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DIFFERENTIAL PARENTAL TREATMENT IN INDIVIDUALS WITH ANOREXIA NERVOSA

By

Janet Wendy Solomon

A THESIS

Submitted to
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ABSTRACT

DIFFERENTIAL PARENTAL TREATMENT IN INDIVIDUALS WITH ANOREXIA NERVOSA

By

Janet Wendy Solomon

Objective: Differential parental treatment is one potentially important nonshared environmental factor that has received little attention in relation to anorexia nervosa (AN). This study expands upon previous research of differential parental treatment by including both twin and parent reports, using MZ twins discordant for AN to separate genetic and environmental effects, and by using a control group of twins with no eating pathology. **Method:** Participants included 27 AN twin pairs and 270 control twin pairs with no eating pathology who took part in the Minnesota Twin Family Study. Parental relationships were measured by the Parental Environment Questionnaire and general disordered eating was assessed with the Minnesota Eating Disorders Inventory. Analyses compared differences between and within disordered eating families and non-disordered eating families. **Results:** Within the AN sample, no differences in parental treatment were found either between or within families. Relationships between PEQ scores and continuous measures of eating (M-EDI) showed differences between-families in the areas of conflict, involvement, and parental regard, and within-families in the areas of involvement and maternal regard. **Discussion:** This study did not find evidence for differential parental treatment in AN, although there was evidence of differential parental treatment with continuous measures of disordered eating. These results suggest that differential parental treatment may be a small but significant nonshared environmental factor that is associated with the development of eating pathology.

TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

Anorexia nervosa (AN) is a complex psychiatric syndrome with an etiology that is unknown. The criteria for diagnosing AN, as delineated by the DSM-IV (APA, 1994), include an intense fear of gaining weight, a refusal to maintain a minimum body weight (i.e.. 85% of expected weight for height and age), a disturbance of body image (i.e., the individual has a distorted perception of her own body), and the absence of at least three consecutive menstrual cycles. Family factors are one set of etiologic influences that have been hypothesized to contribute to the development of AN (Bruch, 1973; Foulkes, 1996; Garfinkel, Garner, & Rose, 1983; Goldstein, 1981; Horesh et al., 1996; Palazzoli, 1974). Recent behavioral genetic research has begun to look at these family influences in-depth, including both genetic and environmental effects. In general, these studies have suggested significant genetic and nonshared environmental influences on eating pathology (Bulik, Sullivan, & Kendler, 1998; Bulik, Sullivan, Wade, & Kendler, 2000; Kendler et al., 1991; Klump, McGue, & Iacono, 2000; Klump, Miller, Keel, McGue, & Iacono, 2001; Kortegaard, 2001; Wade, Bulik, Neale, & Kendler, 2000). Shared environmental influences, which have traditionally been the most commonly studied, have typically not contributed to the variance in AN (Klump et al., 2001; Klump, Wonderlich, Lehoux, Lilenfeld, & Bulik, 2002; Kortegaard, 2001; Wade et al., 2000). Nonshared environmental influences are those that are unique to siblings reared in the same family, such as different experiences within a family based on birth order or gender. By contrast, shared environmental influences are those that are common to siblings reared in the same family, such as socioeconomic status and general child rearing attitudes.

These novel findings suggest a need to investigate specific nonshared environmental influences on the development of AN. Aside from twin research investigating their general effects (see below), very little research has specifically examined those nonshared environmental influences contributing most to the development of AN.

Evidence for General Effects: Twin Studies

Twin studies make it possible to separate genetic from environmental influences, and shared from nonshared environmental influences. Monozygotic (MZ) twins share 100% of their genes and dizygotic (DZ) twins share 50% of their genes. If genetic influences contribute to a trait or disorder, MZ twins will be approximately twice as similar for the trait/disorder than DZ twins, since they share roughly double the genetic material. Shared environmental influence is inferred when MZ and DZ correlations are equal, as these factors are common to co-twins growing up in the same family. Nonshared environment makes co-twins different, and thus nonshared environmental influence is inferred when MZ correlations are less than 1, or when neither MZ nor DZ twins are significantly correlated on behavioral measures of interest (Plomin, DeFries, & McClearn, 1990).

