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Behavioral and Psychophysiological Responsiveness During Child Feeding in Mothers with Histories of Eating Disorders: A Pilot Study

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Abstract

The aim of this pilot project was to describe maternal responsiveness during child feeding in mothers with eating disorder histories through the combined use of observational, self-report, and physiologic methods. For this non-randomized cohort pilot study, 25 mothers with histories of eating disorders and 25 mothers with no history of an eating disorder with children ages 6–36 months were selected such that the groups were similar based on child age group (within 6 months) and child sex. Maternal behavioral responsiveness to child cues was assessed by video-recording and behavioral coding of both a free-play and feeding episode. Physiologic engagement was assessed through measurement of respiratory sinus arrhythmia (RSA) reactivity during free-play and feeding episodes. No differences were detected in observed behavioral responsiveness during feeding or free-play in mothers with eating disorder histories compared with controls. Mothers with eating disorder histories did report more parenting stress, increased anxiety, and exhibited a blunted physiologic stress response (less RSA reactivity) during both feeding and free-play interactions with their children. These results support future larger-scale investigations of RSA reactivity in mothers with eating disorders.

Keywords

eating disorders; mothers; feeding behavior; maternal responsiveness; RSA reactivity; infant feeding

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INTRODUCTION

Maternal Eating Disorders and the Feeding Environment

Children of mothers with eating disorders [anorexia nervosa (AN), bulimia nervosa (BN) and some forms of eating disorder not otherwise specified (EDNOS)] are at greater risk for infant feeding problems and childhood disordered eating than children of mothers without eating disorders (Easter et al. 2013; Micali et al. 2009; Bulik et al. 2005; Whelan and Cooper 2000; Stice et al. 1999; Stein and Fairburn 1989; Brinch et al. 1988). These children also exhibit greater dietary restraint and concern about shape and weight at a young age compared with children of mothers without an eating disorder (Lombardo et al. 2012; Woodside and Shekter-Wolfson 1990). Several pathways have been suggested to explain why the offspring of mothers with eating disorders may be at an increased risk of developing an eating disorder themselves. One proposed pathway is through passive gene-environment correlation (Plomin et al. 1977), whereby these children may inherit genes that place them at greater risk for developing eating disorders (Clarke et al. 2012; Thornton et al. 2011), and they may also be inadvertently exposed to disordered eating attitudes and behaviors in their home environment that could further exacerbate risk (Watkins et al. 2012; Striegel-Moore and Bulik 2007). Independent of genetic predisposition, it is critical that we understand which environmental factors may be influencing the development of healthy eating attitudes and behaviors in the children of mothers with eating disorders in order to help prevent future disordered eating.

Indeed, mothers with eating disorders self-report more controlling (restrictive or pressuring) feeding styles marked by close monitoring of child food consumption and overt control of types or quantities of foods consumed rather than responsive feeding based on child feeding cues (Blissett and Haycraft 2011; Reba-Harrelson et al. 2010; Mazzeo et al. 2005; Duke et al. 2004; Agras et al. 1999). These findings are particularly concerning in light of prospective studies demonstrating that early restrictive feeding practices can lead to eating in the absence of hunger, or dysregulated eating, and higher BMI in later childhood (Anzman and Birch 2009; Fisher and Birch 2002). Direct observational studies of the feeding environment in mothers with eating disorders have found that mealtimes are marked by increased conflict, disorganization, more negative emotions exhibited by both mothers and infants, and fewer positive comments about food and eating compared with control mother-infant dyads (Cooper et al. 2004; Waugh and Bulik 1999; Stein et al. 1994), but no studies have directly evaluated responsive feeding practices in mothers with histories of eating disorders.

Maternal Responsiveness During Feeding

Feeding responsiveness refers to a mother's ability to respond appropriately to her child's hunger and satiety cues. Decreased maternal responsiveness and increased control in the early feeding relationship have been associated with increased eating in the absence of hunger later in childhood (Anzman and Birch 2009; Fisher and Birch 2002), which is thought to contribute to childhood overweight (DiSantis et al. 2011). Low responsiveness during feeding has also been associated with child underweight, as mothers of children with non-organic failure to thrive were observed to be less nurturant with fewer interactions during mealtimes than mothers of comparison children; however the lack of prospective studies in this area makes it unclear if this behavior is causal or simply a response to their infant's poor feeding behavior (Robertson et al. 2011).

Tronick & Gianino (1986) propose that the infant has some emotional self-regulatory capacity in response to perturbations, such as hunger, but this capacity is immature and requires a responsive caregiver within a caregiver-infant dyadic system to recognize the

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source of distress and respond appropriately. Among individuals with eating disorders, lower levels of awareness of the emotional states of others has been reported(Harrison et al. 2009; Zucker et al. 2007; Bydlowski et al. 2005). Furthermore, individuals with eating disorders have been found to have disturbances in their recognition of and response to their own internal hunger and satiety cues(Holsen et al. 2012; Kaye et al. 2011; Pollatos et al. 2008). It stands to reason that if caregivers have diminished capacity to recognize hunger and satiety in themselves, they may have trouble recognizing these states in their infants. Indeed, mothers with eating disorders or body dissatisfaction have been found to be more controlling and to exhibit less flexibility when feeding their children than control mothers(Cooper et al. 2004; Duke et al. 2004). These mothers may also find it difficult to be responsive to their child's hunger and fullness cues during feeding due to heightened anxiety at mealtimes, fears about child weight gain, as well as social cognitive styles influencing their ability to appraise cues (Zucker et al. 2007; Mazzeo et al. 2005).

Physiologic Support for Maternal Responsiveness

Although the literature on responsiveness during feeding for mothers with histories of eating disorders is limited, previous studies of maternal response to infant cues during other challenging situations (i.e., laboratory tasks designed to elicit negativity and distress from both mother and child) suggest that successful maternal regulation (both behavioral and physiological) is critical in order to appropriately attend to, interpret, and respond to the needs of her infant (Mills-Koonce et al. 2007; Mills-Koonce et al. 2009). The quality of a mother's response to her child's needs may depend *in part* on the degree to which physiological regulation supports active attention and engagement with the needs of the child. Indeed, maternal reactivity and regulation, both of which have known biological bases in the autonomic and central nervous systems (Rothbart and Derryberry 1981), have been linked to individual differences in the way mothers respond to their infants' signals. Moreover, there is ample evidence within the animal literature that physiological support is necessary for organized parenting behavior [e.g., (Boccia and Pedersen 2001; Winslow et al. 2000; Porges 1998; Wotjak et al. 1996)].

Measuring Physiologic Responsiveness Using RSA

Measures of the parasympathetic branch of the autonomic nervous system (indexed by cardiac vagal tone or the neural control of the heart via the vagus nerve) provide an assessment of an individual's ability to regulate physiological state in the face of a stressor (Porges 2009, 2007) and have been related to self-regulation, temperament, affect, attention, and underlying regulatory abilities in mammals (Diamond et al. 2011; Ochsner and Gross 2008; Ottaviani 2008; Bornstein and Suess 2000). Respiratory sinus arrhythmia (RSA), or high frequency heart rate variability (rhythmic oscillations in the heart rate pattern), is a measure of beat to beat heart period associated with respiration (Porges 1996; Porges and Byrne 1992). RSA is frequently used as an index of cardiac vagal tone or even as a direct measure of vagal tone (Grossman and Taylor 2007), even though there are many other influences and this is only one component. Of these various influences, however, RSA has been the most consistently examined in relation to dimensions of behavioral functioning in humans. For example, higher resting RSA has been related to several measures of cognitive and emotional processes involving attention and stimulus appraisal, self-regulation, affect, and attention (Oveis et al. 2009; Gyurak and Ayduk 2008; Sack et al. 2004; Kettunen et al. 2000).

Baseline RSA is considered to be a stable neurophysiological mechanism underlying autonomic and behavioral reactivity that provides a measure of resting state in the absence of environmental challenge. Under most conditions, heart rate is kept low and heart rate variability high via the nucleus ambiguus serving as a vagal "brake" in order to curb the use

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of psychophysiological resources. However, under challenging conditions, this "brake" is released and the autonomic nervous system increases metabolic output to deal with external demands; the resulting decrease in heart-rate variability and increase in cardiac output facilitates active engagement and monitoring with respect to a set goal(Porges 2009). This decrease in RSA (or RSA withdrawal) typically occurs when an individual is involved in an activity that requires active coping (Butler et al. 2006; Grossman and Svebak 1987). Furthermore, it has been found to index self-regulation and may underlie the ability to appropriately engage the source of stimulation or take distance from it in both children (Hastings et al. 2008; T. P. Beauchaine et al. 2007) and adults (Butler et al. 2006; T. Beauchaine 2001).

