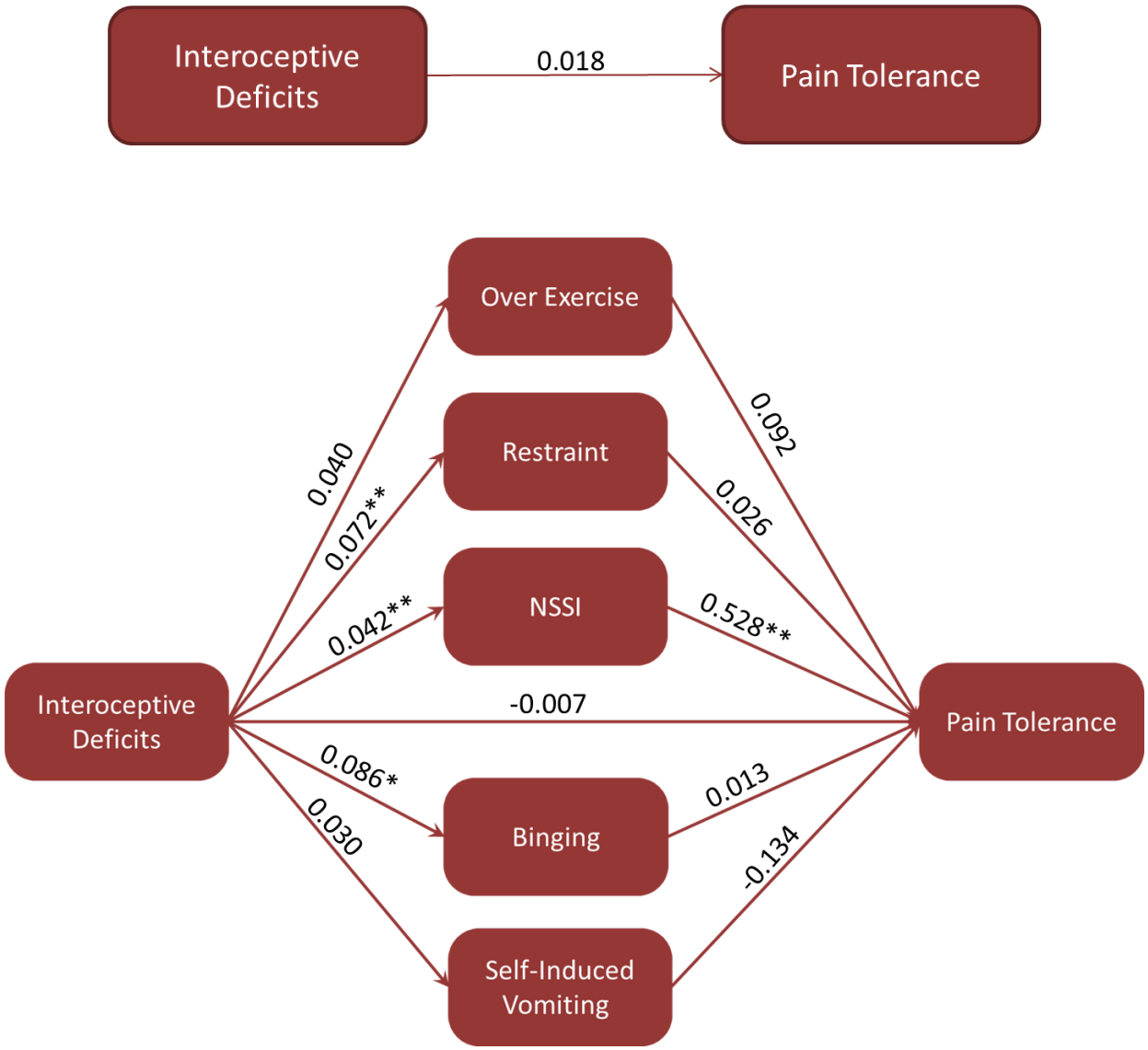

Figure 2: Unstandardized coefficients for the total effect of interoceptive deficits on pain tolerance (top), and for each path in the parallel mediation model (bottom)

Note: *denotes significance at the .05 level; **denotes significance at the .01 level

The total indirect effect (not depicted here) was significant (95% CI [0.01 – 0.05])