As briefly noted above, results from twin studies highlight the importance of genetic and nonshared environmental factors for the etiology of eating pathology (Bulik et al., 1998; Bulik et al., 2000; Kendler et al., 1991; Klump et al., 2000; Klump et al., 2001; Kortegaard, 2001; Wade et al., 2000). In general, findings have suggested that 48-74% of the variance in AN can be accounted for by genetic factors, whereas the remaining variance (24-52%) is accounted for by nonshared rather than shared

environmental influences (Klump et al., 2001; Kortegaard, 2001; Wade et al., 2000). Nonshared environment is thus an important factor in the development of AN, although it has not been widely studied. There is some evidence of shared environmental effects for specific disordered eating characteristics in adult women (e.g., weight concern; Wade, Martin, & Tiggeman, 1998), and for eating attitudes in younger children (Klump et al., 2000). However, nonshared environment is generally found to account for more of the variance in AN and related behaviors than shared environment (Bulik et al., 1998; Bulik et al., 2000; Kendler et al., 1991; Klump et al., 2000; Klump et al., 2001; Kortegaard, 2001; Wade et al., 2000). These findings highlight the need for studies examining the specific nonshared environmental factors contributing to this variance. Indeed, examining these nonshared influences will likely lead to the identification of specific risk factors for AN that can explain why two siblings growing up in the same family might differ with regard to eating pathology.

Types of Nonshared Environmental Influences: Differential Parental Treatment

There are a number of nonshared environmental factors that have been hypothesized to influence the etiology of psychological traits and disorders (Plomin, Chipuer, & Neiderhiser, 1994). These include: differential sibling relationships, whereby each child presents a different environment for their sibling by experiencing each other in different ways (e.g., one sibling is very controlling of the other sibling, who responds with submission); life events, which include differential life experiences that are not shared by siblings (e.g., one sibling develops cancer and the other does not); peer relationships, in which siblings have different peer groups or different experiences within the same peer group (e.g., one sibling is teased incessantly while the other is not); and

prenatal factors, which include different experiences within the womb (e.g., intrauterine growth differences). A final important nonshared environmental factor is differential parental treatment, whereby siblings are treated differently by their parents. Examples of differential parental treatment include parents favoring one sibling over the other or parents treating one sibling more harshly than the other.

Differential parental treatment is an important nonshared environmental factor to study for several reasons. First, traditional theories of AN often identify the parent-child relationship as an important etiological factor in the development of AN (Bruch, 1973; Garfinkel et al., 1983; Goldstein, 1981; Minuchin, 1974; Palazzoli, 1974). For example, Minuchin (1974) identifies enmeshment (i.e., familial overinvolvement) as characteristic of AN families.

Second, findings in other areas have highlighted the importance of differential parental treatment in the development of psychopathology such as antisocial behavior and depressive symptoms (Plomin, Chipuer, & Neiderheiser, 1994; Reiss et al., 1995). These studies have suggested that conflictual and negative parenting behavior directed specifically at one child in the family is associated with antisocial and depressive symptoms in adolescence and adulthood. Finally, studies of nonshared environmental influences on bulimia nervosa (BN), an eating disorder found to be significantly related to AN (Kendler et al., 1991; Walters & Kendler, 1995), indicate that differential parental treatment is a more influential risk factor for BN than other nonshared environmental variables, such as differential sibling or peer relationships (Lehoux, in press; Wonderlich, Ukestad, & Perzacki, 1994).

In summary, previous theory and research suggest that differential parental treatment may be a significant risk factor for a range of psychopathology, including AN. Despite this evidence, very few studies have investigated relationships between differential parental treatment and AN.

Differential Parental Treatment in AN

Shared Environmental Effects

Specific shared environmental influences are considered "between-family" effects that are examined by comparing families with an AN individual to families without any eating disordered members. Because much more research has examined these types of environmental influences than the nonshared ones discussed below, their findings will be briefly reviewed here. Results from these studies can be useful for generating hypotheses about the types of environmental influences that might operate differentially within the family.

In general, these between-family studies have found that families with an AN child differ from families without an eating disordered offspring on a number of dimensions. For example, Minuchin (1974) hypothesized that AN families are typically characterized by four interaction patterns: enmeshment (i.e., family members are overly involved with each other with diffuse interpersonal boundaries), overprotectiveness (i.e., family members are overly concerned about the welfare of other family members), rigidity (i.e., family members do not need or want any change in the family), and poor conflict resolution (i.e., the family has a low threshold for conflict and thus avoids resolving issues). Minuchin further hypothesized that these types of interactions, particularly enmeshment and overprotectiveness, do not allow the AN individual to

become autonomous during adolescence, causing her to assert herself through overcontrolled eating. Subsequent empirical research has supported aspects of Minuchin's theories, finding that parents in families with an AN child tend to be more rigid and controlling (Bruch, 1973; Goldstein, 1981; Garfinkle et al., 1983; Palazzoli, 1974), as well as more overprotective and enmeshed (Foulkes, 1996; Horesh et al., 1996), than control parents.