RSA in Mothers and Individuals with Eating Disorders

It has been demonstrated that mammalian pair-bonding is supported by the vagal system, which physiologically mediates social engagement, facial expressivity, and vocalization (Porges 1998). Thus, as part of the broader caregiving system, maternal vagal regulation in response to infant signals may provide a psychophysiological support system for the organization of effective parenting behaviors. Studies of mother-child interactions have revealed that elevated maternal RSA withdrawal is associated with the maintenance of sensitive care even under conditions of high child distress (Oppenheimer et al. 2013; Mills-Koonce et al. 2009; Hill-Soderlund et al. 2008; Mills-Koonce et al. 2007). Most relevant to the current study, clinical data evaluating vagal tone in women with eating disorders indicate that these women exhibit decreased physiologic stress reactivity (Messerli-Burgy et al. 2010; Lachish et al. 2009; Murialdo et al. 2007), conceivably interfering with appropriate responding during stressful situations such as meals.

Therefore, one aim of the current study was to examine the physiological response (via the parasympathetic branch of the autonomic nervous system) in mothers with eating disorders to interactions with their young children during feeding time—a particularly challenging or stressful experience for this population. This represents the first step in understanding the biological mechanisms contributing to maternal behaviors that may influence children's risk for future disordered eating.

The objective of this pilot study was to describe maternal responsiveness comprehensively in mothers with histories of eating disorders relative to control mothers during both mealtime and playtime through the combined use of observational, self-report, and physiologic methods. As a complement to behavioral coding of maternal-child interactions, we asked mothers to wear an ambulatory electrocardiogram (ECG) during the observation to assess baseline RSA and RSA withdrawal in response to mealtime and playtime interactions. As discussed previously, RSA reactivity is a commonly used measure of physiologic support for emotional engagement and response to environmental stress, yet no studies to date have evaluated RSA reactivity in mothers with histories of eating disorders during emotionally challenging tasks such as mealtimes. Our hypotheses related to maternal responsiveness were threefold: (1) For self-report measures, we hypothesized that mothers with histories of eating disorders would report decreased parenting self-efficacy and increased parenting stress. (2) For observational measures, we hypothesized that mothers with histories of eating disorders would be less responsive to child cues during feeding but not during play. (3) For physiologic measures, we hypothesized that the challenge and stress of mealtime interactions for mothers with histories of eating disorders would result in increased physiologic support for engagement, manifested as increased RSA withdrawal compared to control mothers. Our goal for this pilot study was to demonstrate sufficient evidence supporting larger studies of responsiveness during feeding in mothers with eating disorders.

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METHOD

Participants

The study was approved by the Biomedical Institutional Review Board of the University of North Carolina at Chapel Hill, and all participants provided informed consent. Twenty-five women with a history of an eating disorder with children between the ages of 6 and 36 months were recruited from the community through emails and fliers distributed to university students and staff, mother's clubs, and local daycare centers. A phone screening was conducted, and eligible participants were then interviewed using the Structured Clinical Interview for DSM-IV-Patient Edition (SCID-I/P)(First et al. 2002) to assess for previous history of either AN, BN, or EDNOS. Participants must not have met threshold criteria for AN, BN, or EDNOS in the past 28 days, and must have maintained a BMI of at least 18.5 kg/m^2 for three months prior to participation. Women with a history of only binge eating disorder (BED) or a BMI >30 kg/m^2 were not included in this study due to published differences in feeding patterns in these groups compared to women with AN, BN, and EDNOS (Reba-Harrelson et al. 2010; Faith et al. 2004; Birch and Fisher 2000).

Twenty-five control mothers with no history of eating disorders were similarly recruited from the community, screened over the phone, and interviewed using the SCID-I/P. Eligible mothers who had not met lifetime DSM-IV criteria for AN, BN, BED, or EDNOS according to the SCID-I/P and who had a BMI between 18.5 and 30 kg/m^2 were recruited based on child age group (within 6 month age bands) and child sex. Although participants were not individually matched in a pairwise way, they were recruited such that both groups had the same proportion of child age groups and child sexes.

Exclusion criteria were psychosis, including schizophrenia, bipolar I disorder, current suicidal ideation, alcohol or drug dependence in the past year, and any social service inquiries regarding child neglect. No women were excluded based on these criteria.

Protocol

Two members of the research team visited all participants in their home during a typical feeding time. Upon arrival at the home, study staff positioned two digital camcorders (one to capture the child and the second to capture the mother) on swiveling tripods just outside the room where the feeding interaction would take place and interacted informally with mother and child for a 10–15 minute warm-up period to minimize intrusiveness and reactivity. After the warm-up period, the ECG monitor was put on mothers, and a baseline measure of heart rate was recorded for two minutes. The monitor stayed on mothers throughout the remainder of the visit for continuous recording of heart rate, with start and end times for each task marked within the data. The videographers filmed both mother and child from before the meal (including food preparation) until at least two minutes after the last bite was taken. To minimize intrusiveness, the videographers were instructed to leave the room during any periods when either mother or child was stationary (e.g., the child was in a highchair). Either before or after the meal, mothers were also filmed during ten minutes of free play with their children when they were instructed to play as they typically would in their home. Upon completion of the home visit, mothers were given a packet of questionnaires to complete over the following week and return to the study coordinator in the mail. There was a 100% completion rate from eligible participants.

Clinical Measures

The SCID-I/P was conducted by a clinical interviewer trained to criterion to assess eating disorder history and comorbid Axis I disorders (alcohol or drug dependence and psychosis including schizophrenia and bipolar I disorder). After the home visit, participants completed

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the Eating Disorder Examination Questionnaire (EDE-Q)(Fairburn and Beglin 1994), the Beck Depression Inventory-II (BDI-II)(Beck et al. 1996), and the Beck Anxiety Inventory (BAI)(Beck et al. 1988). The EDE-Q is a well-studied and frequently used 36-item self-report questionnaire designed to assess eating disorder symptoms over the previous 28 days(Fairburn and Beglin 1994). The questionnaire yields four subscale scores (Dietary Restraint, Eating Concern, Shape Concern, and Weight Concern) as well as a Global score, which is the average of the four subscales. Both internal consistency (α= 0.78–0.93) and two-week test-retest reliability (Pearson r= 0.81–0.94) are high for the subscales of the EDE-Q (Luce and Crowther 1999). The BDI-II is a 21-item self-report questionnaire that assesses the severity of current depressive symptoms; the revised version extends the reporting time frame to two weeks (Beck et al. 1996). Good internal consistency was demonstrated in a sample of postpartum mothers completing the BDI-II (α= .94) (Britton 2011) and one week test-retest reliability is high (r= 0.93) (Beck et al. 1996). The BAI is a 21-item self-report questionnaire that assesses current anxiety, focusing on somatic symptoms of anxiety over the past week(Beck et al. 1988). In a sample of psychiatric outpatients, the BAI had high internal consistency (α= 0.92) and test–retest reliability over one week (r= 0.75) (Beck et al. 1988).

Participants' weight was assessed during the home visit using a digital scale (Tanita HD-351 Digital Weight Scale); scales were calibrated regularly according to protocol. A stadiometer was used to assess all participants' height. BMI was then calculated as weight (kg)/(height (m))^2. Children were weighed wearing only a clean diaper using a digital scale (Tanita BD-585 Digital Baby Scale). Child length was measured three times in the recumbent position using a calibrated length board (O'Leary Length Board) by two trained research staff, and the average of the three measurements was recorded. Child weight and length was then converted into age and sex-specific z-scores per the revised growth charts from the Centers for Disease Control and Prevention (Kuczmarski et al. 2000).

Parenting Measures

The Parenting Stress Index-Short Form (PSI/SF) (Haskett et al. 2006) and the Parenting Sense of Competency Scale (PSOC) (Johnston and Mash 1989) were administered to evaluate participants' sense of stress and confidence related to parenting. The PSI/SF is a 36-item self-report questionnaire that yields three subscale scores of parental distress, parent-child dysfunctional interaction, and difficult child in addition to an overall parenting stress score. Participants must choose the degree to which they agree with statements regarding their own sense of parenting stress on a five-point scale (1=strongly disagree to 5=strongly agree). Items are summed to create subscale scores; higher scores represent greater parenting stress. The PSI-SF has good test-retest reliability over a period of one year (r= 0.75 for overall score); subscales are internally consistent (α= 0.74–0.88) and are correlated with measures of parent psychopathology, parent perception of child adjustment, and observed parent and child behavior (Haskett et al. 2006). The PSOC was used in this study for its subscale of parenting self-efficacy, which assesses a parent's perceived competence, problem-solving ability, and capability in the parenting role. Participants respond to statements regarding their own confidence in parenting on a 6-point scale (1=strongly agree to 6=strongly disagree). Subscale scores are means of all items loading onto that sub scale; higher scores represent greater parenting efficacy. Good internal consistency has been demonstrated for the efficacy subscale of the PSOC in a large community-based sample of parents in Canada (α= 0.76) (Johnston and Mash 1989).

