PATTERNS OF MATERNAL FEEDING AND CHILD EATING ASSOCIATED WITH EATING DISORDERS IN THE NORWEGIAN MOTHER AND CHILD COHORT STUDY (MOBA)

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A dissertation submitted to the faculty of the University of North Carolina at Chapel Hill in partial fulfillment of the requirements for the degree of Doctor of Philosophy in the Department of Psychology (Clinical).

Chapel Hill
2008

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ABSTRACT

LAUREN REBA-HARRELSON: Patterns of Maternal Feeding and Child Eating Associated with Eating Disorders in the Norwegian Mother and Child Cohort Study (MoBA)
(Under the direction of Cynthia M. Bulik, Ph.D.)

Mothers with eating disorders (EDs) and their children are an at-risk group. This study explored the impact of broadly defined EDs on maternal feeding practices and children's eating behaviors and psychological symptoms in a sample of 13,006 births in Norway. The Norwegian Mother and Child Cohort Study ("Den norske mor og barn undersøkelsen" – MoBa) is a prospective population-based study of 100,000 births throughout Norway. We compared: (1) self-reported feeding attitudes and practices in mothers with EDs across diagnostic subtypes (anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and mothers with no EDs and (2) maternal-reported eating behaviors and psychological symptoms in these mother's 36 month old children. ED status was measured at 6 months prior to pregnancy or during pregnancy (assessed 17 weeks prenatal). Self-reported maternal restrictive feeding was higher in mothers with BN and BED than those with no ED. Mothers with BED were more likely to endorse pushy feeding and use of food as a reward than those without an ED. Mothers with BN and BED were more likely to report infant eating problems than mothers without EDs. Compared to those with no ED, maternal reported symptoms of child anxiety were higher among those with BN, and child OCD symptoms were higher among those with BN and BED; no differences emerged regarding reported child depressive symptoms. Findings suggest that mothers with some EDs, on average, differ from mothers without in how they feed their children and that their children display different eating behaviors than children of mothers without eating disorders. The cycle of risk associated with transmission of ED from mother to child is a salient public health concern and an important approach to unravel the interactive effects of genetic and environmental influences.

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LIST OF ABBREVIATIONS

1. Anorexia nervosa (assessed prior to pregnancy)   AN

2. Binge eating disorder   BED

3. Bulimia nervosa   BN

4. Eating Disorder   ED

5. Purging in the absence of binge eating   EDNOS-P

PATTERNS OF MATERNAL FEEDING AND CHILD EATING ASSOCIATED WITH
EATING DISORDERS IN THE NORWEGIAN MOTHER AND CHILD COHORT
STUDY (MOBA)

I. Introduction

Children of mothers with eating disorders (EDs) are an at-risk group for the potential development of ED symptomology. Family studies have consistently demonstrated that EDs are strongly familial (Hudson, Pope, Jonas, Yurgelun-Todd, & Frankenburg, 1987; Kassett et al., 1989; Lilenfeld et al., 1998; Strober, Freeman, Lampert, Diamond, & Kaye, 2000; Strober, Lampert, Morrell, Burroughs, & Jacobs, 1990). Further, twin studies have shown that observed familiality is due primarily to genetic factors (Bulik, Sullivan, Wade, & Kendler, 2000; Klump, Burt, McGue, & Iacono, 2007; Klump, Miller, Keel, McGue, & Iacono, 2001; Kortegaard, Hoerder, Joergensen, Gillberg, & Kyvik, 2001; Slof, Mazzeo, & Bulik, 2003; Wade, Bulik, Neale, & Kendler, 2000). Nonetheless, environment may play an important role in the expression of underlying genetic predispositions. The parents who provide offspring with genes that may increase their risk of developing EDs in most cases create the environment in which the children are raised (gene x environment correlation).

Mothers with EDs are typically very concerned about their children's well-being, and do not appear to harm them purposefully (Franzen & Gerlinghoff, 1997; Russell, Treasure, & Eisler, 1998). Indeed, concerns that their ED will negatively affect their children can be an impetus for treatment-seeking in some women (Fahy & Treasure, 1989; Franzen & Gerlinghoff, 1997). Some biological data suggest that this concern is founded. Offspring of

women with EDs are at-risk for perinatal mortality, premature birth, low birth weight and birth defects (Brinch, Isager, & Tolstrup, 1988; Lacey & Smith, 1987; Patel, Wheatcroft, Park, & Stein, 2002), and are also more likely than those of non-ED women to develop feeding problems and non-organic failure to thrive (Stein, Woolley, Cooper, & Fairburn, 1994; Whelan & Cooper, 2000). Many women also express concerns that their children will develop EDs later in life (Fahy & Treasure, 1989; Lemberg & Phillips, 1989). Although genetic studies can investigate some aspects of transmitted risk, research addressing environmental factors is scant. The majority of existing data exploring environmental factors have come from case studies or studies with small sample sizes, studies using ED diagnostic criteria from the DSM-III-R (American Psychiatric Association, 1987), differing from current conceptualizations of ED subtypes, or studies not investigating differences across ED subtypes. Each of these can limit generalizability and applicability to current eating disorders populations.

Of the few extant studies examining the impact of maternal ED status on feeding styles and eating behavior in early childhood, one found that mothers with EDs interact differently with their young children in terms of feeding style than mothers without EDs (Agras, Hammer, & McNicholas, 1999). Mothers with past or current EDs were more likely to demonstrate unregulated or non-nutritive feeding, or concern about their daughters' weight (from age 2 and up) than mothers without EDs. Women with AN were also found to regulate their children's eating behavior for fear of overeating and be more concerned about their children's weight, even when it was within the normal range, than mothers without EDs (Russell et al., 1998). The authors reported that mothers with AN used diluting infant formula, reducing foods in the home, and limiting snacking, sweets and second helpings as

means of limiting their children's intake. Other characteristics observed include greater rigidity at mealtimes, difficulty coping when their children report hunger outside of meals Evans & le Grange, (1995), and active attempts to help their young children lose weight (Agras et al., 1999; Lacey & Smith, 1987). To date, only one prospective study has examined eating habits in offspring of mothers with EDs (Stein et al., 2006), revealing that 10 year old offspring of mothers with EDs exhibit more dietary restraint and place greater value on weight/shape in their self-evaluation than controls.

However, there are significant limitations to these studies. Each either used DSM-III-R criteria to assess ED status or did not explore comparisons across ED subtypes. Although the use of DSM-III criteria was appropriate at the time, changes to the diagnostic criteria and subtyping have occurred in the interim. In many cases, sample sizes were very small (e.g., N=8 in Russell et al., 1998), and findings have not been replicated. Using a comprehensive set of predictor and outcome variables, there have been no prior large-scale, prospective, population-based studies exploring the impact of ED subtype on maternal feeding habits and child eating behaviors and psychological symptoms in young children.

Specific Aims

The current study explored the impact of broadly defined EDs on maternal feeding practices and children's eating behaviors and psychological symptoms in a sample of 13,006 births in Norway with valid data from the Medical Birth Registry of Norway and study questionnaires. The Norwegian Mother and Child Cohort Study ("Den norske mor og barn undersøkelsen" – MoBa) is a prospective population-based study of 100,000 births throughout Norway sponsored by the the Norwegian Institute of Public Health. Assessments include biobanked DNA, psychosocial factors, infections, medication, nutrition, occupational

exposure, use of health services, substance abuse, and socioeconomic indicators. This study's aims are threefold:

1. To compare maternally-reported feeding attitudes and practices in mothers with broadly defined EDs across diagnostic subtypes (AN, BN and BED) with mothers without EDs. ED status was measured at 6 months prior to pregnancy and during pregnancy (asked 17 weeks prenatal), and controlled for child weight status at 36 months and child food allergies.

Hypothesis 1. Mothers with AN before pregnancy will endorse feeding attitudes and practices that restrict their children's eating significantly more than mothers with BN, BED, or no EDs.

Hypothesis 2. Mothers with BN and BED will endorse feeding attitudes and practices that pressure their children to eat significantly more than mothers with AN before pregnancy or no EDs.

Hypothesis 3. Mothers with BN and BED will tend to use food as a reward for their children significantly more than mothers without EDs or mothers with AN before pregnancy.

2. To compare maternally-reported eating behaviors and psychological symptoms (i.e., depression, anxiety, and obsessive-compulsive symptoms) in 36 month old children of mothers with EDs across diagnostic subtypes (AN, BN and BED) with children of mothers with no EDs, while controlling for child weight status at 36 months and child food allergies for reported child eating behaviors.

Hypothesis 1. Thirty-six month old children of mothers with AN before pregnancy will exhibit problematic eating behaviors significantly more than children of mothers without EDs and children or mothers with BN and BED.

Hypothesis 2. Thirty-six month old children of mothers with AN before pregnancy will exhibit obsessive compulsive traits significantly more than children of mothers with no ED and children of mothers with BN and BED. Thirty-six month old children of mothers with AN, BN and BED will exhibit depression and anxiety symptoms significantly more than children of non-ED mothers

3. To examine the relation between maternally reported feeding behaviors (restrictive feeding, pushy feeding, and use of food as reward) and infant eating behavior in 36 month old children.

Hypothesis 1. Maternally-reported restrictive feeding, pushy feeding, and use of food as reward will be significantly associated with eating behaviors in 36 month old children.

The current study has the potential to elucidate the impact of maternal EDs on feeding behaviors, child eating habits, and psychological symptoms–an imperative step in renewing efforts to understand environmental factors that may contribute to EDs and to develop effective interventions. Given strong research suggesting both a genetic and environmental role in the etiology of EDs, unraveling the effects of each contribution is critical to advance understanding of the causes of eating disorders. This study addresses specific environmental factors–maternal feeding behaviors and childhood eating behaviors–that may contribute to the cycle of risk in children of mothers with EDs.

To enhance readability, the umbrella terms "eating disorders" (EDs) and ED-related behaviors are employed. The first term includes several levels of definition. First, EDs encompass the DSM-IV diagnostic criteria for AN, BN, and BED. Second, it refers to

individuals who meet broader diagnostic definitions that reduce thresholds inherent to the formal criteria. ED-related behaviors refer to the core behavioral symptoms of EDs (e.g., restricting, binge-eating, purging via vomiting or laxatives).

The Significance of Eating Disorders

Eating disorders pose a significant public health burden. AN has the highest mortality rate of any psychiatric disorder (Millar et al., 2005; Sullivan, 1995; Zipfel, Lowe, Reas, Deter, & Herzog, 2000), and of those who survive, social maladjustment, low BMI and psychiatric symptoms may continue to be problematic (Bryant-Waugh, Knibbs, Fosson, Kaminski, & Lask, 1988; Crisp, Callender, Halek, & Hsu, 1992; Hall, Slim, Hawker, & Salmond, 1984; Halmi, Brodland, & Rigas, 1976; Ratnasuriya, Eisler, Szmukler, & Russell, 1991; Sullivan, Bulik, Fear, & Pickering, 1998; Theander, 1970).

However, these disorders also carry the burden of considerable stigma. The popular belief persists that EDs are purely sociocultural in origin and that sufferers somehow "choose" to develop these illnesses. An individual with an ED may be perceived as superficial, *deciding* to prioritize thinness above all else, and able to change her thoughts or behaviors of her own volition. While genetic, and biological research suggest strongly that solely sociocultural theories of EDs are misguided (http://www.nimh.nih.gov/events/edsummary.cfm), the persistence of this stereotype has impacted research productivity, and in turn, treatment options. Although advances have been made in the treatment of BN (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000; Bulik, Sullivan, Carter, McIntosh, & Joyce, 1998; Fairburn et al., 1995; Walsh et al., 2000), data are limited regarding efficacious treatment for AN (Berkman et al., 2006; Bulik, Berkman, Brownley, Sedway, & Lohr, 2007). New efforts to understand

the causes of EDs and to develop effective treatments are imperative in addressing these damaging illnesses.

The Epidemiology of Eating Disorders

Prevalence & Incidence

The prevalence of AN varies by sex and age. Lifetime estimates from female general populations range from 0.1% to 1.0% (Hoek, 1991; Walters & Kendler, 1995; Whitehouse, Cooper, Vize, Hill, & Vogel, 1992). From the National Comorbidity Replication sample, Hudson et al. (2007) reported lifetime prevalence estimates of DSM-IV AN to be 0.9%. Similar prevalence estimates exist in Norway, where the prevalence of AN in a female population is estimated to be around 0.4% (Götestam & Agras, 1995). Conflicting data exist regarding the incidence of ED. Some suggest that the incidence of AN is increasing (Eagles, Johnston, Hunter, Lobban, & Millar, 1995; Jones, Fox, Babigan, & Hutton, 1980; Lucas, Beard, O'Fallon, & Kurland, 1988; Moller-Madsen & Nystrup, 1992; Szmukler, McCance, McCrone, & Hunter, 1986; Willi & Grossman, 1983; Williams & King, 1987) , and others report no change (Hoek et al., 1995; Jorgensen, 1992; Nielsen, 1990; Willi, Giacometti, & Limacher, 1990).

BN is more prevalent than AN. Lifetime prevalence estimates using direct interviews range from 1-2.8% for women (Bushnell, Wells, Hornblow, Oakley-Browne, & Joyce, 1990; Garfinkel et al., 1995; Hudson et al., 2007; Johnson-Sabine, Wood, Patton, Mann, & Wakeling, 1988; Kendler et al., 1991; Rand & Kuldau, 1992). In Norway, the prevalence estimates of BN in females range from is 0.7-1.6 % (Götestam & Agras, 1995) . Like AN, BN is overwhelmingly more common in women and unequally distributed by age (Hoek, 1991). Annual incidence of BN ranges from 11.5 per 100,000 person-years (Hoek et al.,

1995) to 26.5 per 100,000 woman-years (Soundy, Lucas, Suman, & Melton, 1995).

Incidence as high as 82.1 per 100,000 woman-years has been reported in individuals between

the ages of 20-24 in a large Dutch sample (Hoek et al., 1995). In community samples,

prevalance estimates for BED range from 3.5-6.6% (Grucza et al., 2007; Hudson et al.,

2007).

Maternal Feeding, Child Eating and Psychological Symptoms in Eating Disorders

Maternal Feeding Patterns

Few studies have explored the impact of ED status on maternal feeding style.