Between-family research has also specifically focused on the mother-daughter relationship as a potential contributor to eating pathology. There is evidence of poor communication (i.e., communication that is characterized by reciprocal criticality and destructiveness) between mothers and AN daughters (Lattimore et al., 2000). Maternal overprotectiveness and control have also been significantly associated with AN (Johnson, 1991; Pike & Rodin, 1991; Walters & Kendler, 1995).

In general, findings from between-family studies suggest that families with an AN individual are more overly involved, controlling, and rigid than families without an eating disordered individual. In particular, it appears that mothers of individuals with AN are more controlling and overprotective than mothers of individuals with no eating pathology. To date, no between-family research has examined the father-daughter relationship, despite recent findings highlighting its importance (Wonderlich et al., 1994). Patterns that characterize families with eating pathology and serve as shared environmental factors for siblings may also operate as nonshared factors as well, affecting siblings differentially within the same family.

Nonshared Environmental Effects

Although findings show that families with an AN child differ from families without a child with AN, most children in these families do not develop AN. Consequently, it is important to examine possible differential parental treatment within the family that may have contributed to the development of AN in one child versus another. These nonshared environmental effects are considered "within-family" influences that are frequently examined by comparing siblings' experiences of parenting within the same family.

Only two studies have examined nonshared environmental effects in AN. Murphy, Troop, and Treasure (2000) conducted a discordant sibling study (i.e., only one sibling has the disorder of interest), with AN women and their unaffected sisters who were closest to them in age and had no history of any eating pathology (i.e., they did not have AN, BN, or "Eating Disorder Not Otherwise Specified"). Both sisters completed the Sibling Inventory of Differential Experience (SIDE; Daniels & Plomin, 1985), which is a measure designed specifically to assess nonshared environmental influences such as differential parental treatment in the domains of affection and control. Subjects reported on their childhood experience over the years when they were living in the parental home, before the onset of the AN.

Findings from this study provided support for some differential parental treatment effects in the areas of maternal and paternal control. Specifically, the authors found that AN women perceived more maternal control than their sisters, whereas their sisters reported experiencing slightly greater paternal control (Murphy, Troop, & Treasure, 2000). Differences in parental affection were nonsignificant (Murphy, Troop, &

Treasure, 2000). These findings suggest that parental control may be one nonshared environmental factor related to AN.

In a second study, Karwautz et al. (2001) examined nonshared environmental factors associated with AN by again investigating sisters who were discordant for AN. Unaffected sisters had no history of any form of eating disorder (i.e., no AN, BN, or Eating Disorder Not Otherwise Specified). Participants with AN and their sisters were less than 10 years apart in age and had lived in the same family for at least eight years. Differential parental treatment was assessed with the Oxford Risk Factor Interview (Fairburn, Welch, Doll, Davies, & O'Conner, 1997), a semi-structured interview designed to examine a range of risk factors for an eating disorder, including differential parental treatment variables (e.g., criticism, affection, control, etc.). Sibling pairs also completed the SIDE described above. On both measures, participants gave retrospective accounts of the time spent in their childhood home, before the onset of AN in the proband.

These authors found some evidence of differential parental treatment effects, although their results did not replicate those of Murphy et al. (2000). Karwautz et al. (2001) found significant results on the Oxford Interview in the "parental expectations" domain; sisters with AN reported experiencing higher parental expectations than their unaffected sisters. However, unlike Murphy et al. (2000), they failed to find differences in the "over-involvement," "minimal affection," and "control" domains on the Oxford Interview, and no significant differences in parental affection or control were found on the SIDE. This study did not differentiate between maternal and paternal treatment, which may explain the discrepant results.

In summary, findings suggest that certain types of differential parental treatment (e.g., higher parental expectations and maternal control) are experienced by individuals with AN. However, these studies have limitations that restrict the strength of conclusions that can be drawn. First, parent reports were not been obtained, and thus it is unclear whether reported differences in parenting reflect objective or perceived differences on the part of the child. Given reported discrepancies between child and parental reports of nonshared environment (Elkins, McGue, & Iacono, 1997; McGue, Walden, Elkins, & Iacono, in press), it is important to examine both parent and child perceptions of these nonshared influences.

