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The Moderating Effect of Skin Conductance Level on the Relationship between Family Conflict and Disordered Eating Behaviors

Laura B. Kenneally

University at Albany, State University of New York

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The Moderating Effect of Skin Conductance Level on the Relationship between Family Conflict
and Disordered Eating Behaviors

An honors thesis presented to the
Department of Psychology,
University at Albany, State University Of New York
in partial fulfillment of the requirements
for graduation with Honors in Psychology
and
graduation from The Honors College.

Laura B. Kenneally

Research Advisor: Elana B. Gordis, Ph.D

Second Reader: Mitchell Earleywine, Ph.D

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Abstract

The purpose of this study was to examine the moderating effect of the sympathetic nervous system (SNS) on the relation between family conflict and disordered eating behaviors. Participants were 67 undergraduate students at the University at Albany, SUNY, ages 17 to 40. Researchers used a retrospective measure of harsh parenting and interparental aggression experienced during childhood and adolescence to determine how sympathetic activation interacts with family conflict in predicting disordered eating behaviors in a sample of university students. SNS activation was measured by baseline skin conductance level (SCL). Results indicated that SCL moderated the relationship between harsh parenting experienced during childhood and eating disorder behaviors. This suggests that perhaps in individuals at risk for disordered eating behaviors as a result of harsh parenting, lower levels of sympathetic activation fail to inhibit the impulse to engage in potentially risky or destructive behaviors.

Keywords: childhood development, psychophysiology, sympathetic nervous system, skin conductance level, impulse control, BAS/BIS, child abuse, family conflict, eating disorders

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Introduction

Exposure to family conflict, such as interparental aggression or harsh parenting, during childhood and adolescence has been shown to lead to many adverse consequences (Cummings & Davies, 2002; Ehrensaft et al., 2003; Jouriles, Murphy, & O’Leary, 1989; Springer, Sheridan, Kuo, & Carnes, 2007). Among these consequences is the development of eating disorders, or disordered eating behaviors (Botta & Dumalo, 2009; May, Kim, McHale, & Crouter, 2006; Wisotski et al., 2006). Seeing as not all children or adolescents who experience family conflict experience these adverse effects however, researchers have begun to look into how individual biological factors, such as autonomic nervous system function, may influence this relationship between family conflict and later problems (El Sheikh et al. 2009; Fowles, Kochanska, & Murray, 2000; Gordis, Feres, Olezeski, Rabkin, & Trickett, 2010; Raine, 2005). These findings have spurred interest in researching the effects of biological mechanisms on other maladaptive outcomes that can reasonably be linked to nervous system function. The interconnections between disordered eating behaviors and impulse control issues suggest that perhaps there are biological underpinnings elevating some individuals’ risk of developing these behaviors. The present study explores how varying types of family conflict, in the form of interparental aggression or parent to child aggression, experienced during either childhood or adolescence, influence the development of disordered eating behaviors. In addition, it examines how these relationships are influenced by sympathetic nervous system activity.

Family Conflict and Eating Disorders

The research regarding the effects of family conflict on eating disorder symptomology is controversial. Research has shown that high levels of family discord predict both externalizing and internalizing behaviors (Boyce & Ellis, 2005; El Sheikh, 2005). Also, the type of conflict

experienced has been found to impact the likelihood of psychopathology and, to some extent, the age at which it occurs (Benjet, Borges, & Medina-Mora, 2010). Many studies have found evidence to support a direct link to the development of eating disorders as a consequence of family conflict (Bowles, Kurlender, & Hellings, 1993; Tata, Fox, & Cooper, 2001; Wisotsky et al., 2006). The types of conflict associated include, but are not limited to, parenting style, perceived family dysfunction, and communication patterns. Wisotsky and colleagues found that as the level of perceived family dysfunction increased, so did eating disorder symptomology and psychopathology (2006). Parent to child conflict, particularly between mothers and daughters, was found to significantly predict weight concerns (May et al., 2006). In accord, Bowles, Kurlender, and Hellings, found that authoritarian parenting styles (low intimacy and democracy paired with higher levels of conflict) significantly increased a child's risk of developing an eating disorder. This finding was further supported and expanded to fathers by Botta and Dumalo, (2009) who suggested that effective communication and conflict resolution between fathers and daughters was protective against anorexic behaviors, whereas communication patterns in which the daughter was consistently giving in to the father's arguments was predictive of anorexia. Poor parent-child interaction in the form of overprotection has also been found to predict disordered eating in females (Tata, Fox, & Cooper, 2001). These studies all support the notion that families with high levels of varying types of conflict were more likely to predict disordered eating behaviors.