Keeping in mind the limitations of low generalizability and failure to study EDs by subtype,

extant studies suggest that mothers with EDs tend to use food for non-nutritive purposes

compared to non-ED mothers (e.g., to communicate affection, or for reward or punishment of

her child) (Agras et al., 1999; Lacey & Smith, 1987). Fear of binge eating in the mother,

extending to her child, may also impact maternal feeding behaviors of mothers with EDs

(Fahy & Treasure, 1989; Patel et al., 2002; Russell et al., 1998), facilitating keeping smaller

amounts of food in the house than mothers without EDs (Russell et al., 1998; Stein et al.,

1994).

Mothers with EDs may have inaccurate perceptions of healthy eating behaviors in

their children in comparison to mothers with no ED history. Data suggest that mothers with

EDs were more likely than controls to report that their child was a slow eater (Agras et al.,

1999), a fussy or picky eater (Evans & le Grange, 1995), or an overeater, even when their

child was malnourished (Honjo, 1996).

Problems with maternal feeding can persist throughout the infant and toddler years. Several reports exist of mothers with AN underfeeding their children (Russell et al., 1998; Scourfield, 1995; Smith & Hanson, 1972; Stein et al., 2006; van Wezel-Meijler & Wit, 1989). Moreover, observational studies suggest women with histories of AN display more inappropriate responses during mealtimes with their infants and toddlers (Stein et al., 1994; Waugh & Bulik, 1999). By 1 year, mothers with ED are more likely to have engaged in mealtime conflict with their infants than control mothers (Stein, Woolley, & McPherson, 1999)

Studies of nonclinical samples have found that children of mothers who are highly controlling of food intake have reduced capacity to self-regulate their own diets (Johnson & Birch, 1994). These maternal behaviors may have serious consequences for children's future eating patterns in terms of potentially developing disordered eating patterns as well as compromising nutrition and health. Beyond modeling dysregulated eating for their children, these mothers may extend their behavior to feeding of their children. The mother may not pay attention to her own hunger and satiety cues, and in turn, be able to recognize those of her children. Potentially, this could have the effect of the child not being able to differentiate her own hunger signals. As she develops and begins to eat on her own, these factors may decrease her ability to self-regulate her own eating behavior. Even if the mother does not have an ED, if the child is at greater genetic risk for the potential development of an ED, the factors of witnessing dysregulated eating in the household, experiencing dysregulated feeding as a child, and ignoring the biological mechanisms that help regulate feeding may contribute to the development of an ED.

Eating and Health Problems in Children of Mothers with EDs

Few studies have examined eating and health issues in children of mothers with EDs across subtypes. Of the extant research, data suggest that children of mothers with AN were undernourished and weighed less than children of mothers without EDs even when there were no significant group differences in birth weights (Mazzeo, Zucker, Gerke, Mitchell, & Bulik, 2005; Russell et al., 1998; Stein, Murray, Cooper, & Fairburn, 1996). In a case review of seven children of mothers with AN histories, van Wezel-Meijler and Wit (1989) described children all of whom had stunting and low weight. When in the hospital, these children ate willingly and gained weight quickly, suggesting that their feeding problems were due to insufficient nutrition at home (Mazzeo et al., 2005; van Wezel-Meijler & Wit, 1989). Relatedly, pica and early digestive problems have been noted as risk factors for symptoms of BN in adolescence (Marchi & Cohen, 1990).

Following children and their parents for the first five years of life, one non-clinical prospective study found that risk for disordered eating behaviors increased annually in children of parents without EDs (Stice, Agras, & Hammer, 1999). Further, maternal dieting behavior, maternal body dissatisfaction, upholding a thin-ideal, and symptoms of BN predicted the onset of eating problems in childhood. In a non-clinical study of 99 women without EDs, Farrow and Blissett (2006) found maternal negativity during mealtime was associated with maternal reports of child eating difficulty in 6 month old infants. Another study conducted in a healthy population found that cognitions common in women with EDs–failure to achieve, dependence and incompetence, and shame beliefs were associated with maternal reports of feeding problems in 7-64 month old girls (Blissett, Meyer, Farrow, Bryant-Waugh, & Nicholls, 2005).

Psychological Symptoms in Children of Mothers with EDs

Depression. Major depression is commonly comorbid with EDs and evident in 55.6%- 68% of women with AN (Halmi et al., 1991; Herzog, Keller, Sacks, Yeh, & Lavori, 1992) and 47-73% of women with BN (Hudson, Pope, Jonas, & Yurgelun-Todd, 1983b; Lee, Rush, & Mitchell, 1985; Mitchell, Hatsukami, Pyle, & Eckert, 1986). Twin studies have reported a genetic correlation of .58 between AN and major depression (Wade et al., 2000), and BN and depression share some genetic factors (Walters et al., 1992). One study found that of the 77 women who met criteria for AN, 18 MZ and 20 DZ female co-twins (49.4%) also had a lifetime diagnosis of major depression (Wade et al., 2000). The best fitting model showed that the proportion of shared genetic variance between anorexia nervosa and major depression was 34% ([CI]=13%–71%). This means that there is a moderate shared genetic liability that may lead to the development of both disorders in affected individuals, as well as unique genetic and environmental factors influencing each. Regarding BN, in a sample of 1033 twin pairs, another study found that genetic factors accounted for a small amount of the variance between major depression and BN (0.456). Unique environmental factors contribute to around 50% of the difference in susceptibility of major depression and BN, and further, each disorder has its own specific environmental triggers. Overall, BN and depression are somewhat genetically correlated, and also have unique genetic and environmental risk factors.

Mood disorders are also significantly more likely to occur in family members of AN probands than controls, suggesting familial co-aggregation of the disorders. (Gershon et al., 1984; Hudson, Pope, Jonas, & Yurgelun-Todd, 1983a; Lilenfeld et al., 1998; Rivinus et al., 1984; Strober et al., 1990; Winokur, March, & Mendels, 1980). Together, these data support

the inclusion of depressive symptoms in children of mother with eating disorders as outcomes in the current analyses.

Anxiety and Obsessive Compulsive Symptoms

Anxiety disorders are also common in clinical and community samples of women with both AN and BN (Braun, Sunday, & Halmi, 1994; Brewerton et al., 1995; Bulik, Sullivan, Carter, & Joyce, 1996; Bulik, Sullivan, Fear, & Joyce, 1997; Deep, Nagy, Weltzin, Rao, & Kaye, 1995; Garfinkel et al., 1995; Godart, Flament, Perdereau, & Jeammet, 2002). In a large community sample, approximately two-thirds of participants with EDs had at least one lifetime anxiety disorder (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004). Approximately 41% of women with EDs had OCD, and 20% endorsed symptoms of social phobia, both predating the development of an ED. Other studies have also documented the emergence of anxiety disorders prior to ED symptoms (Bulik et al., 1996; Deep et al., 1995), suggesting the anxiety disorders as a potential risk factor for the development of EDs (Bulik, 1995; Schwalberg, Barlow, Alger, & Howard, 1992).

Shared genetic factors may also partially account for the comorbidity between anxiety and EDs (Kendler et al., 1995; Melke et al., 2001; Raney et al., 2008). Silberg and Bulik (2005) reported a developmental twin study that identified a unique genetic factor that contributed to early anxiety and early eating symptoms in juvenile twins. They found a common genetic factor influencing liability to overanxious disorder (OAD), separation anxiety disorder (SAD), and eating symptoms, and a common environmental factor influencing susceptibility to symptoms of later eating disorders and separation anxiety. Further, significantly elevated risk for anxiety disorders in family members of women with both AN and BN have been found relative to controls (Lilenfeld et al., 1998). This suggestive

evidence of familial co-aggregation of anxiety and OAD symptoms support the inclusion of anxiety and OAD symptoms in children of mothers with EDs as outcomes in the analyses.

Child Body Mass Index (BMI) and Food Allergies

Several additional factors could directly or indirectly influence the hypothesized maternal feeding habits and childhood eating behavior outcomes. Childhood overweight is a significant public health concern, and along with genetic factors, there is some evidence that it may be correlated with parental feeding style (Carnell & Wardle, 2007; Faith et al., 2004; Johannsen, Johannsen, & Specker, 2006). In a prospective study of 57 healthy families, among children predisposed to obesity, elevated child weight at ages 3, 5, and 7 appears to predict restrictive maternal feeding practices. Additionally, restrictive feeding practices appear to be ineffective, and produce additional weight gain in the children (Faith et al., 2004). Further, parents of leaner children tend to encourage their children to eat more than parents of children of normal weight (Carnell & Wardle, 2007).

Food allergies also tend to influence the way mothers feed their children. As the treatment of childhood food allergies is to avoid feeding problem foods (American College of Allergy, 2006), it is appropriate that most mothers of children with allergies would exhibit restrictive feeding regarding these types of foods. This type of food restriction is warranted given child health status, and differs in intent from restriction for the purpose child weight control. Significantly, childhood BMI and food allergies could confound accurate interpretation of data regarding maternal feeding and child eating in an ED population (i.e., childhood overweight may be a justified health reason for maternal food restriction; food allergies may influence a child's eating habits and a mother's feeding certain foods).

Theoretical Significance

The following is a theoretical model underlying the current study's hypotheses. At the core of this model is one core distinguishing feature across eating disorders subtypes, namely the presence or absence of binge eating. Whereas binge eating occurs in BN and BED, AN is marked more by restrictive eating patterns—although some binge eating can occur. This line of thinking is supported as follows: First, among individuals with EDs, those who endorse binging behavior may represent a distinct phenotype from non-bingers. Recent research on classification has found that the binge eating taxon was qualitatively different from overeating and that certain individuals were highly susceptible to the development of binge eating (Williamson, 2007; Williamson, Gleaves, & Stewart, 2005; Wonderlich, Crosby, Mitchell, & Engel, 2007). Further, data from a genetic study of 2163 female twins using bivariate modeling suggest that binge eating is moderately heritable, and a modest genetic correlation exists between binge eating and obesity (Bulik, Sullivan, & Kendler, 2003). Other studies corroborate this evidence, suggesting the heritability of binge eating to be between 41-82% (Bulik, Hebebrand et al., 2007; Bulik, Sullivan, & Kendler, 1998; Sullivan, Bulik, & Kendler, 1998). Binge eating appears to be a distinct behavioral phenotype, resulting from the interaction between genetic susceptibility and an environment which provides easily accessible, palatable foods.

Evolutionary theory may further explain the existence and persistence of binge eating. The ability to consume large amounts of food in one sitting may have been beneficial for those who lived in areas with periodic food scarcity, necessitating the ability to consume energy when available. Accordingly, binge eating may have ensured survival: a hunger signal in times of famine would initiate increasing energy stores as much as possible. Although the

mechanism that produces binge eating in some and not others is unknown, it is possible that in bingers, anxiety (once related to food scarcity) could trigger excessive eating. One finding suggests that bingers are more reward sensitive, and have greater anxiety, impulsivity, and addictive personality traits than non-binging healthy controls (Davis et al., 2007). Further, bingers may have different responses to hunger and satiety cues than non-bingers with EDs. While an individual with an ED who is not susceptible to binging may restrict and over time, mute her hunger and trick her satiety cues, an individual who is susceptible to binging may have a different interpretation of theses signals. She may restrict a few days a week, but after a while, be unable to quell her hunger, and respond with binging behavior. Alternately, she may never restrict, and binge at times of emotional dysregulation.

Williamson (2007) presents another theory: that people vulnerable to weight gain caused by excessive eating/binging recognize that they are at risk for weight gain. Living in a culture that stigmatizes overweight, they are provoked to restrict caloric intake or use other compensatory behaviors to control weight (e.g., excessive exercise, laxatives, vomiting). This explanation suggests that restriction is a product of the binge eating phenotype, which runs counter to the hypothesis that caloric restraint causes binge eating (Tuschl, 1990; Williamson, 2007).

Assuming a binge eating taxon, and that individuals respond to available food in different ways, it is possible that not only do different cognitive and emotional responses to eating exist but also different responses relative to the process of feeding offspring emerge (Figures 1 and 2). Faced with the task of feeding her child, mothers with different subtypes of eating disorders (i.e. binge eating or non binge eating) may interpret the situation through different lenses.

Non-Binging Eating Disorders

Several factors related to her ED phenotype may influence the lens through which a mother with a non binge-eating ED interprets the feeding task. First, her feeding behavior may be influenced by her core belief that gaining weight is associated with negative outcomes (e.g., low self esteem, rejection, failure). Other common ED core beliefs that may influence her feeding behavior include the perception that life is out of control, a distorted view of her body, and the belief that restrictive eating is normative. From a cognitive-behavioral standpoint, as core beliefs influence one's thoughts, feeling, and behaviors, cognitions such as "food is bad" or "I don't want a fat baby" would logically follow. These thoughts would likely produce significant anxiety, and lead to restrictive feeding behavior. Following this logic, it is possible that the non-binging ED mother's behavior may stem from the altruist motive of protecting her baby from negative consequences associated with perceived fatness.