Observational Measures

The Responsiveness to Child Feeding Cues Scale (RCFCS) (Hodges et al. 2008) was used to code the videotaped mealtime interactions for maternal feeding responsiveness by two

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trained and reliable coders blind to participant group membership. This scale provides scores of maternal responsiveness to child *hunger cues* (coded until the mother begins to prepare food), *receptiveness cues* (coded from food preparation until one minute following the first bite), and *fullness cues* (coded from one minute after the first bite until food is removed) by assessing the presence, frequency, duration, and strength of feeding cues in children in concert with the timing and appropriateness of mothers' responses to these cues. Maternal responsiveness to hunger, receptiveness, and fullness cues subscale scores range from 1–5 with 1 being highly unresponsive and 5 being highly responsive. Because home visits were scheduled at typical mealtimes, and most mothers began to prepare food immediately once filming began, child hunger cues were not captured, and maternal responsiveness to hunger cues could not be evaluated in this study. Twenty percent of the videos were double-coded and agreement was high. Inter-rater intraclass correlation coefficients (ICC) are not presented for these variables, as there was little variability in scores on the RCFCS. Most mothers received a score of 4 or 5 by both coders indicating a high degree of maternal responsiveness during feeding. Thus, it was not appropriate to calculate intraclass correlations (designed for continuous variables or variables with a wide range of ordinal responses) for these two variables, but descriptive statistics of rater agreement are given to provide the reader with a sense of inter-rater reliability. Raters were in exact agreement for 60% of scores of responsiveness to receptiveness cues and 40% of scores of responsiveness to fullness cues. Ratings were in agreement within one point for 90% of scores of both responsiveness to receptiveness and responsiveness to fullness cues.

The ten-minute free play interactions were coded for maternal sensitivity by two trained and reliable coders blind to participant group membership according to a coding system used by the National Institute of Child Health and Human Development Study of Early Child Care (Appelbaum et al. 1997). A composite maternal sensitivity score was generated as a sum of five subscales of sensitivity/responsiveness, positive regard, stimulation of cognitive development, animation, and detachment/disengagement (subtracted) all scored on a scale from 1 to 5. Twenty percent of the videos were double coded, and inter-rater ICC's were calculated per convention with this coding scheme. ICC values for the five subscales within the maternal sensitivity composite ranged from 0.90 to 1.00.

RSA Withdrawal

Before filming began, electrodes were placed on the mother's chest in a modified lead-II configuration (concealed under her clothing) and connected to an ambulatory ECG logger (BM-4, Biomedical Monitoring, Ltd) that clipped to her waist. After the acclimation period, a baseline ECG recording was taken with the mother sitting in silence for two minutes. The ECG continued to record throughout both meal and play times, and research staff recorded both start and end times for each task (baseline, meal, and play).

A data file containing the ambulatory ECG recordings over the entire observation period was transferred to a computer for manual editing of artifacts that resulted from excess movement and calculation of heart inter-beat intervals (IBI) using Nevrokard heart rate variability software (Nevrokard Kiauta, Slovenia). Two data files that required editing of more than ten percent of the data were not included in the analyses. After editing and processing the ECG files, measures of RSA were extracted using Porges' method (Porges 1995). This procedure applies an algorithm to the sequential IBI data using a moving 21-point polynomial to detrend periodicities in heart period slower than RSA. Then, a band-pass filter extracts the variance of the IBIs within the frequency band of spontaneous respiration in adults (0.12–0.40). This estimate of RSA is derived by calculating the natural log of this variance and is reported in units of $\ln(ms^2)$. RSA was calculated in 15 second epochs during the baseline recording and in 30 second epochs during both meal and play

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periods. Meal periods ranged from 6 minutes to 43 minutes with no significant difference in meal time between groups ($t(43)= 0.52$, $p<0.60$), while play periods were all 10 minutes. Vagal tone was indexed by calculating the mean RSA of the epochs within each task (baseline, meal, and play); larger values of RSA indicate greater mean vagal tone. The difference in RSA from baseline provides a measure of RSA withdrawal during each task.

Statistical Analyses

All analyses were performed with SAS version 9.2(*SAS/STAT® Software: Version 9.2*). Means and standard deviations are presented for all continuous variables and percent distribution for categorical variables. Differences between groups were evaluated with student's *t*-tests for continuous variables and Fisher's exact test for categorical variables. Standardized effect sizes are presented as Cohen's *d* for continuous variables. A repeated measures analysis of variance (ANOVA) was used to determine the effect of group (eating disorder versus control), task (mealtime versus playtime), and any group × task interaction on the outcome variable of RSA change (RSA withdrawal). We used analysis of covariance (ANCOVA) to evaluate the impact of one covariate of interest at a time (our relatively small sample size precluded inclusion of multiple covariates in the same model) on the effect of group (eating disorder versus control) on outcome variables of maternal responsiveness to receptiveness and fullness cues, global sensitivity, and RSA change (RSA withdrawal). All significance tests were two-tailed, and a *p*-value of <0.05 was considered significant. In these analyses, women with histories of AN, BN, and EDNOS were evaluated together as a single eating disorder group. This concession was made given that it was a pilot study with a small sample size and is somewhat justified given that similar parenting concerns and general feeding patterns have been reported in mothers with AN, BN, or both (Mazzeo et al. 2005; Patel et al. 2002; Agras et al. 1999; Woodside and Shekter-Wolfson 1990). Many of the studies in this area have also been limited by small sample sizes making comparisons across various eating disorder diagnostic groups difficult. Post hoc follow-up comparisons between mothers with a history of AN only and BN only were conducted on most measures (individuals with a history of more than one eating disorder diagnosis were excluded from these analyses), and effect sizes of the differences are presented; however, due to the small sample size within these subgroups, we lacked the power to detect anything but very large effect sizes, so these comparisons are largely exploratory. Furthermore, to account for multiple comparisons, *p*-values of these post hoc tests were adjusted using the Bonferroni correction method.

One participant was missing data for several demographic variables due to her choice not to disclose information about current weight or level of education. Five participants were missing cardiac data due to either: 1) cardiac recordings that required editing of more than ten percent of the file (two mothers with histories of eating disorders), 2) cardiac recordings that were incomplete due to child or mother interfering with an electrode during the observation (one mother with history of an eating disorder and one control mother), or 3) a participant was not comfortable wearing the ambulatory ECG device (one mother with history of an eating disorder). A post-hoc analysis was conducted comparing those participants with missing heart rate data to the remainder of the sample using the Bonferroni method to correct for multiple comparisons. There were no significant differences in maternal age ($p=1.00$), current BMI ($p=0.71$), current eating disorder symptoms measured with the EDE-Q ($p=1.00$), maternal depression ($p=1.00$), maternal anxiety ($p=1.00$), self-reported parenting self-efficacy ($p=1.00$), self-reported parenting stress ($p=0.31$), observed maternal responsiveness to receptiveness cues ($p=1.00$) or fullness cues ($p=1.00$), or observed maternal sensitivity ($p=1.00$) in those with missing heart rate data compared to the remainder of the sample.

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A sample size of 25 participants per group was chosen for this pilot study in order to have 80% power to detect a large effect [effect size of 0.8 at =0.05 (two-tailed)] (*SAS/STAT® Software: Version 9.2* ; Cohen 1992). Given that this was a small study that was hypothesis driven but also intended to generate hypotheses for future larger studies, it was not reasonable to expect that the study would have power to detect medium or small effect sizes.

RESULTS

Characteristics of Mothers and Children

The sample was comprised of 50 mothers of children ages 6–36 months (25 with a history of an eating disorder and 25 controls). Thirteen of the 25 mothers with a history of eating disorder had a history of AN (9 restricting type and 4 binge-purge type), 13 had a history of BN (6 non-purging type and 7 purging type), 2 had a history of BED (in addition to another past eating disorder diagnosis), and 2 had a history of EDNOS (both met all criteria for AN except their lowest BMI was >18.5 kg/m^2). Three women had a history of two different eating disorder diagnoses (AN and BN, AN and BED, BN and EDNOS), and one woman had a history of three different eating disorder diagnoses (AN, BN, and BED). Mean age of onset for eating disorder was 17.46 ± 4.77 years.