This finding is not universal, however. Hanna and Bond (2006) found that the significant relationship observed between family conflict and eating disorder symptomology in their sample was completely mediated by the participant's perceived negative messages regarding their appearance. Another consideration in recent literature is the suggestion that this relationship

between family conflict and disordered eating works in the opposite direction as well, such that parental conflict and or parent to child conflict is a result of, as opposed to a predictor of, disordered eating (Sim et al., 2009; Spanos, Klump, Burt, McGue, & Lacono, 2010). Wade, Gillespie, and Martin (2007) also found no significant relationship between either anorexia nervosa or bulimia nervosa and parental conflict in a sample of monozygotic twins. This finding was supported and expanded upon by Spanos and colleagues (2010), who studied monozygotic twin girls longitudinally over the course six years. Their results indicated that that parent to child conflict did not significantly predict disordered eating, but that disordered eating did significantly predict parent to child conflict.

These discrepancies in the literature may be accounted for by the type of conflict experienced. Studies looking at maltreatment in the form of neglect or abuse specifically found more consistent results. Johnson and colleagues found that children exposed to neglect or abuse were at a significantly elevated risk of developing eating or weight problems (Johnson, Cohen, Kasen, & Brook, 2002). Similarly, Klemsmeden-Fosse and Holen found that emotional, physical, and sexual abuse significantly predicted a diagnosis of bulimia nervosa (2005). These findings support the results of Grilo and Masheb (2001) that eighty- two percent of individuals with binge eating disorders experienced at least one form of childhood maltreatment. Many of the studies conducted on this topic, however, have defined childhood as “under eighteen,” rather than considering differential effects as a result of different age ranges during which the conflict was experienced.

Considering the high correlations between interparental conflict and harsh parenting, it is difficult to examine them individually in the literature. These wide variations in the literature exemplify the need to continue to research on the individual variables contributing to the

relationship between maltreatment and eating disturbances. The present study seeks to clarify this by looking at both interparental conflict, and harsh parenting, to see how the type of family conflict may uniquely affect the outcome. Also, the age at which the participant experienced the conflict is assessed, in order to see if exposure to the conflict at different ages has a differential impact on the effects.

Biology and Eating Disorders

Not every child exposed to conflict develops eating disturbances. We can conclude that individual factors likely make some children more vulnerable than others. One way this can be explored is by looking at the biological variables involved. A few different biological mechanisms have been implicated in the development of eating disorders. Connan, Campbell, Katzman, Lightman, and Treasure (2003) found evidence for irregular hypothalamic-pituitary-adrenal (HPA) axis function in individuals diagnosed with Anorexia Nervosa. Accordingly, studies have found that children who have been maltreated often have asymmetrical activation of the HPA axis and sympathetic nervous system (Gordis, Granger, Susman, & Trickett, 2006; Gordis, Granger, Susman, & Trickett, 2008). Similarly, Beauchaine (2001) suggested the effects of biological involvement on psychopathology are likely due to complex interactions between the branches of the Autonomic Nervous System (ANS). Another theory proposing a biological role in impulsivity and psychopathology is the Somatic Marker Hypothesis (Damasio, 1996). This theory posits that decision making is influenced by our emotional responses as influenced by feedback from the autonomic nervous system responses. In this way, when the response is not functioning properly, decision making will be affected.

Resting levels of the sympathetic nervous system activity have also been implicated in moderating the relationship between stressful experiences and their consequences (El Sheikh et

al., 2009; Obradovic et al., 2011; Raine, 2005). Sympathetic nervous system activity leads to hydration of the eccrine sweat glands of the hand's palmar surface, which in turn changes the skin conductance level (SCL). Thus, this is a frequent measure of sympathetic activation. Low baseline SCL has been linked to maladaptive behaviors such as aggression, sensation seeking (Raine, 2005; Fowles et al., 2000), and diminished impulse control (Fowles et al., 2000; Tchanturia, 2007). Equally, increased sympathetic activation, characterized by more inhibited behavior, has been found to be associated with internalizing behaviors (Kagan, Reznick, & Snidman, 1987; Scarpa, Raine, Venables, & Mednick, 1997). The prevailing explanation for these associations stem from the fact that SNS activity is thought to reflect patterns of Gray's motivational systems, in particular the behavioral activation system and behavioral inhibition system (Gray, 1987). By this theory, both low behavioral inhibition and high behavioral approach can be problematic, seeing as high BAS indicates higher approach responses regardless of punishments, and low BIS indicates less behavioral inhibition to approach responses as a result of the lower sensitivity to punishment. These mechanisms have been implicated in a large number of clinical disorders including eating disorders, addictions (Dawe & Loxton, 2004), ADHD, and conduct disorder (Beauchaine, 2001).