Second, the non-binging mother's interpretation of the feeding task may also be influenced by personality traits common among individuals with AN, such as harm avoidance and drive for thinness (Lilenfeld et al., 2006). For example, the mother may restrict food to protect the infant from perceived harm associated with food intake, or to promote thinness/perfectionism. These characteristics may foster disordered core beliefs, distorted thinking such as "Food is bad" or "I don't want a fat child," and feelings of anxiety about having an overweight baby which could precede or follow these thoughts. Third, when faced with a negative emotion, a deficit in emotional regulation in individuals with non-binging EDs (Fassino et al, 2002) may make it more likely for them to react with restrictive

(or purging) behavior. Taken together, the features, thoughts and feelings associated with non-bingers make restriction of the infant's intake a likely outcome.
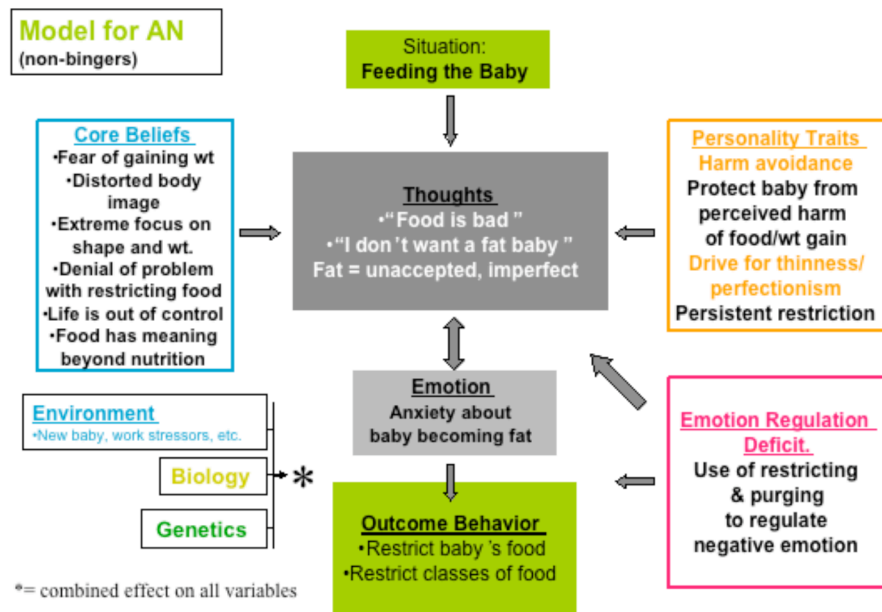
An additional critical point to consider is the impact of a maternal ED on maternal report of her feeding behavior and her child's eating behavior. As EDs are a highly stigmatized subject, a mother who has suffered from negative social impact of her disorder may desire to shield herself and/or her child from similar perceived shame or stigma. This may manifest in an underreporting of perceived restrictive maternal feeding behavior or abnormal child eating behavior. However, this theory assumes that a mother with a non-binging ED (a) has been significantly impacted by associated stigma, and (b) has the ability to objectively perceive disordered feeding or eating. Perhaps it is more likely that maternal report of feeding behavior and child eating behavior would be impacted by the mother's normalization of her own restrictive eating. For example, while a non-binging ED mother may restrict her child's eating, her tendency to restrict may be so normative that she does not see this behavior as restrictive when feeding her child. In the currently study, this must be considered when evaluating findings.

Gene-environment correlation must also be considered. Mothers not only provide their children their genes, but most often, the environment in which they are reared. Due to potential genetic underpinnings, growing up in an environment that models restriction, and the likelihood of family members with AN-associated personality and psychological traits, offspring of non-binging ED mothers may be more likely to endorse problematic eating behaviors than their healthy counterparts. Similarly, these children may be more likely to have obsessive-compulsive spectrum traits due to both genetic susceptibility and growing up in an environment modeling obsessive, restrictive behavior than their counterparts with

Figure 1:

Theoretical Model for AN group (non-bingers)

binging or healthy mothers. They may also be more likely to be depressed or anxious than children of non-ED mothers, due to the familial susceptibility of these psychological traits.
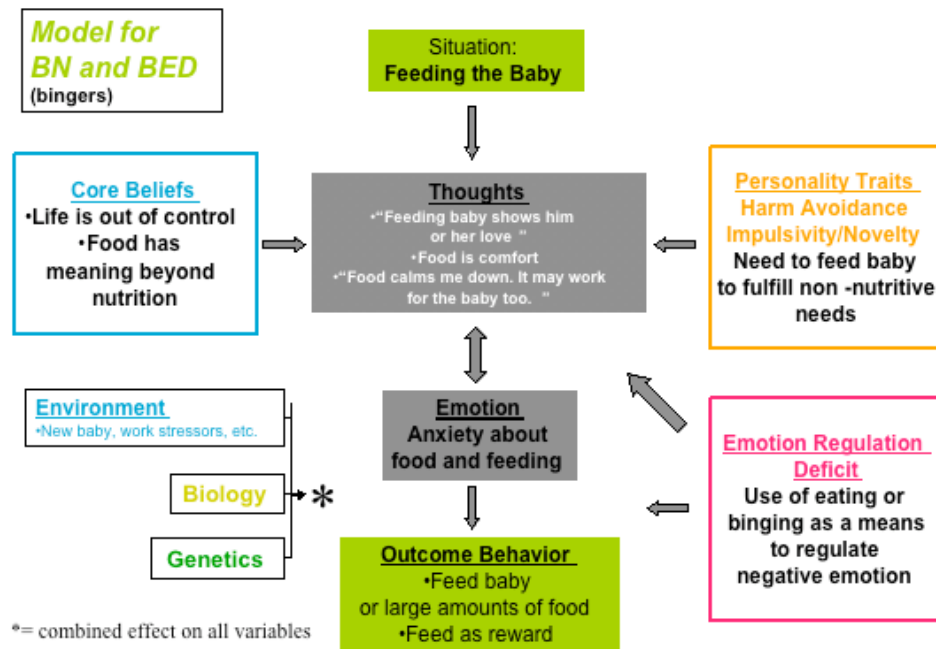
Binge Eating Disorder Subtypes

Presented with the situation of feeding her infant, a mother who has a history of an ED with binging features may react differently from her non-binging counterpart given a different set of psychological and associated factors (Figure 2). First, her core beliefs and associated cognitions and emotions must be considered. While also possessing the belief that life is out of control and that food has meaning beyond its nutritive purposes, a binge eater with an ED may place a different value on food. While an ED non-binger may possess a fear of food, a binger may define food at different times as comforting, calming, or even adversarial. This uniquely constructed core belief will lead to a different set of thoughts, such as "Food shows my baby love" or "Food is comforting." For a non-ED individual, these thoughts may seem somewhat neutral. However, for a mother who binges, for whom excessive eating often becomes an "out of control" reaction to emotion, these thoughts may trigger significant fear and anxiety. In turn, a mother with a binge-type ED may be triggered by this emotion to feed her baby in excess or use the food for non-nutritive purposes, such as a reward.

Second, the interpretation of feeding her baby will also be informed by personality traits associated with binging EDs, such as harm avoidance, leading her to protect the infant from negative emotions by using food to provide comfort or reward. Alternately, the personality feature of impulsivity may motivate feeding for the purpose of sensation seeking. These characteristics may help to foster distorted thinking such as "Feeding shows the baby love," "Food is comfort," or even "Food is the enemy." Anxiety and distress regarding the

Figure 2:

Theoretical Model for BN and BED groups (bingers)

food and feeding may still precede or follow these thoughts. Third, when faced with a negative emotion, a deficit in emotional regulation in individuals with a binging ED may make it more likely for them to react with feeding the baby excessive amounts of food. Given these collective factors, it is likely that outcome behavior would be to pressure the baby to eat, use food as a reward, or feed the infant larger amounts of food than normative.

As mentioned in the prior section, it is important to consider the impact of a maternal ED on maternal report of her feeding behavior and her child's eating behavior. It is possible that stigma may influence the mother to be conservative in reporting perceived abnormal eating behaviors in herself and her child. However, it is also possible that due to the binging ED mother's distorted perception of food, her report of her feeding behavior and the child's eating behavior would be skewed. For example, while she may pressure her child to eat, her emotionally-influenced understanding of food intake may lead her not to report her feeding as 'pushy.' Finally, the currently study predicts that children of BED and BN mothers may be more likely to be depressed or anxious than children of non-ED mothers, due to the familial susceptibility of these psychological disorders.

II. Preliminary Studies

Overview

The MoBa Study

The current study is based on the "Den norske mor og barn undersøkelsen" (MoBa, The Norwegian Mother and Child Cohort Study). Funded by the Norwegian Institutes of Public Health, MoBa is a prospective pregnancy/birth national cohort study of 100,000 births in Norway. Recruitment began in 1999 with funding from the Norwegian government and supplemental funding from non-government organizations. It aims to continue with recruitment until a cohort of 100,000 births is reached, with longitudinal follow-up of the births into adulthood. The target population of this study includes all pregnant women and the fathers of their children in Norway. The goals of MoBa are to find causes of serious diseases in mothers and children through investigating both genetic and environmental factors and their interactions (Norwegian Institutes of Public Health, 2008). Further details regarding these procedures are discussed in Section D.

Prevalence Estimates from the Initial MoBa study

Table 1 presents prevalence of EDs before and during pregnancy in the MoBa sample (Bulik, Von Holle et al., 2007). In a large cohort of pregnant women, this prospective study marks the first investigation of prevalence, remission, continuation, and incidence of broadly defined eating disorders. In this initial report, a total of 41,157 women had valid data available for analysis. Pre-pregnancy prevalence estimates were 0.1% for AN, 0.7% for BN, 3.5% for BED, and 0.1% for EDNOS-P. Estimates during pregnancy were 0.2 % for BN,

4.8% for BED, and < 0.1% purging disorder. Questionnaires did not allow for the assessment of AN during pregnancy. Compared to pre-pregnancy estimates from other studies, the observed pre-pregnancy prevalence of AN is on the low end of reported ranges (Götestam & Agras, 1995). However, estimates from the prior study are not based on pregnancy status. In turn, this discrepancy may be based on the specificity of the sample, including only pregnant women, potentially differing from other women with AN who may show impaired fertility (Stewart, Robinson, Goldbloom, & Wright, 1990), or low desire to have romantic relationships or sexual intercourse (Ruuska, Kaltiala-Heino, Koivisto, & Rantanen, 2003), decreasing their likelihood of becoming pregnant (Bulik, Von Holle et al., 2007).

Table 1:

Prevalence of Eating Disorders Before and During Pregnancy

Disorder	Before pregnancy N (%)	During Pregnancy N (%)	Before and During Pregnancy N (%)
Anorexia nervosa (broad)	37 (0.1%)	--	--
Bulimia nervosa (any type)*	290 (0.7%)	96 (0.2%)	67 (0.2%)
Bulimia nervosa (purging)	118 (0.3%)	52 (0.1%)	37 (0.1%)
Bulimia nervosa (non-purging)	109 (0.3%)	30 (0.1%)	17 (< 0.1%)
BED (broad)	1,405 (3.5%)	1,856 (4.8%)	779 (2.0%)
EDNOS-P**	42 (0.1%)	12 (< 0.1%)	5 (< 0.1%)

* BN any type also includes those individuals who could not reliably be categorized as purging or non-purging due to missing data.; **Purging in the absence of binge eating.

III. Research Design and Methods

The Study Sample

Recruitment and the Establishment of the MoBa Study Population

The MoBa birth cohort study began recruitment from all counties in Norway in 1999, and continued recruitment until April 2008, when 100,000 pregnant women were consented to participate. In recruiting women through ultrasound laboratories in public hospitals and private clinics throughout Norway, MoBa comes in contact with the majority of pregnant women in the country. In Norway, all delivery wards/hospitals are part of the public health care system, most deliveries take place in these institutions, and antenatal care is free. Almost all mothers receive antenatal care and ultrasound examinations before 20 weeks gestation.

Using lists of scheduled appointments at ultrasound labs, MoBa recruits women around two weeks before the routinely performed ultrasound examination (c. 17th week) in pregnancy. Either on paper or electronically, the lists are obtained by the central data facility in Bergen and invitations are generated electronically for mailing. Invitations are not sent if the mother has spontaneously aborted the child. This is important to consider that due to the study's omission of individuals with abortions, the incidence of EDs in the Norwegian population may be underrepresented in the sample. Interested women receive an information folder, complete informed consent, and then receive two questionnaires on diet and health history. If any form is not returned, the woman is approached by a nurse at the ultrasound clinic and asked again to participate.

Following the consenting procedure, the central facility gains access to the Medical Birth Registry, containing information on all pregnancies and childbirths in Norway. This resource affords the opportunity to access detailed health information, and make comparisons between participants and non-participants. A preliminary comparison suggests that MoBa participants tend to be somewhat older (mean 29.2 years versus 28.4) and better educated (56% completed more than 13 years of school versus 33%) than the general population. Rigorous analyses were conducted to identify participation bias in the MoBa and the current study's aims will be minimally impacted by participation bias due to case: control comparisons. Further, the current study performed analyses to assess differences on relevant demographic variables in the sample, and ultimately, did not control for bias.

Current Participation Levels

At the time of the current study, MoBa had enrolled over 72,192 pregnancies. To this point, approximately 42% of invited mothers have agreed to participate in the study. While the response rate is somewhat low, it is not unusual for large studies and is not necessarily associated with sample bias (Hartge, 2006). However, each substudy regularly checks for the possibility of selection bias. One example would be the tendency for research participants to be from a higher socioeconomic bracket than non-participants. Figure 3 describes the data flow further.

The analysis population for the current study included MoBa participants who: (a) had clean data from the MBR (N=63,182), and MoBa Questionnaires 1 (N=51,048), and 6 (N=15,527), (b) had valid values for demographic, predictor, and outcome variables of interest (N=14,189), and (c) had clean data and a singleton birth (N=66,212). If a woman had more than one pregnancy during the study period, only the first pregnancy in the study period

is included in this analysis. Of the 14,189 pregnancies meeting these criteria, 13,006 were included in the analysis population after accounting for those with missing ED status.

In terms of response rates, the first two questionnaires were completed by 94% of women who consented to participate and 90% of women consented to the blood collection. Over 90% of fathers accompanied their partner to this examination, approximately 94% consented to participate, and 85% provided a blood sample. Regarding a questionnaire sent to pregnant women at 30 weeks' gestation, 92% were completed and returned. At the time of birth, 85% of mothers agreed to give umbilical cord blood and maternal blood. Finally, 88% of mothers completed and returned the 6 month questionnaire, and 77% percent completed the 18 month questionnaire. Overall, across the first three questionnaires, the average response rate exceeds 92%.

Assessments

Overview

The assessment batteries developed for the MoBa study are divided into three variable sets: exposure variables, health outcome variables, and other variables. In short, an exposure variable is one named in the study aims as a potential causal factor. A health outcome variable is defined as one that describes or defines a health condition, either from the questionnaire, registry or blood sample. Other variables refer to those which do not meet the aforementioned categories (e.g., socioeconomic status).

Assessment in the MoBa protocol occurs in five waves. The first occurs at 17 weeks prenatal, when the mother is sent an invitation to participate, Questionnaire I (QI), regarding maternal health and reproductive history, medications, dietary supplements,

sociodemographic information (maternal/paternal), work hours and environment, household environment, health habits, eating disorders, weight, physical activity, psychological health, social support, and QII on dietary information. At 30 weeks prenatal, the mother completes a questionnaire regarding obstetric history, exposure history, diet and dietary supplements, work conditions, physical activity, current exposures (smoking, travel, animals), health habits, psychological, social support, and weight. At childbirth, the Medical Birth Registry records birth and infant health information. At 6 months after birth, Questionnaire 4 is sent, regarding child and maternal health, nutrition, and growth and development. At 18 months after birth, Questionnaire 5 is completed by the mother, again regarding child and maternal health, nutrition, and growth and development. Variables from the questionnaire taken at 36 months are described below. The current study used demographic data from the Medical Birth Registry, assessment data relating to maternal feeding styles, child eating behaviors and psychological symptoms from the questionnaire sent at 36 months, and food allergies (present or absent) from the 36th month questionnaire. Queried for the periods of 6 months prior to pregnancy or during pregnancy, ED status was taken from diagnostic questions asked in the 17 weeks prenatal assessment.