Characteristics of participating mothers and their children are presented in Tables 1 and 2. The two groups did not differ significantly on their level of education, race, parity, marital status, or current BMI. The majority of mothers in both groups was white and had at least a college degree. Mothers with histories of eating disorders were significantly older (32.72 years ± 4.61) than control mothers (29.68 years ± 1.99; $t(48)=3.02$, $p<0.005$). In terms of clinical characteristics, mothers with histories of eating disorders had significantly higher scores on current eating disorder symptoms on the EDE-Q compared with control mothers, but scores in both groups were lower than published scores of individuals with current eating disorders (Mond et al. 2004). The groups did not differ significantly on current symptoms of depression measured with the BDI-II, but mothers with histories of eating disorders reported greater anxiety on the BAI (4.56 ± 3.80) than control mothers (2.58 ± 1.80; $t(48)=2.95$, $p<0.005$). Children of mothers with histories of eating disorders did not differ significantly from children of control mothers in their current weight-for-length z-score (Table 2).

The frequency of several common complications during pregnancy were compared and groups did not differ significantly on number of mothers with gestational diabetes, vaginal bleeding, urinary tract infection, problems with placenta, premature labor, premature rupture of membranes, or child receiving neonatal intensive care. Further, these complications were relatively uncommon in this sample with no more than three mothers reporting each of the above complications. Mothers with history of an eating disorder were significantly more likely to report gestational hypertension than control mothers (6 mothers with histories of eating disorders versus 0 control mothers; FET $p<0.03$). No mother in either group reported severe nausea/vomiting/dehydration, incompetent cervix, need for blood transfusion, or trauma during pregnancy.

Parenting Stress and Self-Efficacy

Mothers' self-reported parenting stress and parenting self-efficacy scores are presented in Table 3. While mothers did not differ in terms of their perceived competence, problem-solving ability, and capability in the parenting role (parenting self-efficacy) reported on the PSOC, mothers with histories of eating disorders reported experiencing greater stress related to parenting on the PSI/SF. The greatest differences between groups in self-reported parenting stress were on subscales of parental distress and parent-child dysfunctional

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interaction (Cohen's *d* effect sizes of 0.65 and 0.71 respectively), but groups did not differ on the difficult child subscale. Post hoc comparison of mothers with histories of AN only and BN only revealed no significant difference between eating disorder diagnoses in reported parenting stress (Cohen's *d* effect sizes of 0.01–0.24) or parenting self-efficacy (Cohen's *d*=0.07).

Maternal Responsiveness During Feeding

Results of the behavioral coding of both mealtime and playtime interactions are presented in Table 4. We did not detect a significant difference between groups in mothers' responsiveness to either receptiveness or fullness cues during feeding. The mean scores of responsiveness for both groups ranged from 3.56 – 4.56 reflecting moderate (3) to high (5) responsiveness during feeding. We evaluated covariates of maternal age, BDI score, and BAI score individually along with group membership using ANCOVA, but none of these covariates had a statistically significant effect on maternal responsiveness to receptiveness or fullness cues scores, nor did the addition of these covariates alter the non-significance of group effect. Post hoc analysis revealed no significant difference between mothers with AN only and BN only in responsiveness to receptiveness cues (Cohen's *d*=0.10). Although not statistically significant, there was a large effect size for the difference in responsiveness to fullness cues with mothers with histories of BN only being less responsive than mothers with histories of AN only (Cohen's *d*=1.14).

Maternal Responsiveness During Play

Groups (mothers with eating disorder histories versus controls) also did not differ significantly on mothers' global sensitivity rated during the 10-minute playtime interaction (Table 4). Covariates of maternal age, BDI score, and BAI score did not significantly predict global sensitivity when added in addition to group using ANCOVA, and the effect of group on global sensitivity remained non-significant after the addition of these covariates. Post hoc comparison of mothers with histories of AN only and BN only revealed no significant difference between eating disorder diagnoses in global sensitivity during play (Cohen's *d*=0.05).

RSA Withdrawal During Maternal-Child Interactions

Mean values of RSA for mothers in both groups during the two-minute baseline period, mealtime period, and playtime period are presented in Table 5. As our hypotheses regarded the change in RSA from baseline during both mealtime and playtime tasks, we did not conduct hypothesis testing for differences in mean RSA values during each task; we present these mean values in Table 5 as a reference. However, we were concerned that differences in baseline scores of RSA or vagal tone could influence change scores; we conducted a post hoc comparison of baseline RSA scores and found that the difference was not statistically significant ($t(43) = -1.06$, $p<0.30$).

RSA change scores for both groups are presented in Figure 1. Positive change scores represent a decrease in RSA from baseline (RSA withdrawal). We conducted a repeated measures ANOVA with RSA change (RSA withdrawal) as our outcome variable comparing mothers with eating disorder histories and control mothers during mealtime and playtime tasks. The analysis revealed a main effect of group (eating disorder versus control) on RSA change ($F(1,43)= 7.18$, $p<0.02$). There was no significant effect for task (meal versus play) and no significant group × task interaction. Mothers in the control group displayed significantly greater RSA decreases (greater RSA withdrawal) during both mealtime and playtime tasks than mothers with histories of eating disorders. We also evaluated covariates of maternal BMI, maternal age, and BAI score one at a time in addition to group using ANCOVA, but none of these covariates had a statistically significant effect on RSA change,

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nor did the addition of any of these covariates alter the significance of group effect (for group effect in all analyses of covariance, $p<0.02$). We conducted a similar post-hoc analysis evaluating the effect of eating disorder diagnosis (AN only versus BN only) on RSA change during mealtime and playtime tasks. We found no significant effect of eating disorder diagnosis on RSA change ($F(1,14)= 0.63$, Bonferroni-corrected $p=1.00$), nor was there a significant effect for task or the eating disorder diagnosis × task interaction.

As some mothers had playtime before the meal (13 mothers with eating disorder histories and 12 control mothers) and some after (12 mothers with eating disorder histories and 13 control mothers), a post-hoc analysis was conducted to determine if task sequence had a significant effect on maternal responsiveness. These analyses demonstrated no significant effect of task sequence on observed maternal responsiveness to receptiveness cues ($t(48)= -0.46$, $p=0.65$) or fullness cues ($t(48)= 1.47$, $p=0.15$), observed maternal sensitivity ($t(48)= 0.71$, $p=0.49$), or RSA reactivity ($F(1,43)= 2.15$, $p=0.15$).

DISCUSSION

We found no differences in observed responsiveness during feeding or global maternal sensitivity between mothers with histories of eating disorders and control mothers. However, physiologically, mothers with histories of eating disorders exhibited less RSA withdrawal during both mealtime and playtime interactions with their children. Mothers with histories of eating disorders also reported experiencing greater stress related to parenting, greater somatic symptoms of anxiety, and greater concern with their own eating, shape, and weight.

Observed Responsiveness During Feeding

While we are not aware of any other studies directly measuring feeding responsiveness during observed mealtimes in mothers with histories of eating disorders, several studies have found that mothers with eating disorders report more controlling and less responsive feeding styles via self-report instruments (Reba-Harrelson et al. 2010; Mazzeo et al. 2005; Duke et al. 2004; Agras et al. 1999; Honjo 1996). Our results are the first to demonstrate that during typical feeding interactions, mothers with histories of eating disorders do not differ from controls in their ability to recognize and respond to their children's feeding receptiveness and fullness cues. Mothers with histories of eating disorders have expressed increased concerns about feeding the appropriate quantity and quality of foods to their children (Mazzeo et al. 2005), but in practice, at least in this sample, they are moderately to highly responsive to their children's receptiveness and fullness cues. Our post hoc testing revealed a large effect for the difference between mothers with histories of AN only and BN only in terms of responsiveness to fullness cues. Although this effect was not statistically significant, given the small group size (10 per group) and conservative correction method for multiple testing, we may have lacked the power to detect this effect. These exploratory results support further study to uncover potential differences in maternal responsiveness across eating disorder diagnoses (and subtypes).

Physiologic Responsiveness During Feeding

Clinically relevant differences in RSA reactivity have been reported previously in individuals with eating disorders, with some aspects of RSA abnormalities persisting despite weight restoration and recovery (Messerli-Burgy et al. 2010; Lachish et al. 2009; Murialdo et al. 2007). However, this is the first study to evaluate RSA reactivity in women with histories of eating disorders during maternal-child interactions. Our results support past studies demonstrating decreased RSA reactivity in women with histories of eating disorders suggestive of increased vulnerability to stress (Messerli-Burgy et al. 2010; Murialdo et al. 2007). Contrary to our hypothesis, mothers with histories of eating disorders exhibited no

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RSA withdrawal in response to interactions with their children; mean scores of RSA withdrawal were actually negative, suggesting a paradoxical increase in vagal activity. We predicted that mothers with histories of eating disorders would perceive mealtime interactions as challenging and stressful, thus requiring RSA withdrawal to physiologically confront the challenge and support social engagement; however, the lower level of RSA observed at baseline in these mothers suggests that they may have a lower physiologic capacity to respond to environmental stressors.