These sympathetic processes have multiple implications for behavior. When an individual has lower levels of sympathetic activation, their arousal is naturally lower, and they may engage in sensation seeking (Raine, 2005). Individuals with lower sympathetic activation are also found to demonstrate more fearlessness; they may not be inhibited in the face of risky situations the way individuals with average to higher levels are. Similarly, low levels of SCL are indicative of decreased impulse control (Fowles et al., 2000; Kagan et al., 1987; Scarpa et al., 1997) as a result of their decreased ability to exercise effortful control and inhibit their responses.

This implication has been linked to both eating disorders, and disordered eating behaviors in the literature (Claes, Nederkoorn, Vandereycken, & Guerrieri, 2006; Fernandez- Aranda et al., 2006, Mobbs, Ghisletta, & Van der Linden, 2008). Svaldi, Brand, and Tuschen-Caffier (2010) found that women with binge eating disorder were significantly more likely than healthy controls to make a risky choice when participating in a gambling task. They also found that women with binge eating disorder were less likely than the healthy controls to change their strategy from risky to safe after receiving negative feedback following the risky choice. Similarly, Farmer, Nash, and Field, (2001) found a significant correlation between reward sensitivity (indicative of elevated BAS) and average weekly purge. The participants were unable to resist the impulse even though they were fully aware that it was not a safe idea. Tchanturia and colleagues (2007) similarly found that participants with anorexia nervosa had significantly lower performance on the gambling task than the healthy controls, and also that they had significantly lower levels of anticipatory SCL. Moreover, Dawe and Loxton (2004) suggested that both increased reward sensitivity, an indicator of the BAS, and disinhibited loss of control, indicative of decreased BIS function, are associated with vulnerability towards developing binge eating behavior.

Claes, Robinson, Muehlenkamp, Vandereycken, and Bijttebier (2010) also found a relationship between BIS function and disordered eating. Their results indicated that lower levels of BIS were present among both bingeing purging type and restrictive type eating disorder patients. Also, a discriminatory pattern of BIS functioning was found, such that bingeing purging patients had significantly lower BIS function, effortful control, and cognitive control than did the restrictive eating disorder subgroup. This finding however this was contradicted by Claes, Bijttebier, Mitchell, Zwaan, and Mueller (2011) who found that eating disorders were characterized by high levels of the BIS activation. Research done by Harrison, Treasure, and

Simille (2011) supported this finding, suggesting that individuals with eating disorders have significantly higher sensitivity to punishment (indicative of more behavioral inhibition) than the healthy control group. This incongruity may be explained by the fact that eating disorders in these studies were predominantly driven by a “drive for thinness” rather than bingeing purging behaviors, therefore representing more restricting, rather than impulsive, symptoms. These discrepancies demonstrate the necessity to further research and clarify the role of the BAS/ BIS in patients with disordered eating.

The involvement of impulsivity has broad implications for treatment as well. Cavedini et al. (2006) found that anorectic patients who acted less impulsively during the Gambling Task had significantly greater nutritional improvement after treatment, suggesting that patients with comorbid impulsivity were more resistant to treatment. Interestingly, Harrison and colleagues (2011) found no significant differences in BAS/BIS activation between the acutely ill group of anorectics and the recovered group. These findings may be explained by the idea that while the disorders can be treated, the BAS/ BIS is still predominantly stable over time as a result of its biological foundations. In this case its effect needs to be considered. Some have even suggested the identification of an impulsive subtype of particular eating disorders (Lacey & Evans, 1986). Fay and Eisler (1993), however, found that while impulsivity shaped the expression of the eating disorder, it was not found to pose any significant implications for treatment outcome, and therefore concluded that there was no evidence for a specific subset of the disorder.