Detailed Information on Selected Areas of Interest

The assessment battery for MoBa is extensive. Rather than reproduce the entire battery, the following are detailed descriptions of the areas of interest relevant to the current study.

Eating disorders and related behaviors. In addition to height and weight, all women are asked diagnostic questions relevant to eating disorders. The ED questions in MoBa were taken directly from the Norwegian Twin Panel assessment of eating disorders. These
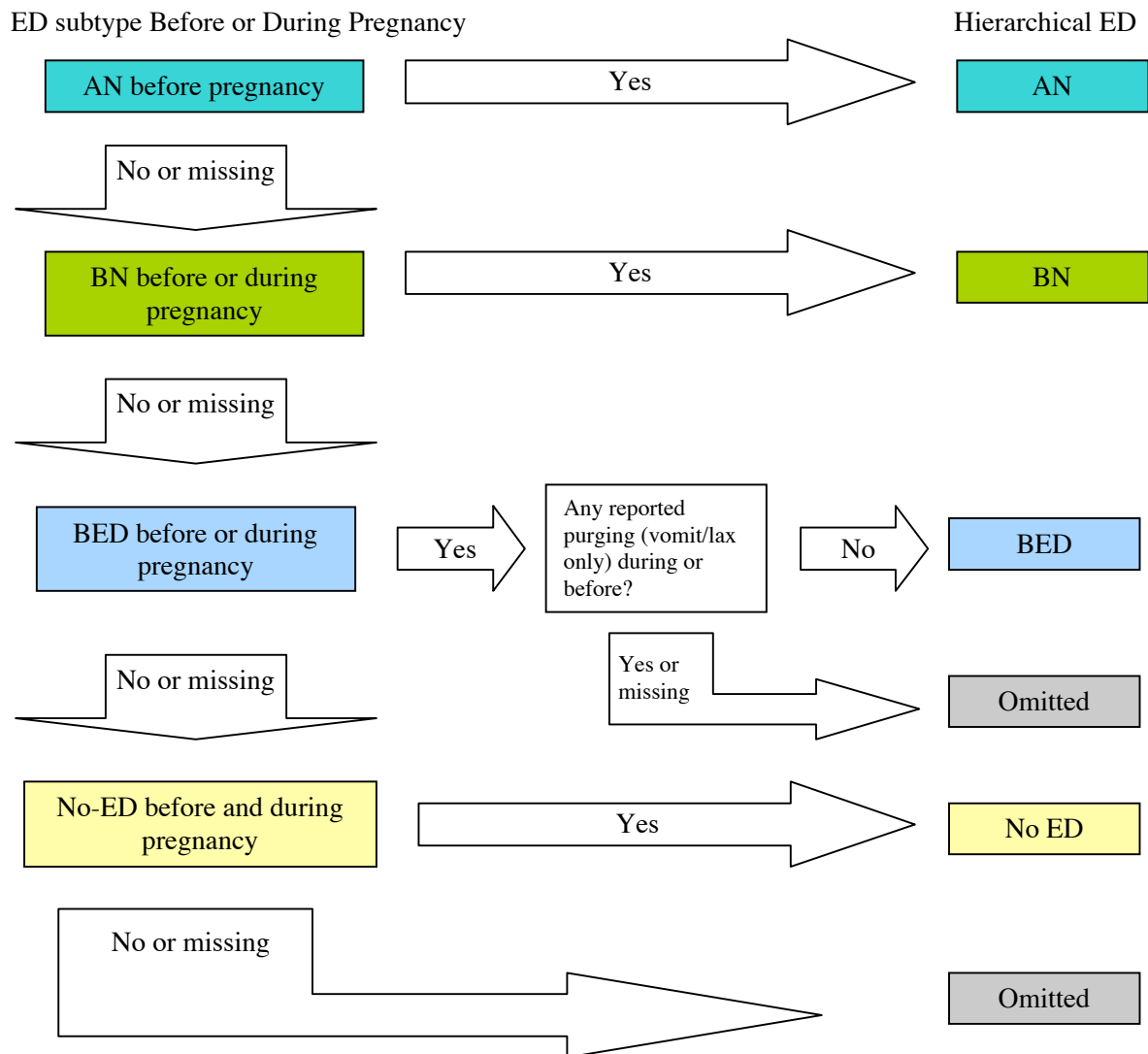
questions have been used widely in international twin studies (Reichborn-Kjennerud et al., 2003; Reichborn-Kjennerud, Bulik, Tambs, & Harris, 2004) and yield replicated prevalence estimates. Prevalences for core ED behaviors are included in Table 1 above. Diagnostic algorithms were constructed from the 17 weeks prenatal questionnaire items to define the presence of broadly defined anorexia nervosa (AN, amenorrhea not required), broadly defined bulimia nervosa (BN, at least weekly frequency of binge eating and purging), broadly defined binge eating disorder (BED, at least weekly frequency of binge eating), and purging in the absence of binge eating (EDNOS-P, purging at least weekly without binge eating) (Bulik, Von Holle et al., 2007). AN was assessed before pregnancy *only* due to practical difficulties in determining low weight in the presence of pregnancy-related weight gain. BN, BED, and EDNOS-P were assessed for both 6 months prior to pregnancy (retrospective assessment) and at the time of survey completion. Self reported weight and height were used to calculate pre-pregnancy body mass index (BMI, kg/m^2) and BMI at the time of assessment. Respondents were specifically asked to distinguish between pregnancy-related vomiting and self-induced vomiting as a purging method. Respondents completed questionnaire 1 at a median of 18.1 weeks gestation with inter-quartile range of 16.7-20.1 weeks, and range 5.3-41.9 weeks. Before or during pregnancy, BED and BN are mutually exclusive diagnoses as determined by the classification algorithm. The category BN included individuals who responded to questions that defined non-purging BN (i.e. fasting and exercise), purging BN (i.e. laxatives and vomiting), and individuals who could not reliably be categorized as purging or non-purging due to missing data.

To establish these diagnostic categories, a hierarchical algorithm was created to avoid overlap if individuals endorsed multiple disorders at different time points (e.g., AN before

pregnancy *and* BN during pregnancy). The hierarchy's order of group assignment is as follows: AN, BN, BED, and Non-ED. Individuals included in the AN group reported meeting diagnostic criteria before pregnancy; data on the presence of AN during or after pregnancy do not exist. If a participant endorsed criteria for AN before pregnancy, she was included in the AN group. If she did not meet AN criteria or data were missing, criteria for BN *before or during pregnancy* was examined for this participant. If she met BN criteria at either time point, she was included in the BN group. Binging status was included because endorsing both binging and purging but not endorsing BN suggest incongruent data. If she did not meet BN criteria, or BN data were missing, she was not included in this group, and criteria for BED before or during pregnancy was examined. If she endorsed BED and did not endorse purging during or before pregnancy, she was included in the BED group. Again, purging status was included because endorsing both purging and binging (via BED criteria) but not endorsing BN suggests incongruent data; participants were omitted if they endorsed purging, or purging data were missing. If she did not meet BED criteria or BED data were missing, criterion for non-ED status before or during pregnancy was examined. If she endorsed non-ED status, she was included in the non-ED group; if not or data were missing, she was omitted from analyses. In the current study, 4 diagnostic groupings were examined based on ED subtype: (1) AN, (2) BN, (3) BED, and (4) a non-ED group. The EDNOS group was not included in the analyses because too few participants met criteria for the sub-threshold category. See Figure 3.

   Medical Birth Registry Form. Information from the Medical Birth Registry Form is available both for the infants in this study as well as from their mothers' births. The Medical Birth Registry Form includes a variety of items that assess pregnancy outcome, fetal weight,

Figure 3:
Eating Disorder Hierarchy Algorithm

ED subtype Before or During Pregnancy   Hierarchical ED

| AN before pregnancy | → Yes → | AN |

No or missing ↓

| BN before or during pregnancy | → Yes → | BN |

No or missing ↓

| BED before or during pregnancy | → Yes → | Any reported purging (vomit/lax only) during or before? | → No → | BED |

→ Yes or missing → Omitted

No or missing ↓

| No-ED before and during pregnancy | → Yes → | No ED |

No or missing ↓ → Omitted

placental weight, and maternal and fetal complications, plus parental circumstances at the time of birth (e.g. marital status, employment status, and smoking during pregnancy). The current study looked at maternal age, marital status, total number of previous live births, maternal education, child sex, birth weight, Apgar scores, gestational length, and gestational age.

   Maternal feeding. From the 36 month questionnaire, the study used measurement of maternal feeding practices derived from the Child Feeding Questionnaire (Birch et al., 2001), an instrument for assessing parental control in child feeding. Specifically, *Restriction* and *Pressure to Eat* subscales were used for the analyses, the only available in the MoBa questionnaires. Validated by Birch et al. (2001), *Pressure to Eat* is a 4-item scale that measures the degree to which the mother encourages the child to eat by behaviors such as insisting that the child eat everything on his or her plate. The questions are as follows: (1) My child should always eat all the food on her plate, (2) I have to be especially careful to make sure my child eats enough, (3) If my child says, "I'm not hungry", I try to get him/her to eat anyway, (4) If I did not regulate my child's eating, she would eat much less than she should. The *Restriction* subscale has been validated using an 8-item scale, for which the 36 month questionnaire offers 5 items. The Restriction subscale statements used in the current study are as follows: (1) I have to be sure that my child does not eat too many sweets (candy, ice cream, cake or pastries), (2) I have to be sure that my child does not eat too many high fat foods, (3) I have to be sure that my child does not eat too much of his/her favorite foods, (4) I intentionally keep some foods out of my child's reach, (5) if I did not guide or regulate my child's eating, he/she would eat too much of his/her favorite foods. The other subscale items were not available in the study questionnaire. The current study also looked at using food as

reward using the Child Feeding Questionnaire item "I offer sweets (candy, ice cream, cake, pastries) to my child as a reward for good behavior." All items is scored from 1 (low restriction/pressure to eat) to 5 (high restriction/pressure to eat).

Child eating. Using the 36 month questionnaire, the study measured child eating habits, including items relating to (1) not eating well, (2) having stomach aches or cramps, (3) vomiting without medical cause, and (4) not enjoying eating. Measurement of eating problems was taken from the CBCL - Child Behavior Checklist (Achenbach, 1991), internationally the most widely used instrument to assess early behavioral problems in children (and toddlers). Food allergies (present or absent) were obtained from maternal report in the 36 months questionnaire. Each item is scored from 1 (Not True) to 5 (Very True).

Child growth, development, and psychological symptoms. From the 36 month questionnaire, the current study used the child age, weight and length measurements to compare to population norms of weight-for-age at 36 months using an algorithm provided by the Centers for Disease Control (2005). From the 36 month assessment, child anxiety, depressive, and obsessive-compulsive symptoms were examined. Questions regarding anxiety were taken from the related subscale of the CBCL (Achenbach, 1991). The anxiety subscale includes items relating to (1) the child being too dependent, (2) getting too upset when separated from parents, and (3) being too fearful or anxious. Measurement of depressive and obsessive compulsive-type traits were taken from subscales of the ITSEA - Infant Toddler Social Emotional Assessment (Carter, Little, Briggs-Gowan, & Kogan, 1999). The depressive subscale includes items regarding (1) lack of energy, (2) having "less fun than other children," and (3) seeming "very unhappy, sad or depressed." The OCD-type behaviors are part of the atypical index and general anxiety subscale of the ITSEA (Carter,

Briggs-Gowan, Jones, & Little, 2003), and items included in the current study were (1) worry about getting dirty, (2) needing things to be clean or neat, and (3) displaying repetitive ordering behavior. All items are scored from 1 (Not True) to 5 (Very True). This widely used measure covers phenomena of importance for early developmental problems and is not overlapping with questions found on the CBCL.

Statistical Analysis Overview

The first basic aim of this study was to describe the data collected. Quantitative descriptive statistics (means and standard deviations), and qualitative descriptive statistics (frequency distributions) were used, along with univariate and bivariate plots. First, comparisons across the four groups (AN, BN, BED, and No ED) were made on a small number of socio-demographic characteristics to ensure that the groups did not differ significantly. Next, for the first two aims, all possible pair-wise comparisons among the four groups were conducted using Tukey's HSD post hoc test to explore group differences at the significance level of 0.05. Exploratory analyses were also conducted to make comparisons within each group. For the final aim, correlations and regression analyses were performed.

Data Management

Data transfer occurred via encrypted files over the internet and via physical transfer of storage media. No identifying information was handled in the current study. Data analysis was performed using the software packages, SAS (SAS Institute Inc., 1999) and JMP (SAS Institute Inc., 1994).

Missing Data Considerations / Initial Analyses

Estimates from the first available subset of data suggested that there were few missing data: the proportion of missing data across assessments ranges from <1-2%.

Nonetheless, some missing data existed. Using JMP (SAS Institute Inc., 1994), missing data or impossible values were examined by performing range and value checking for all key variables. Examples of impossible variables included miscoded (e.g., 999) or blank coded variables, or column shifted variables (e.g., data for one variable entered into the wrong column). Further, using code in SAS, numerical limits were placed on each variable to eliminate impossible and missing values. For continuous variables, quantitative statistics were used to calculate means and standard deviations, plot the data and search for outliers, and created distributional plots and measures of central tendency and spread.

As the MoBa sample consists of a subset of the women in the birth registry who chose to volunteer for study, it was important to examine potential differences between this subset and the women who chose not to volunteer in order to assess any participation bias. Descriptive statistics were used to examine predictors of participation such as age, education, and socioeconomic status.

Computation and Programming

Dr. Bulik and the research team were responsible for transporting data from Norway to the US electronically. The team in Norway was responsible for the statistical programming tasks necessary to prepare the data for transport; all data preparation and analyses were conducted by Ms. Reba-Harrelson.

Statistical Analysis of Specific Aims

Specific Aim 1

To compare feeding attitudes and practices in mothers with broadly defined EDs across diagnostic subtypes (AN, BN and BED) with mothers without EDs. ED status was measured at 6 months prior to pregnancy or during pregnancy (asked 17 weeks prenatal).