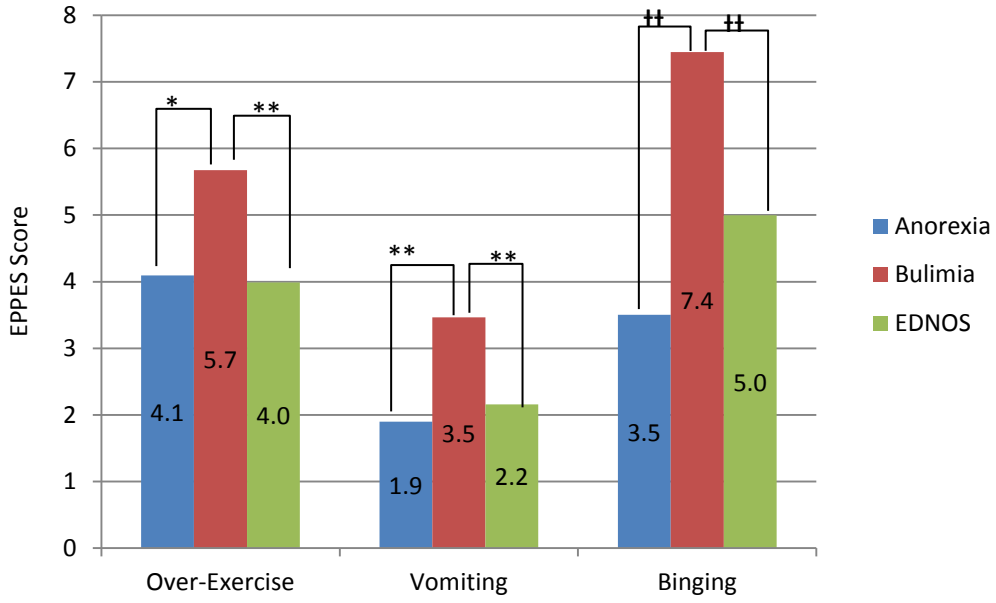

Figure 3a: Differences between diagnostic groups on study variables: Over-exercise, vomiting, and binging

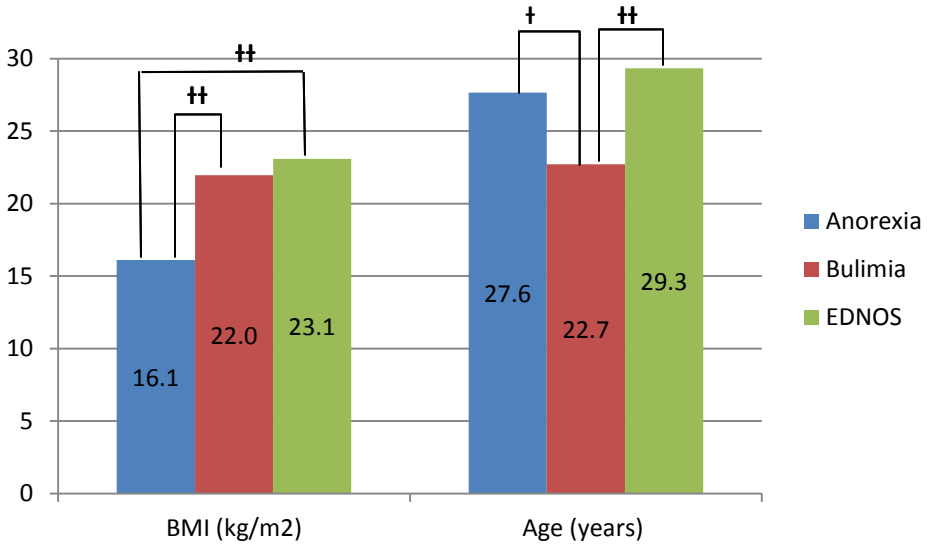

Figure 3b: Differences between diagnostic groups on study variables: BMI and age

Note: * Tukey's HSD post hoc test revealed significant difference at .05 level; ** Tukey's HSD post hoc test revealed significant differences at .01 level; † Dunnett T3 post hoc test revealed significant difference at .05 level; †† Dunnett T3 post hoc test revealed significant difference at .01 level. No significant differences were found between diagnostic groups on mean FAD, pain tolerance, interoceptive deficits, restraint, or NSSI.