However, since then, it has been found that between twenty and thirty percent of patients with bulimia nervosa had a comorbid impulse control disorder, accompanied by higher levels of disruptive symptomology (Fernandez- Aranda et al., 2006; Matsunaga et al., 1998). Lacey and Evans (1986) also demonstrated evidence of an impulsive subtype of individuals with eating

disorders. This evidence of an impulsive subset of patients with eating disorders and impulsivity needs to be further explored in order to make the most of treatment and intervention. Also, it is worth noting that these studies took place in Spain, Japan, and England respectively. The fact that the link between impulsivity and disordered eating has been found cross culturally, as well as in both normative and clinical samples, seems to be indicative of its generalizability and biological underpinnings. Thus, biological mechanisms influencing impulse control have strong implications for the course and treatment of eating disorders, particularly the bingeing purging subtypes of anorexia and bulimia nervosa, binge eating disorder, and potential impulsive subtypes of the various diagnoses.

The present study examines the effects of resting SCL on the relationship between family conflict and later eating disorder behaviors. Seeing as certain types of family conflict have been implicated in the emergence of individuals' disordered eating behaviors, it is predicted that there would be a relationship between interparental aggression and/ or harsh parenting and eating disorder behaviors, such that individuals who experienced higher levels of family conflict will be more likely to engage in disordered eating behaviors. Secondly, considering the apparent implications that sympathetic activation has regarding impulse control, it is predicted that this relationship will be strengthened in individuals with low levels of baseline SCL as compared to those with average or above average levels.

Methods

Participants

The current study examined data available from 92 undergraduate students attending college at University at Albany, SUNY. Of the 92 participants, 25 were excluded due to equipment failure/malfunction or other reasons such as failing to fill out study measures or leaving the study early. The sample consisted of 25 males and 42 females, with a mean age of 20 (range 18-40).

Ethnic composition was 67.2% Caucasian, 13.4% Hispanic, 9.0% Asian, 6.0% Black or African-American, 3.0% Biracial, Unknown, and 1.5% other ethnic background.

Procedure

All procedures were approved by the University at Albany IRB. After consent procedures, participants were led into a room where they were given privacy to complete computer-based self-report measures. Once the first portion of surveys was completed, participants were led into another room containing the psychophysiological recording equipment (James Long Company, Caroga Lake, NY). To collect SCL, we attached Ag/AgCl electrodes, filled with isotonic citrate salt electrode gel with gel contact limited to a 1 cm diameter circle by double-sided adhesive collars, to the volar surfaces of the distal phalanges of the participant's non dominant hand. After ensuring that the psychophysiological recording equipment was working properly and the participant was comfortable, participants were asked to relax and sit still while a three-minute baseline response was collected. The second minute of the baseline was used for analysis because it allowed 1 minute for these measures to stabilize, and because during the third minute, although not examined here, blood pressure was taken which affected some participants' SCL.

Measures

Disordered Eating Behaviors. The Eating Attitudes Test -26 (EAT-26; Garner, Olmsted, Bohr, & Garfinkel, 1982) was utilized to identify participants who qualified as at risk of developing an eating disorder. This assessment is a widely used measure of symptoms and concerns characteristic of eating disorders. It has an internal consistency of .83. In the current sample the internal consistency was .86. The measure consists of 26 items, broken into three subscales: dieting (e.g. "I am aware of the calorie content in the food I am eating"), bulimia and

food preoccupation (e.g. “I have gone on eating binges where I fear I may not be able to stop”), and oral control (e.g. “I avoid eating when I am hungry”). All of the subscales were significantly correlated ($p < .05$) and therefore they were collapsed for analysis. Questions are answered using a forced choice, six-item, Likert scale. The EAT-26 has been shown to distinguish eating disordered populations from normal controls, identify those with eating disorders in nonclinical samples, and identify individuals who are at risk of disordered eating behaviors.

Harsh Parenting. Harsh parenting was measured using a slightly modified version of the Conflict Tactics Scale- Parent to Child (CTS-PC; Straus, Hamby, Finkelhor, Moore & Runyan, 1998). Just as the original CTS-PC did, the modified version asked participants to report parenting tactics used by their mother and father, however they were additionally asked to report tactics used by a stepmother, stepfather, mother’s other partner, father’s other partner, or a foster parent. This was done in an attempt to account more accurately for the participants’ diverse living situations. This 22-item, self report measure of parenting practices is comprised of five subscales: positive parenting, psychological aggression, minor physical assault, severe physical assault, and very severe physical assault. The three dimensions of physical assault are typically combined into an overall physical assault scale. Sample items assessing abuse include “This person hit me on the bottom with something like a belt, hairbrush, a stick or some other hard object” (physical aggression), “This person explained why something was wrong” (positive parenting), and “This person shouted, yelled, or screamed at me” (psychological aggression). These constructs are assessed using a six-point Likert scale (never happened, once, a few times, pretty often, very often, not applicable) to assess the frequency of parental behaviors. This adaptation of the scale from numerical counts of incidences to more general frequency categories reduces concerns regarding the participants’ ability to accurately report numerical frequencies.