After data were cleaned, SAS/STAT ® software, version 9.1 (SAS Institute Inc., 2000-2004) was used to perform statistical analyses for each aim. For the independent variable, mothers were classified into 4 groups based on available ED subgroups: (1) AN, (2) BN, (3) BED, (4) a non-ED group. Those with BN, BED, and no-ED were defined at *one of two* periods of time (before pregnancy *or* during pregnancy). For AN, there was only one diagnostic grouping: before pregnancy. AN was assessed before pregnancy only due to practical difficulties in determining low weight in the presence of pregnancy-related weight gain. BN and BED were assessed for both 6 months prior to pregnancy (retrospective assessment) and at the time of survey completion. The response variables for this aim included maternal feeding practices (as assessed by a 5 questions subscale regarding mother's use of restriction, a 4-question subscale regarding maternal pressure to eat, and one question regarding use of food as a reward when feeding her child). Child weight-for-age and child food allergy status were added to the model as covariates to prevent confounding for feeding behavior related to child overweight or food allergy.

Regarding descriptive statistics, percent distributions of categorical outcome variables across ED subtype including marital status and maternal education, BMI, maternal age, and parity were calculated. For continuous variables, the mean, median, standard deviation, number of observations, and number of missing observations were also computed.

First, comparisons were made across the four groups (AN, BN, BED, and No ED) on a small number of socio-demographic characteristics to ensure that the groups did not differ appreciably. Next, for each hypothesis, all possible pair-wise comparisons were conducted between the four groups using Tukey's honest significant difference (HSD) post hoc test to explore group differences at the significance level of 0.05. For the continuous response

variables, ANOVA was used to describe the relation between ED status and each predictor variable. When appropriate, the Kruskal-Wallis nonparametric test was used to assess the sensitivity of the ANOVA. This test does not assume that samples are normally distributed. Finally, exploratory analyses were conducted to make comparisons within each group.

Specific Aim 2

To compare maternally-reported eating behaviors and psychological symptoms (i.e., depression, anxiety, and obsessive-compulsive symptoms) in 36 month old children of mothers with EDs across diagnostic subtypes (AN, BN and BED) with children of mothers with no EDs.

For the independent variable, mothers were classified into 4 groups based on available ED subgroups: (1) AN, (2) BN, (3) BED, (4) a non-ED group. BN, BED, and no-ED were defined at *one of two* periods of time (before pregnancy *or* during pregnancy). Again, for AN, there was only one diagnostic grouping: before pregnancy. The response variables for this aim included child feeding habits, child anxiety, depressive, and obsessive-compulsive symptoms. Child weight-for-age status at 36 months and child food allergies (present or absent) were added to the model as covariates to prevent confounding for reported child eating behavior related to child overweight or food allergies.

Regarding descriptive statistics, the mean, median, standard error, number of observations, and number of missing observations for categorical outcome variables were calculated across ED subtype including the child's sex, birth weight, Apgar scores (1 minute and 5 minutes), gestational length, and gestational age. Similar to Aim 1, comparisons were made across the four groups (AN, BN, BED, and No ED) to ensure that the groups did not differ appreciably. All possible pair-wise comparisons were conducted for each hypothesis

among the four groups using Tukey's HSD test to explore group differences at the significance level of 0.05. ANOVA was used to describe the relation between ED status and each continuous predictor variables. To make comparisons within each group, exploratory analyses were conducted.

Specific Aim 3

To examine the relation between maternally reported feeding behaviors (restrictive feeding, pushy feeding, and use of food as reward) and infant eating behavior in 36 month old children.

To examine the relation between maternal reported feeding behaviors and child eating behavior, models were created corresponding to restrictive feeding, pushy feeding, and use of food as reward. ANOVAs were used to describe the relation between feeding behaviors and the predictor variable, child eating. Child weight-for-age and child food allergy status were added to each model as covariates to prevent confounding for feeding behavior related to child overweight or food allergy.

<div align="center">*Statistical Power*</div>

Power is the probability, given specific values for the null and alternative hypotheses, that the analysis will reject the null hypothesis when indeed it should be rejected. In the current study, power and sample size calculations were conducted. Of the 13,006 available participants for analyses, 5.70% had ED status (including AN, BN or BED). Due to missing data, a small proportion of women were unable to be classified, but the proportion of missing data was relatively small (8.34%). G*Power3 (Faul, Erdfelder, Lang, & Buchner, in press) was used for power and sample size calculations. We did power and sample size calculations

for a "small" effect size, defined as d=0.20, in the context of a one-way, four-group ANOVA (Cohen, 1988).

Assuming alpha = 0.05, and a total sample size of 13,006, with available sample sizes of those with AN, BN, BED, and No ED, we found >90% statistical power would be achieved for d = 0.20. Thus, for these analyses, the sample is sufficiently powered to reject the null hypothesis for small effect sizes.

IV. Results

Demographic Characteristics Mothers and Children

Of the 14,189 participants in the sample including the variables of interest, 13,006 were available after merging the datasets for each questionnaire and accounting for missing and impossible variables. Figure 3 describes data flow in further detail. Characteristics of all participating mothers are found in Table 2. Consistent with initial reports on the MoBa sample (Bulik, Von Holle et al., 2007; Magnus et al., 2006), the majority of the women were between the ages of 25-34, approximately 95% were married or cohabitating, and individuals were relatively well educated with approximately 77% attending junior college or higher.

Prevalence of EDs and Associated Characteristics

Prevalence of EDs

Seventeen (.13%) met criteria for AN, 98 (.75%) met criteria for BN, 634 (5%) met criteria for BED, and 12,257 (94%) had no ED. The nine individuals with EDNOS (.07%) were excluded from the analyses due to lack of sufficient power to detect significant differences.

Demographic Characteristics

Maternal age at childbirth differed significantly across groups, $F(3, 13,002) = 3.69$, p < .01. Mean age at childbirth of individuals with AN prior to pregnancy was 26.53 years (SD = 3.30), lower than individuals with BN and BED, 30.16 years (SD = 4.94) and 30.09 years (SD = 4.43), or no ED, 29.91 years (SD = 4.46). Parity differed significantly across group,

F(3, 13,002) = 4.39, $p < .004$. Mothers with BED were significantly more likely to have a greater mean number of births than those with No ED. The following had 1 or fewer births before the pregnancy related to the current study: 94% of women with AN, 76% of women with BN, 77.% of women with BED, and 82% of women with no ED. No significant differences emerged regarding partnered status. However, individuals with no ED were slightly more likely to be married or cohabitating (95%) than those with AN (94%), BN (92%), or BED (93%). Maternal BMI differed significantly across groups, F(3, 11,901) = 53.22, $p < .0001$. Expectedly, mothers with AN had lower mean BMIs than all other groups: Women with AN had a mean BMI of 20.06 kg/m_ ($SD = 1.90$), lower than those with BN, 25.56 kg/m_ ($SD = 5.62$), BED, 26.86 kg/m_ ($SD = 5.36$), or no ED, 24.66 kg/m_ ($SD = 4.28$). Mothers with BED had significantly higher mean BMIs than those with BN and no ED. However, mean BMI of women with BN and BED was in the overweight range as defined by the CDC (BMI between 25.0-29.9 kg/m_). The overall sample was well educated: 73.42% of women with AN, 71.42% of BN, 72.92% of BED, and 74.23% of no ED had at least a 3-year high school general studies or junior college education. However, maternal education differed significantly across group F(3, 12365) = 9.77, $p < .001$. Mothers with BED were more likely to be less educated than those with No ED.

Characteristics of the offspring are found in Table 3. Groups did not differ appreciably across demographic characteristics. Sex of infant was male for 47% of women with AN, 50% of BN, 54%, of BED, and 51% of no ED. Mean length at birth (cm) of children of mothers with AN was 50.73 cm ($SD = 1.39$), 50.14 cm ($SD = 2.33$) for children of BN, 50.58 cm ($SD = 2.18$) of children of BED, and 50.41 cm ($SD = 2.44$) for No ED.

Figure 4:

Data Flow

Table 2:

Demographic Characteristics of Mothers across ED subtype

Characteristic	Entire Sample N(%)	AN N(%)	BN N(%)	BED N(%)	No ED N(%)
Maternal Age at Childbirth					
<25	1,604 (11.30)	5 (29.41)	16 (16.33)	75 (11.83)	1,360 (11.10)
25-29	4,971 (35.03)	9 (52.94)	24 (24.49)	213 (33.60)	4,339 (35.40)
30-34	5,395 (38.02)	3 (17.65)	38 (38.78)	253 (39.90)	4,684 (38.21)
35+	2,219 (15.64)	0 (0.00)	20 (20.40)	93 (14.67)	1,874 (15.29)
# Previous Live Births					
0	6,677 (47.06)	11 (64.71)	41 (41.84)	268 (42.27)	5,845 (47.69)
1	4,791 (33.76)	5 (29.41)	33 (33.67)	223 (35.17)	4,147 (33.83)
2+	2,721 (19.18)	1 (5.88)	24 (24.49)	143 (22.56)	2,265 (15.08)
Maternal BMI, 36 months after childbirth (kg/m_)					
<18.50	1,451 (10.)	4 (23.53)	7 (7.14)	66 (10.41)	1,233 (10.06)
18.50-24.99	7,723 (54.53)	12 (70.59)	52 (53.06)	256 (40.38)	6777 (55.29)
25.00-29.99	3,495 (24.63)	1 (5.88)	20 (20.41)	169 (26.66)	3,019 (24.63)
≥30.00*	1,520 (10.71)	0 (0.00)	19 (19.39)	143 (25.56)	1,228 (10.02)

Marital Status					
Married	8,581 (64.08)	9 (56.25)	54 (56.84)	351 (59.19)	7,489 (64.66)
Cohabitating	4,076 (30.44)	6 (37.50)	33 (34.74)	203 (34.23)	3,482 (30.06)
Unmarried/single	478 (3.57)	1 (6.25)	6 (6.32)	25 (4.22)	397 (3.43)
Divorced/Separated/ Widowed	206 (1.54)	0 (0.00)	1 (1.05)	13 (2.19)	170 (1.47)
Other	51 (0.38)	0 (0.00)	1 (1.05)	1 (0.17)	44 (0.38)
Educational Status					
<3 years high school	1,117 (8.28)	1 (7.14)	9 (9.90)	66 (11.09)	880 (7.54)
Vocational high school	1914 (14.19)	3 (21.43)	17 (18.68)	95 (15.86)	1,603 (13.74)
3-year high school general studies, junior college	2,348 (17.41)	5 (35.71)	17 (18.68)	117 (19.53)	1,996 (17.11)
Regional technical college/4-year university degree	5,771 (42.79)	4 (28.57)	33 (36.26)	256 (42.74)	5,062 (43.39)
University, technical college, more than 4 years	2,336 (17.32)	1 (7.14)	15 (16.48)	65 (10.85)	2,124 (18.21)

Table 3:

Demographic Characteristics of Children across Maternal ED status

Characteristic	Entire Sample N(%) or Mean (SD)	AN N(%) or Mean (SD)	BN N(%) or Mean (SD)	BED N(%) or Mean (SD)	No ED N(%) or Mean (SD)
Sex of child (Male)	7,246 (51.07)	8 (47.06)	49 (50.00)	341 (53.79)	6,246 (50.96)
Gestational length (cm)	50.42 (2.41)	50.73 (1.39)	50.14 (2.33)	50.58 (2.18)	50.41 (2.44)
Weight at birth (kg)	3.63 (0.52)	3.60 (0.49)	3.61 (0.64)	3.66 (0.52)	3.62 (0.53)
Gestational age based on ultrasound (days)	279.16 (17.70)	280.94 (7.95)	278.44 (13.22)	279.05 (12.15)	279.20 (12.80)
Apgar Score at 1 minute	8.65 (1.14)	8.76 (0.90)	8.63 (1.12)	8.68 (1.11)	8.65 (1.14)

Mean birth weight (kg) of children of mothers with AN was 3.60 kg (*SD* = .49), 3.61 kg (*SD* = .64) for children of BN, 3.66 kg (*SD* = .52) for children of BED, and 3.62 kg (*SD* = .53) for No ED. Mean gestational age (ultrasound) of children of mothers with AN was 280.94 days (*SD* = 7.95) , 278.44 days (*SD* = 13.22) for children of mothers with BN, 279.05 days (*SD* = 12.15) of children of mothers with BED, and 279.20 days (*SD* = 12.80) for children of mothers with No ED. The mean gestational age fell in the category of normal gestation (average gestational age approximately 280 days) (O'Reilly, 2007). Mean Apgar score at 1 minute of children of mothers with AN was 8.76 (*SD* = .90), 8.63 (*SD* = 1.12) for children of mothers with BN, 8.68 (*SD* = 1.11) for children of mothers with BED, and 8.65 (*SD* = 1.14) for children of mothers with No ED. The mean Apgar score met healthy criteria (>7) for the 1 minute Apgar test (Finster & Wood, 2005).

Specific Aim 1: Maternal Report of Restrictive and Pushy Feeding, and using Food as Reward

Maternal reports of three problematic feeding behaviors_restrictive feeding, pushy feeding, and using food as reward were examined to assess whether mothers' ratings differed across those with AN prior to pregnancy, BN, BED, and no ED. Each item was adapted by MoBa from the Child Feeding Questionnaire (Birch et al., 2001). The relation between ED status and the predictor variables restrictive and pushy feeding behaviors were explored using ANOVA. All pair-wise comparisons across the four groups were conducted with Tukey's HSD post hoc test at the significance level of .05. Unadjusted means for each analysis are reported in Table 4.