The finding of decreased RSA reactivity in mothers with histories of eating disorders has important implications for future studies in this area. Although we did not detect any differences in behavioral responsiveness in our study of children age 6–36 months, it is possible that differences could emerge as children grow older and the stress of parenting increases. It will also be important in future studies to evaluate the children's baseline RSA and RSA reactivity during maternal-child interactions to determine if this decreased physiologic capacity to respond to challenging environmental stimuli could be familial and represent a vulnerability marker for later anxiety disorders or disordered eating.

Inclusion of Mothers Recovered from Eating Disorders

One possible explanation for our results is that our cases were all mothers who were recovered from eating disorders (no longer met all DSM-IV criteria for an eating disorder), while past studies finding that mothers with eating disorders reported being more restrictive or pressuring during feeding (Reba-Harrelson et al. 2010; Agras et al. 1999; Honjo 1996) or exhibited affective differences during observed mealtimes(Waugh and Bulik 1999; Stein et al. 1994) have included either all current or a mix of current and past eating disorder diagnoses. Interestingly, our study showed that while mothers with histories of eating disorders may not differ from control mothers in their responsiveness to their children's feeding cues and overall maternal sensitivity, they still reported greater anxiety and parenting stress, and physiologically they exhibited less RSA withdrawal during interactions with their children than control mothers. Thus, it may be that these mothers, through their recovery from an eating disorder, have learned how to manage their own physiology that may predispose them to greater anxiety and susceptibility to stress. In the observational setting, these mothers were able to respond to their children's cues and exhibit sensitive parenting behavior. However, this physiologic difference in vulnerability to stress could place these mothers at a greater risk for physical and psychological stress-related problems (and perhaps eating disorder relapse) as their children grow older and they face additional parenting challenges.

Limitations

There are several limitations that should be considered when interpreting our results. First, this was a non-randomized cohort study with a relatively small sample size, so we were unable to apply more complex statistical modeling to evaluate relationships across the different modalities of observational, self-report, and physiologic data. Second, as power to detect smaller effect sizes was limited, failure to find a significant effect does not imply the effect does not exist. Third, while the use of direct observation during maternal-child interactions is a strength, particularly when presented alongside self-report and physiologic data, this direct observation is subject to participant reactivity and social desirability bias. In an effort to minimize reactivity, we spent a warm-up period with mother and child in the home before filming began and trained research staff to minimize their visibility during filming. Multiple home visits might have reduced reactivity further, but we were concerned that the increased burden on participants and their families would compromise recruitment and retention. Fourth, we were unable to evaluate maternal responsiveness to hunger cues, which would give us valuable information about mothers' decisions to initiate feeding. We

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were also unable to standardize the sequence of the home assessment protocol; some mothers had playtime before the meal and some after. The distribution of sequences was similar for mothers with histories of eating disorders and controls, minimizing any potential influence on results, and post-hoc analyses demonstrated no significant effect of task sequence on observed maternal responsiveness during feeding. This flexibility in the home assessment sequence was necessary to preserve the natural feeding environment as much as possible; if a mother believed that her child was hungry at the beginning of filming, she was encouraged to feed her child as she normally would. If mothers felt their children were not ready to eat after filming began or if the child typically napped following the meal, mothers were given the option to engage in playtime before the meal to preserve the child's typical schedule. Fifth, the range of child ages (6–36 months) in this study was fairly broad. We recruited mothers such that the groups were similar for child age within 6 month age bands, so the mean child age between groups was not significantly different. However, the largest age group recruited was age 6–12 months—a time period when most children are just becoming accustomed to solid foods. Thus, these mothers' feeding behaviors were likely just being established and could change significantly after the children are weaned. Due to our small sample size, we were unable to conduct a subgroup analysis and evaluate differences in responsiveness measures across age groups. Finally, our sample was largely white and educated, so findings may not be widely generalizable.

Conclusion

In conclusion, many studies have attributed the increased risk of feeding problems and disordered eating in children of mothers with eating disorders to controlling (restrictive or pressuring) feeding practices based on maternal self-report. Our study is the first to evaluate detailed attributes of responsive feeding through direct observation in mothers with histories of eating disorders, and we found no differences in these mothers compared with controls in responsiveness to child feeding cues or overall maternal sensitivity. However, physiologically, mothers with histories of eating disorders exhibited decreased stress reactivity and reported greater anxiety and parenting stress than controls. These results provide initial evidence of a link between a blunted RSA response and eating disorder history and highlight the need for future larger-scale investigations of this association. Understanding maternal RSA response during events that may be particularly stressful for mothers with histories of eating disorders could have important implications for developing programs to help these mothers respond to stressful situations more effectively. Biofeedback training that incorporates breathing exercises, for example, may help to decrease physiological arousal leading to a more regulated emotional state.

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References

Agras S, Hammer L, McNicholas F. A prospective study of the influence of eating-disordered mothers on their children. International Journal of Eating Disorders. 1999; 25(3):253–262. [PubMed: 10191989]

Anzman SL, Birch LL. Low inhibitory control and restrictive feeding practices predict weight outcomes. Journal of Pediatrics. 2009; 155(5):651–656. [pii]. 10.1016/j.jpeds.2009.04.052S0022-3476(09)00448-X [PubMed: 19595373]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Appelbaum M, Batten DA, Belsky J, Booth C, Bradley R, Brownell C, et al. The effects of infant child care on infant-mother attachment security: Results of the NICHD study of early child care. Child Development. 1997; 68(5):860–879.

Beauchaine T. Vagal tone, development, and Gray's motivational theory: toward an integrated model of autonomic nervous system functioning in psychopathology. [Research Support, U.S. Gov't, P.H.S.Review]. Development and Psychopathology. 2001; 13(2):183–214. [PubMed: 11393643]

Beauchaine TP, Gatzke-Kopp L, Mead HK. Polyvagal Theory and developmental psychopathology: emotion dysregulation and conduct problems from preschool to adolescence. Biological Psychology. 2007; 74(2):174–184. S0301-0511(06)00183-9 [pii]. 10.1016/j.biopsycho.2005.08.008. [PubMed: 17045726]

Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: psychometric properties. Journal of Consulting and Clinical Psychology. 1988; 56(6):893–897. [PubMed: 3204199]

Beck AT, Steer RA, Ball R, Ranieri W. Comparison of Beck Depression Inventories -IA and -II in psychiatric outpatients. Journal of Personality Assessment. 1996; 67(3):588–597. [PubMed: 8991972]

Birch LL, Fisher JO. Mothers' child-feeding practices influence daughters' eating and weight. American Journal of Clinical Nutrition. 2000; 71(5):1054–1061. [PubMed: 10799366]

Blissett J, Haycraft E. Parental eating disorder symptoms and observations of mealtime interactions with children. Journal of Psychosomatic Research. 2011; 70(4):368–371. [pii]. 10.1016/j.jpsychores.2010.07.006S0022-3999(10)00281-3 [PubMed: 21414457]

Boccia ML, Pedersen CA. Brief vs. long maternal separations in infancy: contrasting relationships with adult maternal behavior and lactation levels of aggression and anxiety. [Research Support, U.S. Gov't, P.H.S.]. Psychoneuroendocrinology. 2001; 26(7):657–672. [PubMed: 11500248]

Bornstein MH, Suess PE. Child and mother cardiac vagal tone: continuity, stability, and concordance across the first 5 years. Developmental Psychology. 2000; 36(1):54–65. [PubMed: 10645744]

Brinch M, Isager T, Tolstrup K. Anorexia nervosa and motherhood: reproduction pattern and mothering behavior of 50 women. Acta Psychiatrica Scandinavica. 1988; 77(5):611–617. [PubMed: 3269214]

Britton JR. Infant temperament and maternal anxiety and depressed mood in the early postpartum period. Women and Health. 2011; 51(1):55–71.10.1080/03630242.2011.540741. [PubMed: 21391161]

Bulik CM, Reba L, Siega-Riz AM, Reichborn-Kjennerud T. Anorexia nervosa: definition, epidemiology, and cycle of risk. International Journal of Eating Disorders. 2005; 37(Suppl):S2–9. discussion S20–21. [PubMed: 15852310]