The CTS-PC has been found to have an internal consistency of $\alpha=.55$ for overall physical assault, $\alpha=.55$ for psychological aggression, and $\alpha=.70$ for positive parenting (Straus et al., 1998). In our sample, Cronbach's α was .92 for total aggression, across all subscales, experienced during childhood, and .89 for total aggression, across all subscales, experienced during adolescence. Participants were asked to complete the CTS-PC for both childhood and adolescence. Childhood harsh parenting was defined as maltreatment experience occurring prior to age 13, and adolescent harsh parenting was defined as maltreatment experience occurring after age 13. To help ensure the accuracy of recall, participants were asked to think of a major life event that occurred around age 13 to use as an anchor point.

Interparental Aggression. Interparental aggression was measured using a Parent to Parent version of the Conflict Tactics Scale (CTS-2) (Straus, 2004; Straus, Hamby, Boney-McCoy, & Sugarman, 1996). This scale was slightly modified in order to better encapsulate the diverse living situations of the participants. Participants were asked to report retrospectively how often they saw their parents or guardians engaging in conflict, using two sets of 17 items, rated along the aforementioned Likert scale. The same anchor point memory from around age thirteen was used as in the CTS-PC. This measure consists of three subscales: psychological aggression (3 items) (e.g., "One screamed or yelled at the other"), physical assault (12 items) (e.g., "One pushed or shoved the other"), and injury (2 items) (e.g., "One passed out from being hit on the head by the other in a fight"). All subscale scores were summed to provide a total interparental aggression score during childhood and during adolescence. In order to account for the possibility of participants not being raised in a traditional two-parent household, for each aggressive act, the scale was modified to ask how often it was witnessed from each Mother to Father, Father to Mother, Mother to Other Partner, Other Partner to Mother, Father to Other

Partner, Other Partner to Father, and Foster Parent to Partner. Reliability coefficients, without our modifications, are between $\alpha = 0.79$ and 0.95 (Straus et al., 1996). In the current sample, Cronbach's α was $.90$ for total interparental aggression experiences, across all subscales, during childhood, and $.82$ for interparental aggression experiences, across all subscales, during adolescence.

SNS Activity. SNS activity was indicated by SCL during the second minute of the three-minute baseline recorded. To collect tonic SCL, the psychophysiological recording equipment used a 500mV , 30Hz sinusoidal excitation waveform yielding an SCL output of $10\ \mu\text{S/V}$. The A/D converter had a 16-bit resolution and a $\pm 2.5\text{V}$ input range and data were digitized at $1\ \text{kHz}$.

Results

Descriptive information

All statistical analyses were conducted using the SPSS 18.0 software (SPSS, Inc., Chicago, IL). Descriptive data for all study variables by gender appear in Table 1. Because baseline SCL, harsh parenting (both childhood and adolescent), interparental aggression (both childhood and adolescent), and eat26 scores were all substantially skewed, we log transformed and centered adolescent harsh parenting and interparental aggression, as well as childhood interparental aggression, and eat26 scores. Childhood harsh parenting and SCL were square root transformed and centered. The transformed and centered variables were used in all analyses. Table 1 displays both raw and transformed means for interpretability.

Bivariate Correlations

Bivariate correlations examined relations between SCL, harsh parenting, interparental aggression, and eating disorder behaviors (Table 2). Correlations between subscales of the eat26

were significant ($p < .01$). Analyses reveal that harsh parenting experienced during childhood or adolescence was significantly positively correlated with interparental aggression experienced during adolescence and childhood ($p < .01$). Also, interparental aggression experienced during childhood was significantly correlated with interparental aggression experienced during adolescence ($p < .01$), and harsh parenting experienced during childhood was significantly correlated with harsh parenting experienced during adolescence ($p < .01$). None of the variables were significantly correlated with eating disorder behaviors. Analyses of SCL covaried for the effects ethnicity due to the fact that SCL has been found to be lower among African-American samples (i.e., Anderson & McNeilly, 1991).