Table 4:

Unadjusted Means and SDs for each analysis

Outcome Variables	AN		BN		BED		No ED	
	N (%)	Mean (SD)	N (%)	Mean (SD)	N (%)	Mean (SD)	N (%)	Mean (SD)
Restrictive Feeding*	16 (0.12)	**2.25 (0.94)**	95 (0.73)	**2.71 (0.94)**	598 (4.60)	**2.64 (0.87)**	11,557 (88.86)	**2.46 (0.90)**
Pushy Feeding**	16 (0.12)	**2.02 (0.99)**	95 (0.73)	**2.43 (0.94)**	597 (4.59)	**2.39 (0.90)**	11,549 (88.80)	**2.23 (0.85)**
Food as Reward*	16 (0.12)	**1.63 (1.02)**	92 (0.71)	**1.68 (0.90)**	595 (4.57)	**1.91 (1.06)**	11,475 (88.23)	**1.73 (0.98)**
Disordered Eating, Child*	17 (0.13)	**1.24 (0.36)**	98 (0.75)	**1.34 (0.34)**	629 (4.84)	**1.32 (0.32)**	12,139 (93.33)	**1.27 (0.31)**
Anxiety Symptoms, Child*	17 (0.13)	**1.24 (0.23)**	98 (0.75)	**1.32 (0.37)**	629 (4.84)	**1.31 (0.33)**	12,130 (93.26)	**1.24 (0.31)**
Depressive Symptoms, Child	17 (0.13)	1.04 (0.11)	98 (0.75)	1.07 (0.17)	630 (4.04)	1.05 (0.17)	12,115 (95.15)	1.04 (0.14)
OCD Symptoms, Child**	17 (0.13)	**1.43 (0.45)**	97 (0.75)	**1.41 (0.44)**	627 (4.82)	**1.32 (0.37)**	12,114 (93.14)	**1.28 (0.36)**

$*p < 0.001$
$** p < 0.0001$

Adjusting for child food allergies and child weight status, reported restrictive feeding behavior significantly differed across ED group $F(5, 9805) = 6.98, p < .0001$. The coefficient of determination was $R\_ = .01$. Mothers with BN ($M = 2.82, SE = .10$) and BED ($M = 2.70, SE = 0.04$) both reported significantly higher levels of restrictive feeding than mothers with no ED ($M = 2.55, SE = .02$); mothers with AN did not significantly differ ($M = 2.28, SE = .25$) from those with no EDs. Adjusting for child food allergies and child weight status, pushy feeding behavior also differed significantly across groups, $F(5, 9,797) = 6.35, p < .0003$. The coefficient of determination was $R\_ = .004$. Mothers with BED ($M = 2.37, SE = .04$) reported higher levels of pushy feeding behavior than mothers with no ED ($M = 2.23, SE = .02$); mothers with AN ($M = 1.92, SE = .24$) and BN ($M = 2.43, SE = .10$) did not significantly differ from mothers with no EDs. Adjusting for child food allergies and child weight status, use of food as reward also differed significantly across groups, $F(5, 9,742) = 7.03, p < .0001$. The coefficient of determination was $R\_ = .002$. Mothers with BED ($M = 1.94, SE = .05$) reported higher levels of using food as a reward than mothers with no ED ($M = 1.74, SE = .02$); mothers with AN ($M = 1.39, SE = .27$) and BN ($M = 1.65, SE = .11$) did not significantly differ. Because of skewed residuals for the use of food as reward variable in the Tukey-Kramer test, a Kruskal-Wallis non-parametric test was used to assess the sensitivity of the first analysis. In contrast to Tukey's HSD, Kruskal-Wallis places no assumption that all samples under comparison are normally distributed. The results of this test corroborate that there is a statistically significant difference among the four ED subgroups, $\_\_(3) = 19.86, p < .0002$, on use of food as reward as a maternal feeding style. To assess the impact of maternal weight status on reported restrictive feeding, pushy feeding, as use of food as a reward, maternal BMI was added as covariate to each model. Whereas

maternal BMI had a significant effect as an independent predictor of the response ($p < .001$), it did not alter the significance or magnitude of the relation between ED status and feeding style in any of the analyses.

Specific Aim 2: Maternal Report of Child Eating Behavior and Psychiatric Symptomatology

Maternal reports of child eating behaviors and psychiatric symptoms were examined to assess whether mothers' ratings differed across those with AN prior to pregnancy, BN, BED, and no ED. Measurement of eating problems was taken from the 4-item *Eating Disturbance* subscale of the CBCL - Child Behavior Checklist (Achenbach, 1991), as were measures of anxiety symptoms. Measurement of depressive and obsessive compulsive traits were taken from Infant Toddler Social Emotional Assessment subscales (Carter et al., 1999).

The relation between maternal ED status and child eating and psychiatric symptom predictors were described using ANOVA. All pair-wise comparisons across the four groups were made using Tukey's HSD post hoc test at the significance level of 0.05.

Child eating behavior

Measured at 36 months, maternal-reported child eating behavior is defined by the 4-item *Eating Disturbance* subscale of the CBCL - Child Behavior Checklist (Achenbach, 1991). This includes questions regarding not eating well, not enjoying food, vomiting without medical cause, and eating non-food items. Adjusting for food allergy and child weight status, reported child eating behavior significantly differed across ED status, $F(5, 9{,}817) = 7.03$, $p < .0001$. The coefficient of determination was $R\_ = .04$. Mothers with BN ($M = 1.38$, $SE = .03$) and BED ($M = 1.34$, $SE = .01$) reported higher levels of disordered eating behaviors in their children than mothers with no ED ($M = 1.29$, $SE = .007$). Significant

differences were not found between mothers with AN ($M = 1.20$, $SE = .08$) and mothers with no EDs.

Psychiatric symptoms

Reported infant anxiety significantly differed across ED status, $F(3, 12,870) = 13.75$, $p < .0001$. The coefficient of determination was $R\_ = .003$. Mothers with BN ($M = 1.32$, $SE = .03$) and BED ($M = 1.31$, $SE = .01$) reported higher levels of anxiety symptoms in their children than mothers with no ED ($M = 1.24$, $SE = .003$). Significant differences were not found for mothers with AN ($M = 1.23$, $SE = .08$) in comparison to mothers without EDs.

While reported infant depression significantly differed overall across ED status, $F(3, 12,856) = 3.83$, $p < .009$, there were no differences across pairwise comparisons. The coefficient of determination was $R\_ = .0009$. Means were as follows: mothers with AN ($M =1.04$, $SE = .03$), BN ($M = 1.07$, $SE = .01$), BED ($M = 1.05$, $SE = .006$), and no ED ($M = 1.04$, $SE = .001$). Reported infant OCD symptoms differed among groups, $F(3, 12,851) = 6.78$, $p < .0001$. The coefficient of determination was $R\_ = .002$. Mothers with BN ($M =1.41$, $SE = 0.04$) reported higher levels of OCD symptoms in their children than mothers with no ED ($M = 1.28$, $SE = .003$). Significant differences were not found for mothers with AN ($M =1.43$, $SE = .09$) and BED ($M = 1.32$, $SE = .01$) relative to mothers without EDs.

Specific Aim 3: Relation between Maternal-Report of Feeding and Child Eating Behavior

To examine the relation between maternal reported feeding behaviors and child eating behavior, three models were created corresponding to restrictive feeding, pushy feeding, and use of food as reward. Again, assessment of child eating behaviors was taken from the 4-item *Eating Disturbance* subscale of the CBCL (Achenbach, 1991). Child weight-for-age and

child food allergy status were added to each model as covariates. After adjusting for child

food allergy and child weight-for-age status, maternally reported restrictive feeding was

significantly associated with child eating behavior in the positive direction, $F(3, 9,519) =$

$16.4, p < .0001$. The coefficient of determination was $R\_ = .03$. A significant positive relation

also emerged between pushy feeding and child eating status, $F(3, 9,513) = 654.5, p < .0001$.

The coefficient of determination was $R\_ = .09$. Further, reported use of food as reward was

significantly, positively associated with child eating behavior $F(3, 9,459) = 189.2, p < .0001$,

with a coefficient of determination of $R\_ = .05$.

V. Discussion

The impact of eating disorders on maternal feeding habits, child eating behaviors, and psychological symptoms is a salient clinical and public health issue, yet there have been no prior prospective population-based cohorts of mothers with EDs that have examined this issue. This study explored the impact of broadly defined maternal EDs on maternal feeding practices and children's eating behaviors and psychological symptoms in a sample of 13,006 36 month children from the The Norwegian Mother and Child Cohort Study (MoBa).

Prior to addressing the primary aims, it is important to note that the prevalance of the EDs in the sample is lower than general Scandinavian population studies (Keski-Rahkonen et al., 2007). Of respondents, .13% met criteria for AN, 0.75% for BN, and 5% for BED, only slightly over 5% of the entire sample. One possible explanation for this finding is that the current study may be limited in being able to measure current and lifetime symptom severity in individuals with EDs. Recent population data from a large cohort of Finnish twins suggest that AN may be underdetected due to sample bias associated with common health care system samples (Keski-Rahkonen et al., 2007). The study detected a higher prevalence of AN (2.2%) and a higher recovery rate (68% within 5 years) than prior estimates, suggesting the existence of a subset of women with AN who may have a milder course of symptomatology and never seek health care. Moreover, the current sample is composed of pregnant women and further, those who are willing to partake in an extensive battery of assessments and clinical tests. Women with more severe ED symptomatology may be less likely to get

pregnant, and participant burden may select out women with an ED for whom the paperwork and tests seem overwhelming in the face of psychopathology.

Moreover, the prevalence estimates of BN in Norwegian females range from is 0.7-1.6 % (Götestam & Agras, 1995), and estimates from American community samples of those with BED range from 3.5-6.6% (Hudson et al., 2007; Grucza et al., 2007). The current study's prevalence estimates for BN and BED are at the low end and in the middle, respectively, relative to prior population ranges. It must be considered that the current study may be limited in being able to measure lifetime or current symptom severity in individuals with EDs. In turn, those who may have milder binging or purging symptoms may not meet criteria for BN or BED, leading to lower prevalence estimates in the sample. Moreover, while confidentiality of the questionnaires was explained to participants, it must be considered that considerable stigma associated with ED may lead to underreporting, especially among pregnant women, and in turn, under-detection of EDs in the sample.

Regarding demographic characteristics of the sample, significant differences emerged regarding maternal age at childbirth, parity, educational status, and BMI. Maternal age at childbirth differed significantly across groups, with mothers with AN having a younger age at the childbirth documented in the study than those with BN, BED, and no ED. While not significant, mothers with AN in the sample were less likely to have had prior births than other groups. This may be an artifact of women with AN participating in the study at a younger age than mothers with BN, BED, and no ED, who appear to have started having children at an earlier age. The overall sample was well educated, though mothers with BED were more likely to be less educated than those with No ED. Given that participation in research studies is often associated with higher educational status, this finding is not

surprising. While those with BED were generally less educated that those with no ED, the differences appeared to be minimal and related to pursing more than 4 years of college. Expectedly, mothers with AN had significantly lower BMIs than all other groups. Women with BED had a significantly higher BMI than those with BN and no ED. These differences may be due to the effect of specific behaviors associated with each disorder. Women with BED are more like to binge without compensatory behaviors than other groups, leading to higher weight status than those with BN and No ED, who binge with compensatory behaviors, or don't binge at all, respectively (Grucza et al., 2007). However, women with both BN and BED met CDC criteria for overweight. Overall, it must also be considered that due to the small sample size of those with AN, the study was underpowered to detect all differences. This is discussed below further.

Regarding the first specific aim, significant differences emerged on all self-reported maternal feeding behaviors across ED status. Overall, self-reported maternal restrictive feeding was higher among mothers with EDs marked by binge eating (i.e., BED and BN) than those with no ED. Mothers with BED were also more likely to endorse pushy feeding than those without EDs. Finally, maternal use of food as reward was greater among mothers with BED than those with no ED. These finding suggest that disordered maternal feeding behaviors are notably different among mothers with EDs, specifically those with binging-type EDs, as compared to mothers without EDs.

While the current study cannot speak to causation, this tendency towards aberrant feeding behavior may pose a noteworthy health risk for the children of these mothers, one that may contribute to the long-term risk of the development of EDs in their offspring. If mothers tend to push their children past their normative eating boundaries via dysregulated

feeding, the children may be hampered in learning their own self-regulation. This is supported by prior findings suggesting the responsiveness to internal cues is disrupted by imposed child-feeding, redirecting the child's attention from internal hunger and satiety signals to the eating environment (Birch & Fisher, 2000; Birch et al., 2001; Francis, Hofer, & Birch, 2001; Johnson & Birch, 1994). Specifically, data have shown that pushing children to eat "healthy" foods may lead to the child disliking those foods, and restricting a child's intake of "junk" foods tend to increase the intake of the foods when parents are not overseeing child eating (Birch & Deysher, 1986; Birch & Fisher, 2000; Birch et al., 2001). Moreover, reduced awareness of hunger and satiety cures has been correlated with increased child weight status (Birch et al., 2001; Johnson & Birch, 1994). Collectively, these findings have importance from the standpoint of future public health studies, and the treatment and prevention of EDs, as well as childhood obesity.

Restrictive Feeding

The first hypothesis, that mothers with AN before pregnancy will endorse feeding attitudes and practices that restrict their children's eating significantly more than mothers with BN or BED, or no EDs, was not upheld. In fact, restrictive feeding was more strongly associated with BN and BED subtypes than with AN relative to the no ED group. It is important to consider the size of the effect regarding the correlation of determination ($R_=$ 0.01), suggesting that 1% of the total variation in scores on maternally reported restrictive feeding can be explained by the linear relationship between restrictive feeding and ED subtype. This is considered a "small" effect using the conventions of Cohen (1988). This may be related to the study's sample size and potential signal-to-noise ratio.

Overall, examining effect size is important in determining whether findings are of statistical versus practical/clinical significance. Cohen intended for his categorization of effect size to be provisional: the context in which the effects exist, the methods used, and area of study must be taken into consideration (Morgan, Leech, Gloeckner, & Barrett, 2004). However, because the effect sizes in this study were relatively small, interpretation is limited in terms of clinical importance. Nonetheless, the study's findings have notable theoretical importance. The finding that restrictive feeding is more prevalent among mothers with BN and BED than other groups provides insight into specific differences in how mothers with EDs feed their children. This is a step in the process of more clearly defining environmental factors that may both emerge from disordered maternal eating and contribute to disordered eating in offspring. Ultimately this could lead to the identification of rational environmental variables to be included in models of gene x environment interaction.

The result is also intriguing in relation to the theory that mothers with binge-type EDs may possess the core belief that food has non-nutritive meaning such as comforting or calming. While this model states that core beliefs may lead to positive thoughts about feeding the baby (e.g., to show her love or comfort), in fact, the opposite may occur. While some positive emotions may be related to eating, mothers with binge-type EDs may have a globally negative association with food. Though food may provide momentary relief from negative emotions during a binge, the long-term effects---distress, guilt, anxiety, shame, and stigma, may have greater impact on these women's cognitions relating to feeding their children.