Butler EA, Wilhelm FH, Gross JJ. Respiratory sinus arrhythmia, emotion, and emotion regulation during social interaction. Psychophysiology. 2006; 43(6):612–622. PSYP467 [pii]. 10.1111/j.1469-8986.2006.00467.x. [PubMed: 17076818]

Bydlowski S, Corcos M, Jeammet P, Paterniti S, Berthoz S, Laurier C, et al. Emotion-processing deficits in eating disorders. International Journal of Eating Disorders. 2005; 37(4):321–329.10.1002/eat.20132. [PubMed: 15856501]

Clarke TK, Weiss AR, Berrettini WH. The genetics of anorexia nervosa. Clinical Pharmacology and Therapeutics. 2012; 91(2):181–188. [pii]. 10.1038/clpt.2011.253clpt2011253 [PubMed: 22190067]

Cohen J. A power primer. Psychological Bulletin. 1992; 112(1):155–159. [PubMed: 19565683]

Cooper PJ, Whelan E, Woolgar M, Morrell J, Murray L. Association between childhood feeding problems and maternal eating disorder: role of the family environment. British Journal of Psychiatry. 2004; 184:210–215. [PubMed: 14990518]

Diamond LM, Hicks AM, Otter-Henderson KD. Individual differences in vagal regulation moderate associations between daily affect and daily couple interactions. Personality & Social Psychology Bulletin. 2011; 37(6):731–744. [pii]. 10.1177/0146167211400620014616721140062001461672114006200 [PubMed: 21393615]

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

DiSantis KI, Hodges EA, Johnson SL, Fisher JO. The role of responsive feeding in overweight during infancy and toddlerhood: a systematic review. International Journal of Obesity (Lond). 2011; 35(4):480–492. [pii]. 10.1038/ijo.2011.3ijo20113

Duke RE, Bryson S, Hammer LD, Agras WS. The relationship between parental factors at infancy and parent-reported control over children's eating at age 7. Appetite. 2004; 43(3):247–252. [PubMed: 15527926]

Easter A, Naumann U, Northstone K, Schmidt U, Treasure J, Micali N. A Longitudinal Investigation of Nutrition and Dietary Patterns in Children of Mothers with Eating Disorders. Journal of Pediatrics. 2013 S0022-3476(12)01427-8 [pii]. 10.1016/j.jpeds.2012.11.092

Fairburn CG, Beglin SJ. Assessment of eating disorders: interview or self-report questionnaire? International Journal of Eating Disorders. 1994; 16(4):363–370. [PubMed: 7866415]

Faith MS, Berkowitz RI, Stallings VA, Kerns J, Storey M, Stunkard AJ. Parental feeding attitudes and styles and child body mass index: prospective analysis of a gene-environment interaction. Pediatrics. 2004; 114(4):e429–436. [PubMed: 15466068]

First, MB.; Spitzer, R.; Gibbon, M.; Williams, JB. Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Patient Edition. (SCID-I/P). New York: Biometrics Research, New York State Psychiatric Institute; 2002.

Fisher JO, Birch LL. Eating in the absence of hunger and overweight in girls from 5 to 7 y of age. American Journal of Clinical Nutrition. 2002; 76(1):226–231. [PubMed: 12081839]

Grossman P, Svebak S. Respiratory sinus arrhythmia as an index of parasympathetic cardiac control during active coping. Psychophysiology. 1987; 24(2):228–235. [PubMed: 3602275]

Grossman P, Taylor EW. Toward understanding respiratory sinus arrhythmia: relations to cardiac vagal tone, evolution and biobehavioral functions. [Research Support, Non-U.S. Gov't Review]. Biological Psychology. 2007; 74(2):263–285.10.1016/j.biopsycho.2005.11.014. [PubMed: 17081672]

Gyurak A, Ayduk O. Resting respiratory sinus arrhythmia buffers against rejection sensitivity via emotion control. Emotion. 2008; 8(4):458–467. [pii]. 10.1037/1528-3542.8.4.4582008-09984-004 [PubMed: 18729578]

Harrison A, Sullivan S, Tchanturia K, Treasure J. Emotion recognition and regulation in anorexia nervosa. Clinical Psychology & Psychotherapy. 2009; 16(4):348–356.10.1002/cpp.628. [PubMed: 19517577]

Haskett ME, Ahern LS, Ward CS, Allaire JC. Factor structure and validity of the parenting stress index-short form. Journal of Clinical Child & Adolescent Psychology 35. 2006; 2:302–312.10.1207/s15374424jccp3502_14.

Hastings PD, Nuselovici JN, Utendale WT, Coutya J, McShane KE, Sullivan C. Applying the polyvagal theory to children's emotion regulation: Social context, socialization, and adjustment. Biological Psychology. 2008; 79(3):299–306. [pii]. 10.1016/j.biopsycho. 2008.07.005S0301-0511(08)00172-5 [PubMed: 18722499]

Hill-Soderlund AL, Mills-Koonce WR, Propper C, Calkins SD, Granger DA, Moore GA, et al. Parasympathetic and sympathetic responses to the strange situation in infants and mothers from avoidant and securely attached dyads. Developmental Psychobiology. 2008; 50(4):361–376. [PubMed: 18393278]

Hodges EA, Liu Y, Johnson S, Hughes SO, Fisher JO. Responsiveness to child feeding cues: An observational scale. Obesity. 2008; 16(Suppl 1):S273.

Holsen LM, Lawson EA, Blum J, Ko E, Makris N, Fazeli PK, et al. Food motivation circuitry hypoactivation related to hedonic and nonhedonic aspects of hunger and satiety in women with active anorexia nervosa and weight-restored women with anorexia nervosa. Journal of Psychiatry and Neuroscience. 2012; 37(5):322–332. [pii]. 10.1503/jpn.11015610.1503/cjs.110156 [PubMed: 22498079]

Honjo S. A mother's complaints of overeating by her 25-month-old daughter: a proposal of anorexia nervosa by proxy. International Journal of Eating Disorders. 1996; 20(4):433–437. [PubMed: 8953332]

Johnston C, Mash EJ. A measure of parenting satisfaction and efficacy. Journal of Clinical Child Psychology. 1989; 18:167–175.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Kaye WH, Wagner A, Fudge JL, Paulus M. Neurocircuity of eating disorders. Current Topics in Behavioral Neuroscience. 2011; 6:37–57.10.1007/7854_2010_85.

Kettunen J, Ravaja N, Naeaetaenen P, Keltikangas Jaervinen L. The relationship of respiratory sinus arrhythmia to the co activation of autonomic and facial responses during the Rorschach test. Psychophysiology. 2000; 37:242–250. [PubMed: 10731774]

Kuczmarski RJ, Ogden CL, Grummer-Strawn LM, Flegal KM, Guo SS, Wei R, et al. CDC growth charts: United States. Advance Data. 2000; (314):1–27. [PubMed: 11183293]

Lachish M, Stein D, Kaplan Z, Matar M, Faigin M, Korsunski I, et al. Irreversibility of cardiac autonomic dysfunction in female adolescents diagnosed with anorexia nervosa after short- and long-term weight gain. World Journal of Biological Psychiatry. 2009:1–9.

Lombardo C, Battagliese G, Lucidi F, Frost RO. Body dissatisfaction among pre-adolescent girls is predicted by their involvement in aesthetic sports and by personal characteristics of their mothers. Eating and weight disorders : EWD. 2012; 17(2):e116–127. 8606 [pii]. [PubMed: 23010781]

Luce KH, Crowther JH. The reliability of the Eating Disorder Examination-Self-Report Questionnaire Version (EDE-Q). International Journal of Eating Disorders. 1999; 25(3):349–351. [pii]. 10.1002/(SICI)1098-108X(199904)25:3<349::AID-EAT15>3.0.CO;2-M [PubMed: 10192002]

Mazzeo SE, Zucker NL, Gerke CK, Mitchell KS, Bulik CM. Parenting concerns of women with histories of eating disorders. International Journal of Eating Disorders. 2005; 37(Suppl):S77–79. discussion S87–79. [PubMed: 15852326]

Messerli-Burgy N, Engesser C, Lemmenmeier E, Steptoe A, Laederach-Hofmann K. Cardiovascular stress reactivity and recovery in bulimia nervosa and binge eating disorder. International Journal of Psychophysiology. 2010; 78(2):163–168. S0167-8760(10)00664-1 [pii]. 10.1016/j.ijpsycho.2010.07.005. [PubMed: 20667455]

Micali N, Simonoff E, Treasure J. Infant feeding and weight in the first year of life in babies of women with eating disorders. Journal of Pediatrics. 2009; 154(1):55–60. e51. [PubMed: 18783793]

Mills-Koonce WR, Gariepy JL, Propper C, Sutton K, Calkins S, Moore G, et al. Infant and parent factors associated with early maternal sensitivity: a caregiver-attachment systems approach. Infant behavior & development. 2007; 30(1):114–126. [PubMed: 17292784]

Mills-Koonce WR, Propper C, Gariepy JL, Barnett M, Moore GA, Calkins S, et al. Psychophysiological correlates of parenting behavior in mothers of young children. Developmental Psychobiology. 2009

Mond JM, Hay PJ, Rodgers B, Owen C, Beumont PJ. Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. Behaviour Research and Therapy. 2004; 42(5):551–567. [pii]. 10.1016/S0005-7967(03)00161-XS000579670300161X [PubMed: 15033501]

Murialdo G, Casu M, Falchero M, Brugnolo A, Patrone V, Cerro PF, et al. Alterations in the autonomic control of heart rate variability in patients with anorexia or bulimia nervosa: correlations between sympathovagal activity, clinical features, and leptin levels. Journal of Endocrinological Investigation. 2007; 30(5):356–362. [PubMed: 17598965]

Ochsner KN, Gross JJ. Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. Current Directions in Psychological Science. 2008; 17:153–158.