Regression analyses predicting disordered eating

We examined the effects of interparental aggression and harsh parenting experienced during childhood and adolescence, and baseline SCL in explaining disordered eating behaviors. Two models were examined, one looking at the effects of childhood experience on eating disorder behaviors, and the other looking at the effects of the adolescent experience. Interaction effects were calculated by multiplying the transformed and centered scores, and all predictors were entered simultaneously. The first model examined eat26 scores as a function of childhood harsh parenting, childhood interparental aggression, baseline SCL, and their two and three way interactions. The second model examined eat26 scores as a function of adolescent harsh parenting, adolescent interparental aggression, baseline SCL, and their two and three way interactions. Regression analyses for childhood experiences appear in Table 3 and regression analyses for adolescent experiences appear in Table 4. The analysis covaried for the effects of gender in the sample, as well as for ethnicity for the aforementioned reasons (i.e., Anderson & McNeilly, 1991). For these purposes, participants were coded as black so long as they had at

least one parent identified by the participant as being of African American or Caribbean descent. Covariates were non-significant in accounting for disordered eating behaviors.

The first model examined the main and interactive effects of childhood harsh parenting, childhood interparental aggression, and baseline SCL on disordered eating behaviors (measured by the eat26). Although the overall equation was not significant, there was a significant interaction effect between childhood harsh parenting and baseline SCL predicting eat26 scores. We probed this interaction according to the procedures described by Aiken and West (1991). At values of SCL 1 *SD* below the mean, the relationship between childhood harsh parenting and eating disorder behaviors was significant and positive ($b=.514, t= 2.518, p= .01$). At values of SCL 1 *SD* above the mean, the relationship between harsh parenting and eating disorders was negative and non-significant ($b=.237, t=1.294, p= .202$).

The second model examined the main and interactive effects of harsh parenting and interparental aggression experienced during adolescence and SCL on eating disorder behaviors. Although the overall model was not significant, a main effect was found such that SCL was predictive of disordered eating behaviors. Seeing as no further significant results were found, no further analysis was performed.

Discussion

The present study examined the relationship between interparental aggression, harsh parenting, and disordered eating behaviors. We also examined how this relationship was moderated by activation of the sympathetic nervous system in the form of basal SCL. Results indicate that sympathetic inhibition helps account for individual differences in the development of negative outcomes as a result of childhood harsh parenting. There was a significant interaction between harsh parenting experienced during childhood and SCL predicting disordered eating

behaviors. When this interaction was probed in accordance with procedures described by Aiken and West (1991), it was found that SCL moderated the relationship between harsh parenting and disordered eating behaviors such that the lower the level of basal skin conductance the stronger the relationship between harsh parenting and disordered eating. Also, in the adolescent model, a significant main effect of SCL on disordered eating behaviors was found. No significant relationship was found, however, between experiences of adolescent harsh parenting and disordered eating, nor was there evidence of interparental aggression leading to disordered eating, whether it was experienced in either childhood or adolescence. These findings partially support the hypotheses that family conflict would lead to higher levels of disordered eating behaviors, and that this relationship would be moderated by the sympathetic nervous system.

This finding can be understood in the context of the implications of decreased sympathetic activation for impulse control (Fowles et al., 2000). If an individual is not inhibited, they are more likely to engage in risky and self destructive behaviors such as excessive dieting or purging after meals as a result of their increased difficulty suppressing impulses. This occurs despite knowledge that these behaviors are harmful. This finding extends the results of previous studies which focused predominantly on the effect of SCL on aggression and depression (Beauchaine, 2001; El Sheikh, 2009; Raine, 2005). The current finding also serves to clarify some of the inconsistencies surrounding the effects of family conflict on eating disorders. The fact that interparental aggression was not found to be predictive of any disordered eating behaviors is very interesting, and is consistent with the aforementioned literature on family conflict and eating disorders that found no significant relationship (Sim et al., 2009; Spanos, et al., 2010; Wade et al., 2007). It also suggests that there is something particularly important about the effect of conflict directed at the child that influences the development of disordered eating

behaviors, as consistent with the findings of Grilo & Masheb (2001), Johnson et al. (2002), and Klemmeden-Fosse & Holen (2005). Also, the fact that that harsh parenting experienced during the teen years was not predictive of disordered eating suggests that there is also a time frame for which an individual may be particularly susceptible to developing negative effects of maltreatment. Future research needs to delve more thoroughly into varying types of conflict as well as the ages at which it occurs in order to develop a more lucid and comprehensive conceptualization of the effects. Also, it is worth noting that the Dieting subscale of the eat26 was also significantly predicted by harsh parenting experienced during childhood. This may be a result of the fact that strict dieting and restricting have been found to lead to overeating (Polivy & Herman, 1998; Smith, Williamson, Bray, & Ryan, 1999). The dieting and restricting may be the individual's attempt to control their impulse, often culminating in a binge when the person can restrict no longer. This may be particularly relevant within normative, college aged samples (Mobbs et al., 2008).