Women with binge-type EDs may demonstrate restrictive feeding to protect their children from the negative consequences of feeding them too much. A distorted perception of

healthy portion control among women with binge-type EDs may influence restrictive

feeding. Lack of control over eating may influence her confidence in regulating her child's

eating appropriately. In turn, restricting her child's food intake may be perceived as the only

way to prevent over-feeding the child. Further, mothers with EDs tend to have a deficit in

interpreting their own hunger and satiety cues (Dalle Grave, Di Pauli, Sartirana, Calugi, &

Shafran, 2007). In turn, her ability to perceive the cues expressed by her child's behavior

(e.g., excitement about eating, wanting more or less food) may be unobserved or

misinterpreted, contributing to the outcome of restrictive feeding.

Given known differences in maternal feeding styles associated with maternal

overweight and obesity (Francis et al, 2001; Johannsen et al., 2006), this study also explored

the extent to which our observed associations were explained by maternal weight status, not

the presence of an eating disorder. On average, women with BN and BED fell into the

overweight category, whereas women with AN and no ED were on average normal weight.

However, after assessing the impact of maternal weight status on reported restrictive feeding,

pushy feeding, as use of food as a reward, whereas maternal BMI had a significant effect as

an independent predictor of the child eating behavior, it did not impact the significance of the

relation between ED status and feeding style in any of the analyses. Thus eating disorders

appear to influence maternal feeding style independent of maternal BMI.

Conversely, mothers with AN may not engage in restrictive feeding to protect their

children from the negative consequences of their own restrictive ED behavior, such as stigma

or health risk. Another explanation is that many studies have found that women with AN tend

to underreport their symptoms (Couturier & Lock, 2006; Vandereycken & Van Humbeeck,

2008). This may be due to the denial of their symptoms due to distorted perceptions about the

severity of the disorder (Couturier & Lock, 2006). Underreporting may also be a product of being untruthful about AN symptoms–usually not an act of intentional deception, but rather a means of preserving their primary coping skills, or avoiding the notable stigma associated with the disorder. While participants were aware of the confidentiality of the questionnaires, it is possible that those with AN who are untruthful about their symptoms are so used to the secrecy of these behaviors, that they would not acknowledge their presence in any context.

Further, the current study may be limited in being able to measure current and lifetime symptom severity in individuals with EDs. As mentioned previously, AN may be underdetected due to sample bias associated with common health care system samples (Keski-Rahkonen et al., 2007). A group of women with AN may have milder symptoms and never seek health care. If those reporting AN in the current study were those with milder symptomatology or near recovery, their responses to outcome measures would likely differ from those with more severe AN. Again, sample selection limited to pregnant women and those who are willing to partake in an extensive assessment process may further contribute to a lower estimate of ED symptomatology, as well as sample bias.

Pushy Feeding

The second hypothesis, that mothers with BN and BED would endorse feeding attitudes and practices that pressure their children to eat significantly more than mothers with AN before pregnancy or no EDs was partially confirmed. Pushy feeding was more strongly associated with the BED subtype than with AN and BN relative to the no ED group. This finding must be considered in context of the "small" effect size in regards to the correlation of determination ($R_{\_} = 0.004$), suggesting that 0.4% of the total variation in scores on pushy feeding can be explained by the linear relationship between pushy feeding and ED subtype.

Again, this finding may be considered theoretically relevant: contributing to the overall picture of how mothers, specifically those with binge-type EDs, endorse disordered feeding practices. This is salient to future research in providing a starting point in investigating how mothers with EDs differentially feed their young children. As with restrictive feeding behavior, pushy feeding may be exacerbated by the mother's inability to perceive her child's expression of hunger/satiety cues. Further, while this finding seems to stand in contrast to the result that mothers with BED also tend to restrict their child's intake more than mothers with no ED, it is possible that binge eating behavior is related to any type of disordered child feeding. By definition, BED is associated with "out-of-control" eating, novelty seeking, harm avoidance (Fassino et al., 2002) and emotional dysregulation related to binge eating (Whiteside et al., 2007). Collectively, these characteristics may make feeding a child in a pushy manner a likely reaction to an emotionally loaded situation for a person with BED. In this group, the feeding style may be influenced by cognitions that pushing food on the child, even it she does not want it, could have an calming, rewarding, or even punitive effect on the child (Agras et al., 1999; Lacey & Smith, 1987).

Though this logic suggests that mothers with BN should endorse higher pushy feeding than those without a binging history, the presence of purging behavior in this population may lead to a differential feeding response to those who binge without compensatory behavior. Why significance emerged only in mother's with BED and not BN may be related to the presence of purging behavior in BN and its impact on the mother's core beliefs. An individual who purges is usually making an effort to conpensate for the potential weight-gain associated with binge eating. Pehaps the elevated focus on shape and weight in women with BN is protective of pushy-feeding. The mother with BN may be more invested

in protecting her child from suffering the consequences of weight-gain than mothers with

BED, leading women with BN not to differ significantly in pushy feeding those with BED,

AN, or no ED.

Use of Food as Reward

The third hypothesis, that mothers with BN and BED will tend to use food as a

reward for their children significantly more than mothers without EDs or mothers with AN

before pregnancy, was partially upheld. Use of food as reward was more strongly associated

with the BED subtype than with AN and BN relative to the no ED group. However, 0.20 %

of the total variation in scores on maternally reported use of food as reward can be explained

by the linear relationship between use of food as reward and ED subtype. Again, while the

effect of the finding was "small", it is important theoretically, lending further evidence that

women with binge-type EDs may endorse disordered feeding behaviors more than those in

other ED subgroups. Collectively, with the finding on restrictive and pushy feeding, it is

strongly suggested that mothers with binge-type EDs exhibit a number of problematic

feeding styles, all of which point toward a general pattern of controlling the feeding of their

young children (e.g., pushy, using food as a reward, and restricting feeding). This could

suggest a feeding pattern that indicates a failure to respond to or inability to recognize child-

generated cues, which could theoretically impair a child's ability to develop appropriate

feeding self-regulation.

Again, in individuals with BED, food may take on meaning beyond being a source of

nutrition. These mothers may define feeding at different times as comforting, calming, or

even adversarial, and in turn feed her child to communicate these emotions (Agras et al.,

1999; Lacey & Smith, 1987). Another possibility is that mothers with BED are using food as

reward in ways that are consistent with the literature on overweight and obese mothers. Indeed, 26.66% of those with BED ≥ 25.00, and 25.56% of those with BED had a BMI ≥ 30.00. The sensitivity of dopamine reward pathways has been implicated in the risk for compulsive overeating. Examining psychological and biological markers of reward sensitivity, one study found that individuals with BED and obese reported greater reward sensitivity than normal-weight controls, specifically those carrying the A1 allele (Davis et al., 2008). Using fMRI technology, Batterham et al. (2007) recently linked specific peptides (YY3-36) to the activation of nonhomeostatic brain regions, providing insight into the neural response to specific satiety signals that regulate food intake, potentially different in those with obesity and those of normal weight status. Together with the current obesogenic environment of modern culture, this evidence suggests that individuals who are overweight, obese, or display compulsive overeating may biologically differ from normal weight controls regarding reward pathways regulating food intake (Batterham et al., 2007). In turn, mothers with BED may feed their baby's based with a heightened perception of food as reward based on a unique biological mechanism.

Individuals with AN tend to score lower on measures of novelty seeking than women with BED and BN (Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006). This absence of this personality factor may protect AN mothers from providing her child with a novel stimulus, such as food, to produce a positive response. Though individuals with BN tend to also have novelty seeking personality traits, the personality trait of drive may mediate their use of food as reward. One study found that women with BN score significantly higher on measures of drive for thinness than obese women with BED (Barry, Grilo, & Masheb, 2003);

many other studies have found that women with AN also have high levels of drive for thinness (Lilenfeld et al., 2006).

Women with AN and BN may not see food as a rewarding substance due to its association with weight gain. This may be supported by data from a recent fMRI study showing that recovered women with AN showed greater hemodynamic activation in the caudate than comparison women than controls (Wagner et al., 2007). The authors hypothesize that this may relate to an impaired ability to identify the emotional significance of a stimulus and altered reward processing in women with AN. In turn, mothers with AN may not perceive food to be rewarding, and subsequently, not use food as a positive stimulus in relation to feeding their children.

Women with EDs tend to have difficulty with emotion regulation (Lilenfeld et al., 2006; Whiteside et al, 2007). Attempts to address unwanted emotions often manifests in the use of ED behaviors, which in the short term, can provide a false sense of control or short-lived relief. Such difficulty in controling emotions, and use of disordered behaviors to do so may translate in their approach to regulating their children's emotional expression via feeding practices. For example, if a child is crying, the BN or BED mother may use pushy feeding as a means to quell the negative emotion. This model holds true for restrictive and pushy feeding, and use of food as a reward. Women with BN may tend to favor restrictive feeding due to aforementioned core beliefs about shape and weight, whereas women with BED may choose a behavior based on her beliefs that food is rewarding and associated with and positive emotions. As with restrictive feeding behavior, pushy feeding and use of food as reward may be exacerbated by the mothers inability to perceive her child's expression of hunger/satiety cues.

Child Disordered Eating

The fourth hypothesis, that 36 month old children of mothers with AN before pregnancy will exhibit problematic eating behaviors significantly more than children of mothers without EDs and children of mothers with BN and BED, was not supported. Specifically, the measure of infant eating problems included items relating to not eating well, having stomach aches or cramps, vomiting without medical cause, and not enjoying eating.

Regarding the significant findings, child eating problems were more strongly associated with the BN and BED subtype than with AN relative to the no ED group. Again, this finding must be balanced with an interpretation of the effect size of the correlation, again considered "small" using Cohen's convention: 4% of the total variation in scores on maternally reported infant eating problems can be explained by the linear relationship between eating behavior and ED subtype. This finding is theoretically important in beginning to elucidate differences in eating behaviors in children of mothers across ED subtypes–differences which may increase as the child matures.

One possible explanation for this finding may be due to vigilance on the part of mothers with binge-type disorders to detect early emergence of eating problems in their children. These mothers have likely experienced negative outcomes associated with their own eating behavior, and have specific interest and a heedful approach in identifying disordered-type eating in their offspring. As women with BN and BED are highly susceptible to these factors (Whiteside et al., 2007), their ability to detect the symptoms in their child may lead to attentive reporting of their child's eating problems. It must also be considered that sharp awareness of these symptoms could lead to over-reporting. Another explanation may be related to the prior finding: that those who report higher levels of dysregulated

feeding also report higher levels of infant eating problems. While it is beyond the scope of

this study to comment on the directional nature of this association, the relation between

dysregulated child eating and maternal feeding may create a uniquely problematic cycle for

mothers with a history of BN and BED.

Child Anxiety and OCD

The current study's 5th hypothesis stated the following: 36 month old children of

mothers with AN before pregnancy will exhibit obsessive compulsive traits significantly

more than children of mothers with no ED and children of mothers with BN and BED; 36

month old children of mothers with AN, BN and BED will exhibit depression and anxiety

symptoms significantly more than children of non-ED mothers. In part, this hypothesis was

upheld.

Significant differences emerged across ED status on maternal reported infant anxiety

and OCD symptoms. Higher child anxiety symptoms were reported in children of mothers

with BN and BED more than in children of mothers without EDs. Mothers with BN reported

higher OCD symptoms in their children than mothers without EDs. The effect sizes for both

anxiety and OCD analyses were considered small by the convention of Cohen. Respectively,

0.2% and 0.3% of the total variation in scores on maternally reported child anxiety and OCD

symptoms can be explained by the linear relationship between each variable and ED subtype,

suggesting that while findings were significantly different, they may not be clinically

important. However, it must be considered that the children in the sample are 36 months old,

early in the trajectory of potentially developing more pronounced psychopathology. Given

that the current data represent an early observation point, they are noteworthy in providing

theoretical information for future studies investigating the potential emergence of

symptomatology in later childhood and beyond.

This finding is surprising given that prior studies have shown that anxiety disorders are

common in clinical and community samples of women with AN as well as BN (Braun et al.,

1994; Brewerton et al., 1995; Bulik et al., 1996; Bulik et al., 1997; Deep et al., 1995;

Garfinkel et al., 1995; Schwalberg et al., 1992). However, research is scant regarding anxiety

symptoms in the offspring of infants of mothers with a history BED. This finding may be

explained by familial aggregation of anxiety based of both genetic and environmental factors

(Silberg & Bulik, 2005). Mothers with BN reported higher OCD-type symptoms in their

children than mothers without EDs. Given a significantly elevated risk OCD in family

members of women with BN relative to controls (Lilenfeld et al., 1998), this finding may

further support familial aggregation of OCD behavior in those with a history of BN.

While scores on maternal-reported child OCD symptoms were slightly higher

numerically in the children of women with AN (M =1.43, SE = .09), than those with BN (M

= 1.41, SE = .04), BED (M = 1.32, SE = .01), or No ED (M = 1.28, SE = .003), findings were

non-significant. The OCD scale of the ITSEA (Carter et al., 2003) measures the child's

worry about getting dirty, need to be clean or neat, and repeatedly put things in order. Due

both to the probability of familial transmission of OCD traits from AN mother to child and

the mother's modeling OCD behaviors in the home environment, it would be expected that

differences between those with AN and other groups would emerge. Due to the high

comorbidity of anxiety disorders and AN (Godart et al., 2005), these symptom descriptions

may be especially difficult to identify for mothers with AN, who may normalize or deny

anxious behavior, leading to underreporting. Moreover, the small sample size of the AN group may explain the current study being underpowered in regards to this group.