Second, previous studies have generally not explored paternal relationships, despite research with BN showing significant paternal effects on this disorder (Wonderlich et al., 1994). Third, research has suggested significant genetic influences on environmental measures (Plomin & Bergeman, 1991), making it difficult to determine whether observed differences are purely environmental or reflect, in part, genetic differences among siblings. Environmental measures may inadvertently measure both genetic and environmental variables because certain individual characteristics, such as temperament, are genetically influenced and affect the way that people perceive their environment. Thus, experiences with parents may appear to be environmental, but they are filtered through the individual, which necessarily includes genetic influence. Previous studies are limited in their ability to differentiate the effects of genes from nonshared environment because they include siblings whose behavioral and environmental differences may reflect genetic as well as environmental influences. The only type of study immune to this limitation is the discordant monozygotic twin design;

this controls for genetic effects by including MZ co-twins who have identical genetic material. All differences that are found between MZ twins must therefore be due to nonshared environment.

Finally, studies conducted thus far have not included a control group of siblings and parents, which makes it difficult to assess whether differential parental treatment is more prevalent between siblings discordant for AN than between siblings with no eating pathology (Klump et al., 2002). Observed effects in differential parental treatment in siblings discordant for AN may be normative and not significantly different from families with no eating pathology. Including a control group without eating pathology therefore helps determine whether AN families differ significantly from control families on this dimension.

Conclusions

Although there is substantial evidence for the importance of nonshared environment in the development of AN, more research is needed to elucidate the specific nonshared environmental factors accounting for the observed variance. Findings thus far have suggested that individuals with AN experience higher parental expectations and more maternal control than their sibling without eating pathology (Kaurwautz et al., 2001; Murphy et al., 2000). These findings highlight the potential role of differential parenting in the development of AN.

The present study examined both between- and within-family environmental influences on AN in a population-based sample of female twins. Specifically, this study first examined between-family factors, and then determined whether these factors operated in a nonshared manner as well. Primary analyses examined differences between

twins with AN and twins with no eating pathology on the Parental Environment Questionnaire (PEQ; Elkins et al., 1997) which assesses parental treatment in the areas of conflict with parent, involvement with parent, daughter's regard for parent, parent's regard for daughter, and structure imposed by parents (e.g., rules). Secondary analyses examined relationships between scores on the PEQ and the Minnesota Eating Disorder Inventory (M-EDI; Klump, McGue, & Iacono, 2000) among the entire Minnesota Twin Family Study (MTFS) twin sample. The M-EDI assesses eating attitudes and behaviors in the areas of body dissatisfaction, compensatory behavior, binge eating, and weight preoccupation.

This study improves upon previous research in this area by including both child and parental reports, by examining MZ twin pairs discordant for AN to separate genetic from environmental effects, and by including a control group of twins with no eating pathology in order to determine whether differential parental treatment in AN families varies from families with no eating pathology. Findings from this study have the potential to significantly increase understanding of specific nonshared environmental factors influencing AN.

Specific Aims and Hypotheses

Primary Aims

The primary aims of this study were to examine how differential parental treatment influences AN. Both between-family and within-family differences were examined in analyses.

Between-Family Effects

The first primary goal of the proposed study is to examine between-family, or shared environmental, influences by comparing parental treatment in families with AN and families with no eating pathology.

- Hypotheses 1: AN twins as a group will report more involvement with their mothers than control twins.
- Hypothesis 2: AN twins will report more structure by both their mothers and fathers than control twins.
- Hypothesis 3: AN twins will report less involvement with their fathers than control twins.
- Hypothesis 4: AN parents will report more conflict than the parents of control twins.

Within-Family Effects

The second primary goal of the study is to examine whether these shared environmental effects operate within the family as nonshared environmental influences by comparing differences in parental treatment within AN families to families with no eating pathology. Discordant twin pairs (both MZ and DZ) will be included in these analyses in order to determine whether differential parental treatment is associated with various outcomes in children with regard to eating pathology.

- Hypothesis 5: AN twins will report more involvement with their mothers than their co-twins, in comparison to control twin pairs.
- Hypothesis 6: AN twins will report less involvement with their fathers than their co-twins, in comparison to control twin pairs.

- Hypothesis 7: AN twins will report experiencing lower parent regard than their co-twins, in comparison to control twin pairs.
- Hypothesis 8: AN parents will report more conflict with the AN twin than with her co-twin, in comparison to parents of control twin pairs.

The third objective of the study is to examine purely nonshared environmental influences by examining within-pair differences reported by MZ pairs discordant for AN.

- Hypothesis 9: The above noted differences in parental treatment (e.g., more maternal involvement, less paternal involvement, more parental structure, and lower parental regard for AN twins) will continue to be significant in the MZ twin only sample.