This study was limited in that our sample was taken from a university, suggesting that the participants were likely of higher socioeconomic status and more highly educated than the general population. Also, the sample was primarily Caucasian, and thus generalization to other ethnicities may be limited. In addition, our sample also represented a normative population rather than a clinical one, so the generalization those clinically diagnosed with eating disorders is limited. Another limitation is the fact that behavioral inhibition has been accounted for in the literature using various measures including SCL, heart rate, reward and punishment sensitivity, the BAS/BIS. As a result, it is difficult to be certain they are all measuring exactly the same construct. Also, seeing as for the purposes of this study we covaried by gender, the findings do not differentiate between males and females. It would be beneficial to test the hypothesis again in

a sample with a large amount of participants of each gender to see if this finding is at all different in either. Similarly, seeing as neither vagal tone nor SCL reactivity to a stressor were examined in this study, the potential effects of those mechanisms on eating disorders should be explored. Moreover, the measures of harsh parenting and interparental aggression were both retrospective, and therefore may be subject to recall bias. Future research should consider reassessing these constructs longitudinally.

Clinical Implications

Fully understanding the individual factors contributing to a problem is crucial in determining proper treatment. If the problem has a biological component, in this case low levels of basal SCL, treatment that focuses merely on correcting the symptomatic behaviors will not be as effective as one that is more comprehensive. Boyce et al. (2001) suggested that screening children based on neurobiological markers in advance of symptoms may be a useful way to decrease the incidence of psychopathology. The study found that patterns of biological mechanisms, such as the interacting of the SNS and PNS, were not only predictive of children having symptoms, but could discriminate between children with externalizing symptoms and internalizing symptoms. Similarly, Claes and colleagues (2010) posited that they could differentiate between eating disorder types based on measures of BIS activation and effortful control. The ability to identify different characteristics within a disorder allow for treatment to be better suited to that individuals personal needs.

This idea is supported by the finding of Cavedini (2006) that participants with the impulsive behaviors did not respond as well to treatment as those who had the eating disorder alone. An individual who is fully aware that their behaviors are out of control and still cannot stop may benefit more from treatment focused on building and maintaining impulse control. Fay

and Eisler (1993) found no significant difference in treatment outcome based on the level of impulsivity, however if those who were struggling with impulse control had undergone treatment targeting it, they may have shown even more improvement. Further, the fact that there is a biological influence suggests that psychopharmacology geared toward these mechanisms would be useful to explore. Skin conductance level has been found to be consistent over time (El Sheikh, 2007) however if sympathetic arousal could be kept at a health baseline by way of a drug it may make it easier for the individuals to gain control of the symptoms of their disordered eating.

The present study emphasizes the importance of examining the individual biological factors that contribute to psychopathology. Future research should continue to examine the implications of decreased sympathetic activation on varying forms of psychopathology. Also, SCL reactivity as well as the interaction between the SNS and the PNS should be examined to establish how they pertain to disordered eating symptomology. Finally, research should continue to examine the time specific and type specific effects of family conflict on the development of disordered eating behaviors.

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Table 1

Means and SDs of study variables by gender

	Non- Transformed		
	Females (n=42)	Males (n= 25)	F (gender)
EAT-26	5.72(5.75)	6.24(4.73)	2.273 ^a
Baseline SCL	13.09 (6.16)	13.53(5.05)	.293 ^b
Childhood Harsh Parenting	19.58(14.06)	15.54(12.00)	.067 ^c
Adolescent Harsh Parenting	6.00(10.94)	6.98(11.43)	.987 ^d
Childhood Interparental Aggression	1.52(2.75)	1.83(2.66)	.962 ^e
Adolescent Interparental Aggression	1.80(2.78)	1.70(2.39)	.016 ^f
	Transformed		
EAT-26	.6720(.39318)	.7481(.34608)	3.197 ^a
Baseline SCL	3.5062(.91210)	3.7936(1.48431)	.434 ^b
Childhood Harsh Parenting	4.2446(1.64448)	3.7936(1.48431)	.000 ^c
Adolescent Harsh Parenting	.4532(.56677)	.5817(.53992)	1.746 ^d
Childhood Interparental Aggression	.2447(.34137)	.3078(.34245)	1.116 ^e
Adolescent Interparental Aggression	.2999(.34350)	.2863(.34435)	.017 ^f

Note: EAT = Eating Attitudes Test, SCL= Skin conductance level, HP= harsh parenting. PP= interparental aggression. ^aFor *F*-test, df= 1,83. ^bFor *F*-test, df= 1,76. ^cFor *F*-test, df= 1,65.