Oppenheimer JE, Measelle JR, Laurent HK, Ablow JC. Mothers' vagal regulation during the Still-Face Paradigm: Normative reactivity and impact of depression symptoms. Infant behavior & development. 2013; 36(2):255–267. S0163-6383(13)00004-0 [pii]. 10.1016/j.infbeh.2013.01.003. [PubMed: 23454427]

Ottaviani C, Shapiro D, Davydov DM, Goldstein IB. Autonomic stress response modes and ambulatory heart rate level and variability. Journal of Psychophysiology. 2008; 22:28–40.

Oveis C, Cohen AB, Gruber J, Shiota MN, Haidt J, Keltner D. Resting respiratory sinus arrhythmia is associated with tonic positive emotionality. Emotion. 2009; 9(2):265–270. [pii]. 10.1037/a00153832009-04472-012 [PubMed: 19348538]

Patel P, Wheatcroft R, Park RJ, Stein A. The children of mothers with eating disorders. [Research Support, Non-U.S. Gov't Review]. Clinical child and family psychology review. 2002; 5(1):1–19. [PubMed: 11993543]

NIH-PA Author Manuscript NIH-PA Author Manuscript NIH-PA Author Manuscript

Plomin R, DeFries JC, Loehlin JC. Genotype-environment interaction and correlation in the analysis of human behavior. Psychological Bulletin. 1977; 84(2):309–322. [PubMed: 557211]

Pollatos O, Kurz AL, Albrecht J, Schreder T, Kleemann AM, Schopf V, et al. Reduced perception of bodily signals in anorexia nervosa. Eating behaviors. 2008; 9(4):381–388. [pii]. 10.1016/j.eatbeh.2008.02.001S1471-0153(08)00004-4 [PubMed: 18928900]

Porges SW. Cardiac vagal tone: a physiological index of stress. Neuroscience and Biobehavioral Reviews. 1995; 19(2):225–233. 014976349400066A [pii]. [PubMed: 7630578]

Porges SW. Physiological regulation in high-risk infants: A model for assessment and potential intervention. Development and Psychopathology. 1996; 8:43–58.

Porges SW. Love: an emergent property of the mammalian autonomic nervous system. Psychoneuroendocrinology. 1998; 23(8):837–861. [PubMed: 9924740]

Porges SW. The polyvagal perspective. Biological Psychology. 2007; 74(2):116–143. S0301-0511(06)00176-1 [pii]. 10.1016/j.biopsycho.2006.06.009. [PubMed: 17049418]

Porges SW. The polyvagal theory: new insights into adaptive reactions of the autonomic nervous system. Cleveland Clinic Journal of Medicine. 2009; 76(Suppl 2):S86–90. [pii]. 10.3949/ccjm.76.s2.1776/Suppl_2/S86 [PubMed: 19376991]

Porges SW, Byrne EA. Research methods for measurement of heart rate and respiration. [Review]. Biological Psychology. 1992; 34(2–3):93–130. [PubMed: 1467397]

Reba-Harrelson L, Von Holle A, Hamer RM, Torgersen L, Reichborn-Kjennerud T, Bulik CM. Patterns of maternal feeding and child eating associated with eating disorders in the Norwegian Mother and Child Cohort Study (MoBa). [Research Support, Non-U.S. Gov't]. Eating behaviors. 2010; 11(1):54–61.10.1016/j.eatbeh.2009.09.004. [PubMed: 19962121]

Robertson J, Puckering C, Parkinson K, Corlett L, Wright C. Mother-child feeding interactions in children with and without weight faltering; nested case control study. Appetite. 2011; 56(3):753–759. [pii]. 10.1016/j.appet.2011.02.016S0195-6663(11)00099-7 [PubMed: 21396417]

Rothbart, MK.; Derryberry, D. Development of individual differences in temperament. In: Lamb, ME.; Brown, AL., editors. Advances in developmental psychology. Vol. 1. Hillsdale, NJ: Erlbaum; 1981. p. 37-86.

Sack M, Hopper JW, Lamprecht F. Low respiratory sinus arrhythmia and prolonged psychophysiological arousal in posttraumatic stress disorder: heart rate dynamics and individual differences in arousal regulation. Biological Psychiatry. 2004; 55(3):284–290. S0006322303006772 [pii]. [PubMed: 14744470]

SAS/STAT® Software: Version 9.2. Cary, NC: SAS Institute Inc;

Stein A, Fairburn CG. Children of mothers with bulimia nervosa. BMJ. 1989; 299(6702):777–778. [PubMed: 2508920]

Stein A, Woolley H, Cooper SD, Fairburn CG. An observational study of mothers with eating disorders and their infants. Journal of Child Psychology and Psychiatry and Allied Disciplines. 1994; 35(4):733–748.

Stice E, Agras WS, Hammer LD. Risk factors for the emergence of childhood eating disturbances: a five-year prospective study. International Journal of Eating Disorders. 1999; 25(4):375–387. [PubMed: 10202648]

Striegel-Moore RH, Bulik CM. Risk factors for eating disorders. American Psychologist. 2007; 62(3):181–198. [PubMed: 17469897]

Thornton LM, Mazzeo SE, Bulik CM. The heritability of eating disorders: methods and current findings. Curr Top Behav Neurosci. 2011; 6:141–156.10.1007/7854_2010_91. [PubMed: 21243474]

Watkins B, Cooper PJ, Lask B. History of eating disorder in mothers of children with early onset eating disorder or disturbance. European Eating Disorders Review. 2012; 20(2):121–125.10.1002/erv.1125. [PubMed: 21823212]

Waugh E, Bulik CM. Offspring of women with eating disorders. International Journal of Eating Disorders. 1999; 25(2):123–133. [PubMed: 10065389]

Whelan E, Cooper PJ. The association between childhood feeding problems and maternal eating disorder: a community study. Psychological Medicine. 2000; 30(1):69–77. [PubMed: 10722177]

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Winslow JT, Hearn EF, Ferguson J, Young LJ, Matzuk MM, Insel TR. Infant vocalization, adult aggression, and fear behavior of an oxytocin null mutant mouse. Hormones and Behavior. 2000; 37(2):145–155. [PubMed: 10753584]

Woodside D, Shekter-Wolfson L. Parenting by patients with anorexia nervosa and bulimia nervosa. International Journal of Eating Disorders. 1990; 9:303–309.