Secondary Aims

The secondary aims of this study were to examine the relationship between eating pathology and differential parental treatment in a larger sample using a continuous measure of eating pathology (Minnesota Eating Disorder Inventory; M-EDI). A larger sample size will lead to increased statistical power and to examine specific nonshared environmental influences on disordered eating symptoms. Analyses will examine both between-family and within-family differences in a manner similar to the previous analyses.

Between-Family Effects

The first secondary aim is to examine relationships between parental treatment and M-EDI scores in a population-based sample of female twins.

- Hypothesis 10: There will be a positive relationship between M-EDI scores and involvement with mothers; twins with higher M-EDI scores

will report more involvement with their mothers than twins with low M-EDI scores.

- Hypothesis 11: There will be a positive relationship between M-EDI scores and structure; twins with higher M-EDI scores will report experiencing more structure than twins with low M-EDI scores.
- Hypothesis 12: There will be a negative relationship between M-EDI scores and involvement with fathers; twins with higher M-EDI scores will report less involvement with their fathers than twins with low M-EDI scores.

Within-Family Effects

The second secondary aims will examine whether shared environmental effects operate in a nonshared manner, as discussed in the primary aims section. Within-pair differences among MZ and DZ twin pairs on the M-EDI and PEQ scores will be used to index differential parental treatment and differences in eating pathology. Correlations will be used to relate differential parental treatment with twin differences in eating pathology, using differences in M-EDI and PEQ scores among twin pairs will also be conducted.

- Hypothesis 13: There will be a positive relationship between difference scores on the M-EDI and involvement with mothers; twins with higher EDI scores will experience more involvement with their mothers than their co-twins.
- Hypothesis 14: There will be a negative relationship between difference scores on the M-EDI and involvement with father; twins

with higher M-EDI scores will report less involvement with their fathers than their co-twins.

- Hypothesis 15: There will be a negative relationship between M-EDI difference scores and parent regard; twins with higher M-EDI scores will report experiencing lower parental regard than their co-twins.

The final secondary aim will examine relationships depicted above for MZ twins only. These analyses will examine whether relationships are due primarily to nonshared environmental influences.

- Hypothesis 16: Observed relationships between differences in parenting and M-EDI scores (i.e., positive relationship for involvement with mother, negative relationships for parent regard and involvement with father) will continue to be significant in the MZ twin sample.

METHODS

Participants

Participants included a cohort of female twins (n = 321), measured at 17 and 20 years of age, who took part in the Minnesota Twin Family Study in 1990. The MTFS is a longitudinal, population-based study investigating the development of substance use and other disorders in reared-together female twins and their parents. Families with twins were ascertained using state of Minnesota birth records and were contacted using information from public databases, such as telephone directories and driver's license registrations. The MTFS staff was able to locate over 90% of twin births in Minnesota per any given birth year (Lykken, Bouchard, McGue, & Tellegen, 1990).

Twenty-seven twin pairs in which at least one twin had AN were identified from the larger sample and were included in analyses. In addition, twenty-seven control twin pairs with no eating pathology (i.e., no AN, BN, or binge eating disorder) were chosen through random selection. Analyses necessarily included different sample sizes, as different sets of twins were needed for analyses of each aim (see tables).

Zygosity Determination

Twin zygosity was determined in several ways. Measures of zygosity included a parental zygosity questionnaire regarding the physical similarity of the twins, a project zygosity estimate determined by a research assistant's assessment of the twins' similarity in eye color, ear configuration, and general physical resemblance, and an algorithm diagnosis calculated from ponderal index, cephalic index, and fingerprint ridge count. Disagreements among these three indexes were resolved through a serological examination of 12 blood group antigens and protein polymorphisms. A validation study was run using 50 pairs of twins, all of whom had the serological test. In every case in which the three zygosity estimates agreed, the serological analysis confirmed the agreement.

Measures

AN Diagnoses: DSM-IV (APA, 1994) AN diagnoses were assessed with the Eating Disorder Structured Clinical Interview (EDSCI), a structured clinical interview based on the Eating Disorders Module of the Structured Clinical Interview for DSM-III-R (SCID) (Spitzer, Williams, & Gibbon, 1987). The EDSCI was administered by trained clinical interviewers who had a bachelor's degree or higher in psychology. These

interviewers were trained by MTFS clinical psychologists and clinical psychology doctoral students. The interviewers coded items on a three-point scale: threshold (definitely present), subthreshold (e.g., in severity, frequency, or pervasiveness), or absent. Item codings made by the interviewers were reviewed by teams consisting of two or more doctoral students in clinical psychology. The AN diagnoses were then made by computer algorithm. Interviewers and reviewers were unaware of twin zygosity during assessments.