^dFor *F*-test, df= 1,68. ^eFor *F*-test, df= 1,81. ^fFor *F*-test, df= 1,80.

Table 2

Correlations

	1	2	3	4	5	6
1. HP- Child	-					
2. HP- Adolescent	.571**	-				
3. PP- Child	.543**	.345**	-			
4. PP- Adolescent	.541**	.320**	.809**	-		
5. EAT	.097	-.006	-.026	.136	-	
6. Baseline SCL	-.074	-.028	-.111	-.122	-.185	-

Note: HP= Harsh parenting, PP= Interparental aggression, EAT= Eating attitudes test, SCL=

Skin conductance level.

** $p < .01$

Table 3

Regression analyses accounting for disordered eating from childhood harsh parenting, interparental aggression, SCL, and their interactions

	β	t	F	R^2
EAT-26				
Total Equation	-	-	1.593	.174
HP	.139	1.034	-	-
PP	-.124	-.941	-	-
SCL	-.170	1.242	-	-
HP x PP	.007	.050	-	-
HP x SCL	-.349	-2.680*	-	-
PP x SCL	.019	.145	-	-
HP x PP x SCL	.074	-.510	-	-

Note: EAT-26 = Eating attitudes test, SCL= Baseline skin conductance level, HP= harsh parenting, PP= Interparental Aggression. Covariates included ethnicity and gender

* $p < .05$. For F -test, $df=7$.

Table 4

Regression analyses accounting for disordered eating from adolescent harsh parenting, interparental aggression, SCL, and their interactions

	β	t	F	R^2
EAT26				
Total Equation	-	-	1.505	.158
HP	.049	.354	-	-
PP	-.073	-.550	-	-
SCL	-.322	-2.356*	-	-
HP x PP	-.181	-1.319	-	-
HP x SCL	-.212	-1.559	-	-
PP x SCL	.105	.785	-	-
HP x PP x SCL	-.100	-.708	-	-

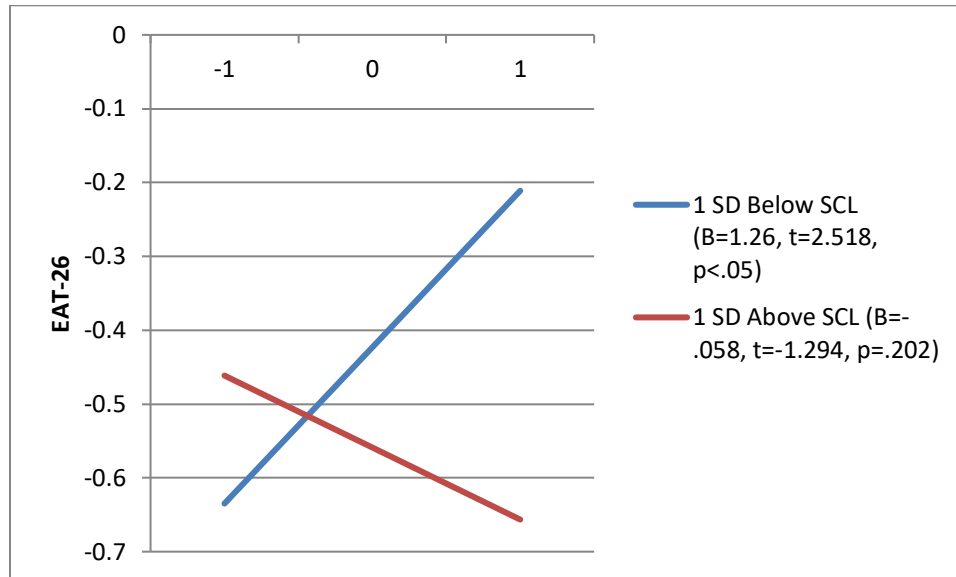
Note: EAT= Eating attitudes test, SCL= Baseline skin conductance level, HP= harsh parenting,

PP= Interparental Aggression. Covariates included ethnicity and gender

* $p < .05$. For F -test, $df=7$.

Figure 1

Probing a significant three way interaction between harsh parenting, skin conductance level, and disordered eating behaviors



Note: EAT-26= Eating Attitudes Test, SCL= Skin conductance level, SD= Standard deviation