Wotjak CT, Kubota M, Liebsch G, Montkowski A, Holsboer F, Neumann I, et al. Release of vasopressin within the rat paraventricular nucleus in response to emotional stress: a novel mechanism of regulating adrenocorticotropic hormone secretion? [Research Support, Non-U.S. Gov't]. The Journal of neuroscience : the official journal of the Society for Neuroscience. 1996; 16(23):7725–7732. [PubMed: 8922428]

Zucker NL, Losh M, Bulik CM, LaBar KS, Piven J, Pelphrey KA. Anorexia nervosa and autism spectrum disorders: guided investigation of social cognitive endophenotypes. Psychological Bulletin. 2007; 133(6):976–1006. [PubMed: 17967091]

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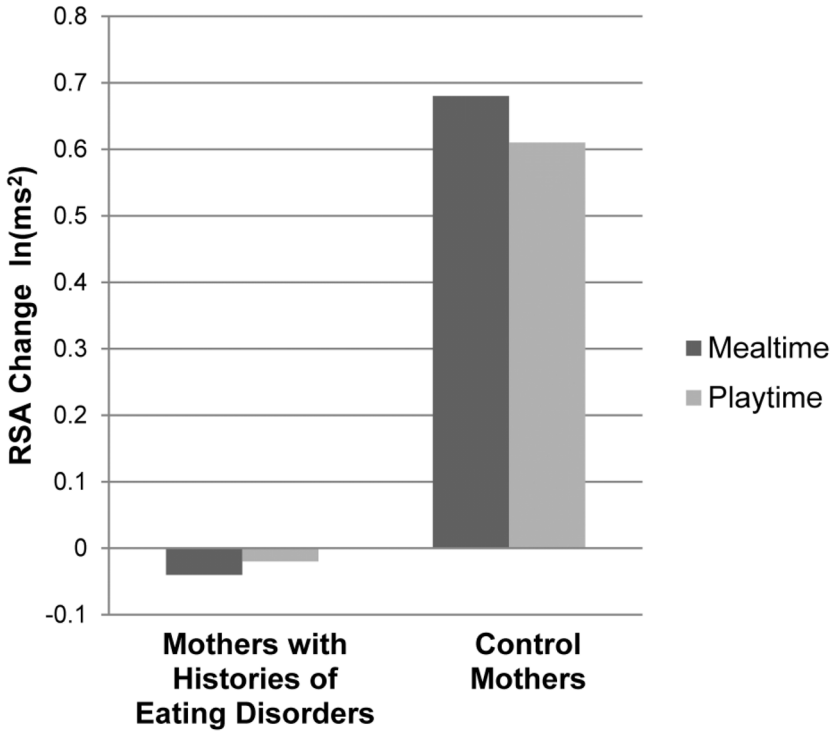

Figure 1.
RSA change scores from baseline during both mealtime and playtime maternal-child interactions are presented. Positive values for RSA change indicate a decrease in RSA from baseline (RSA withdrawal). $F(1,43)=7.18$, $p<0.02$ for effect of group (mothers with histories of eating disorders versus control mothers) on RSA change; effect of task and group × task interaction were not statistically significant.

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Table 1

Demographic and Clinical Characteristics of Mothers

	Mothers with Histories of Eating Disorders		Control Mothers		Test Statistic	*p*-value
	Mean	**SD**	**Mean**	**SD**		
Age (y)	32.72	4.61	29.68	1.99	$t(48) = 3.02$	$p < 0.005$
Current BMI (kg/m^2)	22.37	2.69	23.04	3.04	$t(47) = -0.82$	$p < 0.42$
Lowest Adult BMI (kg/m^2)	18.22	1.90	20.34	2.31	$t(48) = -3.55$	$p < 0.001$
Highest Adult BMI (kg/m$^{2)}$	24.25	3.46	24.08	2.98	$t(48) = 0.18$	$p < 0.86$
Age at Eating Disorder Onset (y)	17.46	4.77				
	No.	**%**	**No.**	**%**		
Parity					FET	$p<0.99$
1 child	18	72	19	76		
>1 child	7	28	6	24		
Single Parent	7	28	3	12	FET	$p<0.29$
Education					FET	$p < 0.32$
GED or High School	1	4	0	0		
Some College	0	0	3	12		
College Graduate	7	29	8	32		
Post Graduate Degree	16	67	14	56		
Race					FET	$p < 0.99$
White	24	96	24	96		
Other	1	4	1	4		
	Mean	**SD**	**Mean**	**SD**		
EDE-Q (Global)	1.71	1.40	0.91	0.83	$t(48) = 2.46$	$p < 0.02$
BDI-II	5.68	5.46	4.32	4.69	$t(48) = 0.94$	$p < 0.35$
BAI	4.56	3.80	2.58	1.80	$t(48) = 2.95$	$p < 0.005$

FET=Fisher's Exact Test, EDE-Q=Eating Disorders Examination Questionnaire, BDI=Beck Depression Inventory, BAI=Beck Anxiety Inventory

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Table 2

Demographic and Clinical Characteristics of Children

	Children of Mothers with Histories of Eating Disorders		Children of Control Mothers		Test Statistic	*p*-value
	No.	**%**	**No.**	**%**		
Child Sex						
Male	13	52	13	52		
Age Group						
6–12 months	8	32	8	32		
13–18 months	5	20	5	20		
19–24 months	4	16	4	16		
25–30 months	3	12	3	12		
31–36 months	5	20	5	20		
	Mean	**SD**	**Mean**	**SD**		
Age (mo)	18.92	9.76	19.56	9.35	$t(48) = -0.24$	$p < 0.81$
Current Weight-for-Length z-score	0.16	0.60	0.15	1.01	$t(47) = 0.03$	$p < 0.98$

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Table 3

Mothers' Self-Reported Parenting Stress and Parenting Self-Efficacy

	Comparison of Mothers with Histories of Eating Disorders and Controls					Post-hoc Comparisons of Mothers with AN Only and Mothers with BN Only			
	Mothers with Histories of Eating Disorders (n = 25)	Control Mothers (n = 25)				History of AN only (n = 10)	History of BN only (n = 10)		
	Mean (SD)	Mean (SD)	Test Statistic	*p*-value	Cohen's *d*	Mean (SD)	Mean (SD)	*p*-value[a]	Cohen's *d*
PSI/SF Total Stress	70.52 (15.97)	61.75 (12.27)	$t(47) = 2.15$	$p < 0.04$	0.63	69.90 (20.02)	69.70 (14.07)	$p = 1$	0.01
Parental Distress	26.40 (7.82)	22.00 (5.95)	$t(48) = 2.24$	$p < 0.03$	0.65	27.00 (9.91)	25.40 (7.38)	$p = 1$	0.19
Parent-Child Dysfunctional Interaction	20.12 (5.20)	16.84 (4.18)	$t(48) = 2.45$	$p < 0.02$	0.71	19.40 (5.10)	19.30 (4.22)	$p = 1$	0.02
Difficult Child	24.00 (6.41)	22.29 (5.21)	$t(47) = 1.02$	$p < 0.32$	0.30	23.50 (7.25)	25.00 (5.93)	$p = 1$	0.24
PSOC Self-Efficacy	4.55 (0.96)	4.66 (0.76)	$t(48) = -0.44$	$p < 0.67$	0.13	4.69 (0.84)	4.74 (1.01)	$p = 1$	0.07

PSI/SF–Parenting Stress Index-Short Form; higher scores represent greater stress, PSOC–Parenting Sense of Competency Scale; higher scores represent greater self-efficacy

[a]Comparison of mothers with history of AN only and BN only represents additional post hoc testing; p-values presented are adjusted using Bonferroni correction to account for multiple comparisons

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Table 4

Maternal Responsiveness During Feeding and Sensitivity During Play

	Comparison of Mothers with Histories of Eating Disorders and Controls					Post-hoc Comparisons of Mothers with AN Only and Mothers with BN Only			
	Mothers with Histories of Eating Disorders (n = 25)	Control Mothers (n = 25)				History of AN only (n = 10)	History of BN only (n = 10)		
	Mean (SD)	Mean (SD)	Test Statistic	*p*-value	Cohen's *d*	Mean (SD)	Mean (SD)	*p*-value[a]	Cohen's *d*
Behavioral Coding of Feeding									
Responsiveness to receptiveness cues	4.56 (0.96)	4.40 (0.91)	$t(48) = 0.60$	$p < 0.55$	0.17	4.40 (0.84)	4.50 (1.27)	$p = 1$	0.10
Responsiveness to fullness cues	3.56 (0.96)	3.84 (1.07)	$t(48) = -0.97$	$p < 0.33$	0.28	4.10 (0.74)	3.20 (0.92)	$p < 0.24$	1.14
Behavioral Coding of Play									
Global Sensitivity	9.52 (4.24)	10.00 (3.55)	$t(48) = -0.43$	$p < 0.67$	0.12	9.40 (4.79)	9.60 (4.33)	$p = 1$	0.05

Responsiveness to feeding cues scale scores range from 1 (unresponsive) to 5 (highly responsive); Global sensitivity composite scores range from −1 (low sensitivity) to 19 (highly sensitive)

[a]Comparison of mothers with history of AN only and BN only represents additional post hoc testing; p-values presented are adjusted using Bonferroni correction to account for multiple comparisons

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Table 5

Mean RSA During Mealtime and Playtime

	Mothers with Histories of Eating Disorders	Control Mothers
	Mean (SD)	**Mean (SD)**
RSA		
Baseline	6.04 (1.32)	6.47 (1.42)
Mealtime	6.08 (1.05)	5.80 (1.16)
Playtime	6.06 (1.12)	5.86 (1.20)

RSA–Respiratory sinus arrhythmia