To ensure the integrity of the eating disorders diagnoses, the EDSCI of twins who met criteria for one or more EDSCI items were also reviewed by two experts in the assessment of eating disorders (Klump et al., 2001). In addition, twin responses on the Minnesota Eating Disorders Inventory (M-EDI; Klump et al., 2000; von Ranson et al., submitted), a self-report questionnaire of overall eating pathology (see more details below), were used to confirm significant disordered eating symptoms.

Two levels of diagnoses were used. Subjects who met full criteria for AN ("definite" diagnosis) or fell one symptom short ("probable" diagnosis) were combined to form a "probable/definite" group. Low body weight (i.e., at least 10% below ideal body weight) was a necessary symptom for inclusion in this group. Subjects exhibiting characteristics of AN ("AN Characteristics") comprised the second group. Twins were included in the "AN Characteristics" category if they were at least 10% below ideal body weight, they met criteria for at least one cognitive symptom of AN, and they scored above the mean (i.e., 11.0) for all twins with "probable/definite" eating disorders on the M-EDI. The combined category, including "probable/definite" diagnoses of AN and "characteristics" of AN, was used to increase statistical power of analyses. The decision

to combine cases is supported by research showing that AN symptoms lie on a continuum, with subthreshold and threshold AN diagnoses resembling each other (Walters & Kendler, 1995). As further evidence of this, differences in parental relationships were not found between the subthreshold and threshold AN twins included in this study (data not shown).

It should also be noted that the AN twin group consisted of women defined as currently ill (n = 13) or recovered (n = 14) at the time of assessment. Twins were considered ill if they reached their lowest weight or had other significant AN symptoms within one year prior to the time of assessment. Twins were categorized as recovered if they reached their lowest weight or did not have other significant AN symptoms more than one year before the assessment. No differences in parental relationships were found between ill and recovered AN women in this study (data not shown). Therefore, the groups were combined for the analyses.

The reliability of all AN diagnoses was examined by comparing the diagnoses assigned by two independent review teams. The kappa coefficient (.63) for the EDSCI diagnoses for AN was moderate; however the additional review of these diagnoses by eating disorder specialists and the inclusion of M-EDI scores as additional diagnostic criteria ensured the integrity of the AN diagnoses.

Minnesota Eating Disorder Inventory: Twin participants completed the 30-item Minnesota Eating Disorder Inventory (M-EDI; Klump et al., 2000; von Ranson et al., submitted) to assess eating attitudes and behaviors. The M-EDI is a revised version of the original Eating Disorders Inventory (EDI; Garner, Olmsted, & Polivy, 1983); the original M-EDI was modified by MTFS researchers to make it suitable for use with

preadolescent girls. A previous factor analysis (Klump, McGue, & Iacono, 2000) of the M-EDI yielded four subscales: Body Dissatisfaction (dissatisfaction with the size and/or shape of one's body), Weight Preoccupation (preoccupation with dieting, weight, and the pursuit of thinness), Binge Eating (the tendency to engage in episodes of overeating as well as having attitudes conducive to binge eating), and Compensatory Behavior (the tendency to use or contemplate using inappropriate compensatory behaviors such as self-induced vomiting and laxatives to control weight). The M-EDI also includes a total score, which is the combined score of all 30 items. The M-EDI scales are scored in the traditional "pathological" direction with high scores indicating greater degrees of the measured construct.

The reliability and validity of the M-EDI have been supported by previous studies (Klump et al., 2000; von Ranson et al., submitted). Specifically, internal consistency and three year test-retest statistics were adequate among the subscales. It is noteworthy that the test-retest statistics remained high over a three-year span, as these values reflect not only measurement error but also trait stability over a three-year period when numerous situational changes are occurring (i.e., going to college). These statistics thus attest to the strong reliability of the measure. Previous studies have also supported the ability of the M-EDI to discriminate between eating disordered subjects and controls (von Ranson et al., submitted).

Parental Treatment: Parent-child relationships were assessed with the Parental Environment Questionnaire (PEQ; Elkins et al., 1997), which was developed by MTFS researchers to measure the quality of relationships between parents and children. The PEQ includes five subscales derived from factor analysis (Elkins et al., 1997): conflict

(i.e., child is criticized and seen as a source of irritation, parent-child relationship is marked by frequent arguments), parent involvement (i.e., parent takes an active interest in child's activities, parent and child communicate openly), regard for parent (i.e., parent is seen as someone to emulate, parent is a source of pride), regard for child (i.e., parent is proud of child), and structure (i.e., parent specifies a set of rules that he/she expects to be followed). Twin and parent reports were obtained on the PEQ, such that parents reported on their relationship with each twin separately, and twins reported on their relationship with each parent. As noted in Table 1, internal consistency reliabilities for the PEQ scales were sufficient for both twin and parent reports.