Child Depression

Unexpectedly, while an overall significant effect was found, no significant differences occurred in pairwise comparisons of maternal-reported child depressive symptoms across ED status. Of the total variation in scores on maternally reported child depressive symptoms, 0.09% can be explained by the linear relationship between depressive symptoms and ED subtype. Regarding the overall effect, effect size may be considered very "small" using Cohen's conventions. Whereas there is significant evidence for familial aggregation of depression in EDs (Hudson et al., 1983b; Silberg & Bulik, 2005), the lack of differences across groups may relate to the overall difficulty in capturing depression, an internalized (less visible) characteristic, in young children. The ITSEA (Carter et al., 2003) captures depression with items including the child having less fun than other children, lacking energy, and showing unhappiness, sadness, or depression, a generally internalized symptom. Whereas there is evidence of stability within the domain of externalizing problems in infants, less is known about the stability of early internalizing and regulatory problem behaviors (Carter et al., 2003), perhaps leading to a decreased likelihood in detecting depression overall. Moreover, the developmental psychopathology literature provides little systematic research on assessing depression in preschool-age children and infants (Klein, Dougherty, & Olino, 2005). While some data suggest that depression can be identified in preschool age-children using modified DSM-IV criteria with a shorter duration requirement (Luby et al., 2004), overall, it remains unclear whether symptoms are comparable to those identified in older children, adolescents, and adults (Garber & Horowitz, 2002). It is possible

that the sensitivity of the current study's assessment was unable to capture symptoms of depression among 36 month old children.

Further, the emergence of significant differences among ED groups on child anxiety symptoms but not depressive symptoms may be explained by developmental data suggesting anxiety can be detected earlier in children. While a number of issues regarding measurement must be considered (e.g., maternal, teacher, caregiver, child report, child observation), findings suggest that depressive symptoms can onset as early as age 3 (Luby et al., 2004). However, recent studies show that symptoms of anxiety may be detected even earlier. Using maternal and caregiver report and child observations, a pilot study assessing generalized anxiety disorder and social phobia symptoms found significant differences among two year olds (Warren, Umylny, Aron, & Simmens, 2006). Further, in a sample of 1,235 parents, validity data for the parental report Infant Toddler Social and Emotion Assessment suggest that symptoms of anxiety can be identified as early as 12-36 months (Carter et al., 2003). This has been corroborated by other community and clinical studies in young school age children, finding that specific anxiety disorders precede the onset of depressive symptoms (Kovacs & Devlin, 1998; Orvaschel, Lewinsohn, & Seeley, 1995). Collectively, these data may suggest that symptoms of anxiety are observable at an earlier age than those of depression, potentially explaining a difficulty in the detection of child depression relative to anxiety by mothers in the current study.

Maternal Reported Feeding and Child Eating Behaviors

All maternal reported feeding behaviors–restrictive, pushy, and use of food as reward, were significantly associated with child eating behaviors. The effect size for each analysis was .03, .09, and .05, respectively, "small" using the conventions of Cohen. While the

analyses were not specific to women with EDs, it is notable that a relation exists between the way a mother feeds her child and the way the child eats. However, it must be considered that mothers who perceive and report their own problematic feeding styles are more likely to notice and report disordered eating behavior in their children.

Combined with the prior findings that disordered feeding is more prevalent in ED mothers than non-ED mothers, the relation between maternal feeding and child eating is important to consider further. While the current analyses do not determine directionality, future studies should investigate how restrictive, pushy, and food-as-reward feeding styles may contribute to the risk of developing problematic eating patterns in children of mothers with EDs. Further, the impact of child eating behavior on maternal feeding style in women with EDs warrants examination as evocative gene-environment correlation suggests that children can evoke certain behaviors in their caregivers who provide their environment.

VI. Limitations

The study's strengths must be evaluated in concert with its limitations. First, the majority of data were collected from self-report measures; some were not (e.g., birth weight, Apgar scores). While the impact of demand characteristics on accurate report must be considered, there is some evidence suggesting the contrary. Survey research has found that in contrast to interviews, self-report formats provide anonymity that may facilitate superior disclosure of highly sensitive material (Catania et al., 1992; Rolnick, Gross, Garrard, & Gibson, 1989; Turner et al., 1998). This may be particularly salient in the current study, in which we collected data regarding highly stigmatized topics, such as behaviors associated with ED status. However, it must also be taken into consideration that none of the survey research was conducted in a sample of mothers, pregnant women, or women with EDs. Further, given denial or normalization of ED symptoms in women with AN, the potential for underreporting must be considered. To additionally assure participants of their privacy, the MoBa study sent all questionnaires to participants by mail. Mothers were informed that involvement in the study was independent of their prenatal care and not shared with their medical practitioners. Another limitation was the study's inability to collect information of children from multiple sources. Whereas parents are often accurate reporters of their young children's externalized behaviors on validated measures (Achenbach, 1991; Carter et al., 2003), it would be optimal to compare parent report with observations of other relevant individuals in the child's life, such as caregivers, grandparents, and teachers. However, given the size of the sample, the attendant costs, and sites dispersed across Norway, this was not

possible. Further, it must be considered that the effect sizes of all significant findings were relatively small according to the conventions of Cohen (Cohen, 1988). In turn, interpretation of the current study's findings should be limited to theoretical significance. Because there are few data from large scale epidemiological studies presenting insight into feeding behaviors of mothers with EDs and eating behaviors and psychological symptoms of their children, the study's findings may be particularly informative for future studies.

Directional bias must also be considered. In the case of ED thoughts and behaviors, given the impact of stigma, it is more likely that individual would conceal these symptoms than positively report non-existent behaviors. The impact of this conservative bias would lead to difficulty in detecting a significant difference. Another limitation of the study is the homogenous nature of the Norwegian population, and subsequently, participants. As the study sample is limited to primarily Nordic ancestry, ability to generalize findings to individuals of other races and ethnicities is limited. Further, because not all individuals in the population participated in the study, potential selection bias should be noted. However, as health care is offered without cost to the majority of women in Norway, providing access to prenatal care, there is a greater likelihood of reaching most pregnant women in recruitment efforts. Further, selection bias is less of a concern in the current study, with case: control aims, than if inferences about the population were made. Additionally, although the current sample had a somewhat low participation rate (42%), this does not necessarily assume bias unless those who chose not to participate differed significantly from those who did participate on the variables of interest. Prior published education data suggest that MoBa participants may be slightly more educated than the general Norwegian population. Of MoBa participants, 58% attended some form of college, whereas 46% of non-participant women

aged 25-29 years and 43% of women aged 30-39 years reported attending college (Bulik, Von Holle et al., 2007). Moreover, a possible socioeconomic difference between participants and non-participants is suggested by lower rates of preterm birth (7.2% vs 7.7%) and low birth in participants (4.6% vs 5.1%) (Magnus, 2006).

Finally, it must be noted that no significant differences were found relative to mothers with AN. This may be accounted for by the limitation of the small sample size of the AN group. Because confidence to detect a signal is dependent upon sample size, signal to noise ratio, and/or effect size, the AN group's small N may have impacted the probability of being able to reject the null hypothesis when it was true or false (e.g., Type I or III error). Alternately, participants with AN in the current study could have had relatively mild cases of AN with less physical sequelae, leading to more normative responses on outcome measures. The existence of a subset of women with AN who may have a milder course of symptomatology and never seek health care may be supported by recent population data from large cohort of Finnish twins, suggesting that AN may be underdetected due to sample bias associated with common health care system samples (Keski-Rahkonen et al., 2007). The current study was limited in its ability to comprehensively assess current and lifetime symptom severity in individuals with EDs.

In contrast to the noted limitations, the MoBa dataset provides a unique opportunity to conduct large-scale, long-term prospective epidemiological research. The social environment in Norway provides a setting in which such data collection is possible, and the well educated population has a history of voluntary participation in medical research. Naturally, bias must be considered regarding education rate of participants and non-participants.

VII. Significance and Future Directions

The current study confirms that reported maternal feeding behaviors and child eating patterns significantly differ across ED status. In particular, mothers with binge-type EDs may be more likely than other subgroups to use disordered behaviors in feeding their young children. Further, these mothers had the tendency to endorse most or all types of disordered feeding–restrictive, pushy, and use food as reward, indicating a global dimension of controlling their child's eating. Overall, this may suggest that mothers with binge-type disorders have a relationship with food infused with emotional overtones, potentially leading to disordered thoughts that the food will provide comfort, appeasement, or distraction for her child. Children of mothers with binge-type EDs were also more likely to display disordered eating behaviors. It is possible that dysregulated feeding displayed by mothers with BN and BED may impact the child's eating behavior, leading to problematic behaviors such as stomach cramps, vomiting, and pickiness as assessed by the CBCL. Moreover, mothers who display emotional reactions around food and disordered eating themselves may model an approach to food that is adapted by their children.

However, it must be considered that the current findings regarding maternal feeding and child eating behaviors are through the lens of the mother's report. As discussed previously, mothers with binge-eating type disorders may differ from AN mothers in their ability to perceive disordered feeding and eating behaviors.

Maternal feeding styles may be a direct environmental influence on the "cycle of risk" for EDs (See Figure 4). In part, this may be supported by the finding that maternal

feeding style and child eating behavior are significantly associated. This risk is best understood though the model of gene-environment correlation. EDs are strongly familial, primarily due to genetic factors (Bulik et al., 2000; Klump et al., 2001; Kortegaard et al., 2001; Wade et al., 2000). Yet both shared and unique environmental factors have been suggested to play a significant part role in the expression of genetic underpinnings (Klump et al., 2007; Klump et al., 2001; Kortegaard et al., 2001; Wade et al., 2000).

While a mother passes her genes down to her child, she also plays a large role in rearing. Early environment is considerably influenced by the feeding relationship between mother and child. Mothers with ED are known to be less likely to eat with or in front of their children (Franzen & Gerlinghoff, 1997; Russell et al., 1998), possibly contributing to an absence of a healthy eating model. The child might then be doubly disadvantaged. On one hand, she or he may have an elevated genetic risk for developing EDs. Simultaneously, the strained eating environment may act as an environmental risk factor for disordered eating, but one that is partially mediated by the maternal genotype.

Other environmental factors also play a role in the cycle of risk. For example, the role of the biological father, partner, step-parent or caregiver, the school environment, and multiple cultural and community factors can have significant impact on a child (Amato & Gilbreth, 1999; Dunn, Davies, O'Connor, & Sturgess, 2000). Indeed, other healthier feeding environments may exist that counterbalance the impact of pushy or restrictive feeding, for example. Children may be differentially robust to the impact of these maternal feeding styles. Whereas some may be constitutionally quite robust and maintain their ability to self-regulate, others may be more vulnerable to external influences and consequently develop an inability to read their own internal hunger and satiety cues, thereby increasing their risk for an ED.

Nonetheless, there is a large body of research supporting the particular importance of the maternal role in child development and eating behaviors.(Agras et al., 1999; Essex et al., 2006; Johannsen et al., 2006; Pickles et al., 1994). A recent prospective, multi-informant study of 379 families found that children were at risk of later mental health problems if their mothers were distressed during the infancy period; this was further associated with more generalized maternal and child distress and dysregulation during the preschool period (Essex et al., 2006) . Combined with various other environmental factors, disordered maternal feeding behavior may pose a significant liability in triggering ED symptoms and perpetuating the cycle of risk in genetically susceptible individuals.
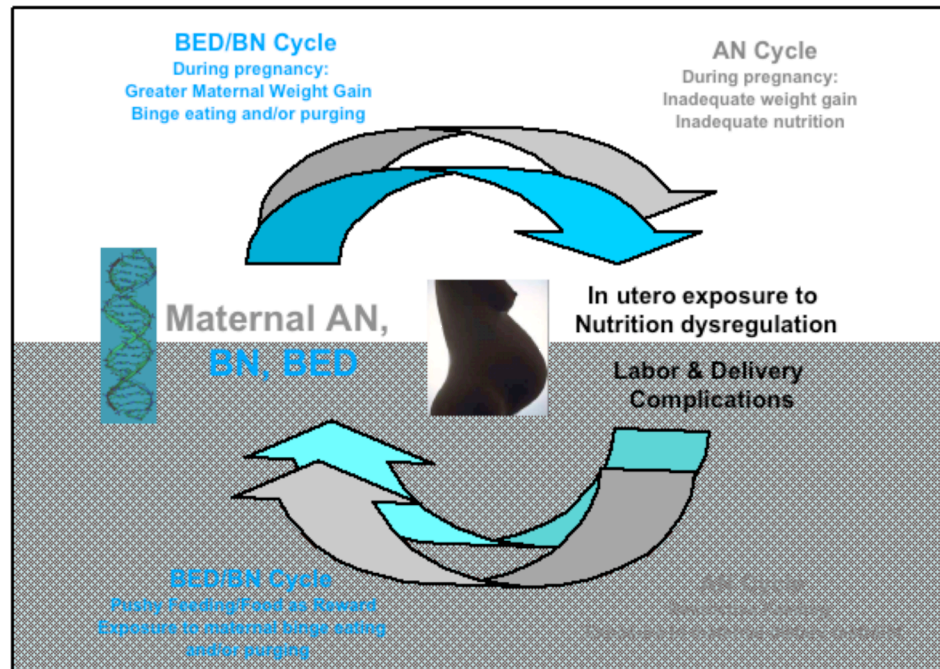
Individual characteristics of the child, of the mother, and of their relationship during the development of infant and early childhood feeding patterns are extremely important in informing future research, and ultimately, the assessment of early feeding disorders, the development of tailored diagnostic measurements, and targeted and effective intervention. The current study's results may provide important direction in elucidating potential phenotypes for genetic studies. Given that they reflect a maternal perception of feeding behavior and child eating behavior, they may also provide insight into continued investigation of how mothers with EDs may differentially view the act of feeding their children. Moreover, the findings may guide variable selection for studies which may yield similar differences but of a greater magnitude–perhaps in samples with mothers with greater clinical severity or with children at later developmental stages.

A prospective, population-based sample, the MoBa data set provides a unique opportunity to explore the entire cycle of risk. As data collection continues, the infants that are the focus of this study will be followed through the age of risk for EDs and beyond. Thus,

the true prospective environmental risk of maternal feeding patterns on eating styles,

nutrition, and the development of EDs may be explored in these children as they move into

adolescence and adulthood. Ideally, future studies could investigate this cycle of risk across

multiple generations. The longitudinal design of the MoBa will enable the study of how

maternal feeding styles and child eating behaviors may change over time. In the long run,

this may support the development of targeted interventions aimed at mothers with EDs and

their children to prevent the cycle of risk for ED transmission or interrupt a cycle in progress.

Finally, the existence of the BioBank including DNA from the infants and their parents will

facilitate future molecular genetic work. This exciting aspect of MoBa will allow for future

exploration of the contributions of genetics to the development of AN, BN, and BED.

Ultimately, expounding on the current investigation of maternal feeding and child eating

behaviors will allow for more specific investigations of gene x environment interaction and

correlation in the etiology of EDs.

Figure 5:

Cycle of Risk of ED Transmission from Mother to Child (Bulik, 2008)

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