Statistical Analyses

Correlations between parent and child reports, in addition to mother and father reports, were calculated for each of the PEQ scales. As this is one of the first discordant twin studies of AN, a more liberal p-value of .05 was used for analyses.

The remaining statistical analyses were conducted in three steps corresponding to each primary and secondary aim.

Primary Aims

Analysis 1: The first set of analyses compared group means on the PEQ subscales, in order to examine between-family differences in parenting across AN and control families. Independent sample t-tests were conducted to compare PEQ means between twins with AN and twins with no eating pathology.

Analysis 2: The next set of analyses examined within-family differences to help determine whether twins with AN experience differential parental treatment to a greater

extent than their co-twins and individuals with no eating pathology. MZ and DZ twins discordant for AN (n = 23) were included. Twins who were concordant for AN (n = 4) were excluded, because they did not reflect different outcomes in eating pathology that could be studied in relation to differential parental treatment. A repeated-measure analysis of variance (ANOVA) test was used. However, instead of the typical method of comparing individuals at two different time periods, twins and their co-twins were evaluated where Twin 1 was considered to be equivalent to Time 1 and Twin 2 was equivalent to Time 2.

Analysis 3: The final set of analyses examined only MZ twins discordant for AN (n = 11), in order to determine whether observed differences were purely environmental in origin. A repeated-measures ANOVA was used to examine whether differences in PEQ scores were similar to those found in the combined MZ/DZ twin analyses.

Secondary Aims

Secondary analyses were conducted in the entire female twin cohort with M-EDI scores in order to determine whether there was a relationship between eating pathology and differential parental treatment in a larger sample of twins using a continuous measure of eating pathology. These analyses were again conducted in three sections similar to those described above under "Primary Aims".

Analysis 1: The first set of analyses examined the relationship between M-EDI scores and PEQ scores, to test for a relationship between eating pathology and parental

treatment. Correlations were calculated between M-EDI total scores and PEQ scale scores from the entire twin population (n = 321).

Analysis 2: The next set of analyses examined whether differences in M-EDI scores within twin pairs were associated with differential parental treatment. Both MZ and DZ twins were included in these analyses. Difference scores were used to index differential parental treatment and twin differences in eating pathology, and they were calculated consistently in the same direction (twin 1 – twin 2) on both measures (i.e., the M-EDI and PEQ) in order to observe the accurate direction of the relationship (i.e., positive or negative). Correlations were then calculated between within-twin pair difference scores on the M-EDI and the PEQ.

Analysis 3: The final set of analyses examined only MZ twins (n = 210) in order to determine whether observed sibling differences were primarily the result of genetic or environmental influences. Correlations were calculated between within-pair difference scores on the M-EDI and the PEQ.

RESULTS

Correlations between Child and Parent Reports on the PEQ

Correlations between child and parent reports, in addition to mother and father reports, showed significant positive relationships on most of the PEQ scales (see Table 2). Mothers and fathers showed moderate to high significant positive correlations, suggesting that they seemed to view parental relationships similarly. However, parents

and children viewed their relationships less similarly. Mothers and daughters were similar in perceptions of conflict, involvement, and regard for parent, but less similar in regard for child and structure. Fathers and daughters had the most disparity between reports with low to moderate correlations on all PEQ scales, suggesting that they had some different perceptions of their relationship, especially in the areas of regard for child and structure.

Primary Aims

Between-Family Effects

Analyses examining differences in parental treatment between AN and control twins are presented in Table 3. Despite the expectation that there would be differences in parental treatment between the two groups, no significant between-family differences were found with either the twins' or parents' reports.

Within-Family Effects

The second and third sets of hypotheses predicted differences in parental treatment between MZ and DZ twins discordant for AN, in comparison to control twin pairs with no eating pathology. However, no significant within-family differences were found between these groups using twin or parent reports (see Table 4). In addition, there were no significant differences among MZ twins discordant for AN and control twin pairs (see Table 5). These findings suggest that differences in parental treatment were not related to differences in AN within twin pairs.

Secondary Aims

Between-Family Effects

Additional analyses of between-family effects were conducted with all twins in the sample, using the M-EDI. These analyses were used to examine whether disordered eating characteristics show relationships with parental treatment. Negative relationships between disordered eating and paternal involvement were predicted and this was supported by twin and parent reports (see Table 6). All M-EDI scales showed significant negative relationships with parental and twin reports of paternal involvement, suggesting that more disordered eating was associated with less paternal involvement.

Other hypotheses were generally not confirmed, however. It was hypothesized that there would be positive relationships between disordered eating and maternal involvement, and between disordered eating and parental structure. However, twin and parent reports indicated that all M-EDI scales showed significant negative relationships with maternal involvement and showed no significant relationships with structure. These results suggest that higher levels of disordered eating are actually associated with less maternal involvement, and that structure is not associated with disordered eating.

Other significant findings emerged that were not addressed in the original hypotheses. All M-EDI scales showed significant positive relationships with parental and twin reports of maternal and paternal conflict, indicating that twins who experience more conflict with their parents show more disordered eating attitudes and behaviors. Twin and parent reports of general disordered eating (M-EDI Total score), and Binge Eating in particular, showed significant negative relationships with regard for parent, while Body Dissatisfaction also showed significant negative relationships with regard for father. These findings suggest that various forms of disordered eating are associated with lower regard for parents by children.

An interesting difference between twin and parent reports emerged with the regard for child variable, which showed small to no relationships with disordered eating in parent reports, and showed significant relationships with all M-EDI scales in the twins' reports. Twin reports indicated that all M-EDI scales showed significant negative relationships with regard for child, while mothers did not show this pattern and father reports only showed a significant negative relationship between Compensatory Behavior and regard for child. This discrepancy suggests that there is a different perspective among parents and children about the esteem in which children are held by their parents, which may be related to disordered eating. Children who do not feel loved and valued by their parents also show more disordered eating, whether or not the parents actually feel this way about their children.

Within-Family Effects

Relationships between difference scores among twin pairs on the M-EDI and PEQ scales are presented in Table 7. Hypotheses predicted that there would be negative relationships between differential disordered eating and differential paternal involvement, and between differential disordered eating and differential regard for child, in one twin relative to the other. Positive relationships between differential maternal involvement and differential disordered eating were also predicted.

Fewer significant relationships were found than in the between-family analyses, suggesting that some of the between-family effects do not operate in a nonshared manner. Some of the hypotheses were supported by the data. For example, negative relationships were observed between twin reports of differential paternal involvement and disordered eating variables, such that differential paternal involvement was related to increased

Weight Preoccupation and Body Dissatisfaction in one twin relative to the other. This suggests that the twin with higher Weight Preoccupation and Body Dissatisfaction scores also reported the least amount of paternal involvement. However, these relationships were only apparent in twin reports. In addition, differential disordered eating, as indexed by most M-EDI scales, showed significant negative relationships with twins' report of differential paternal and maternal regard. These results were only partially supported in parents' reports where father's regard for child was significantly correlated with Binge Eating difference scores, but no significant associations emerged in mothers' reports. Therefore, twin perceptions of parental regard are related to differential development of disordered eating, while parent reports do not show this relationship. This suggests that the perception of being held in lower regard by one twin relative to the other is related to disordered eating, whether or not parents actually feel lower regard for the child. These findings also indicate that twin reports of paternal involvement and regard for child and their relationship to disordered eating operate both between- and within-families. However, parent reports of these parental relationship variables operate only between-families.

One hypothesis was not supported by the data. Differential Compensatory Behavior, reported by twins and mothers, and differential Weight Preoccupation reported by twins only, showed significant negative relationships with differential maternal involvement and the other scales did not have significant relationships with maternal involvement. These results indicate that greater levels of some forms of disordered eating are actually associated with less maternal involvement within-families. Maternal

involvement was also found to operate as a factor related to disordered eating between-families.

Another relationship was found that was not addressed by the hypotheses, between conflict and disordered eating; relationships were also found with the conflict variable in the between-family analyses, without being predicted. Twin reports of differential Weight Preoccupation showed significant positive relationships with maternal and paternal conflict, and differential Binge Eating showed significant negative relationships with differential paternal conflict. This suggests that higher levels of weight preoccupation were associated with higher levels of maternal and paternal conflict experienced by one twin relative to the other. Parent reports did not completely corroborate twin reports. Mothers' scores showed no relationships between differential conflict and any of the M-EDI scales and fathers only showed positive relationships between differential Compensatory Behavior and differential paternal conflict. Twin reports of differential conflict were found to operate within-families, as well as between-families, while for parents, parental conflict effects were only found in the between-family analyses.

Hypotheses for the final set of analyses were that observed differences in the previous analyses with MZ and DZ twins would continue to be significant within the MZ twin sample. These analyses were conducted to determine how much of the observed effects between differential parental treatment and differential disordered eating were truly environmental in origin. Correlations between difference scores on the M-EDI and PEQ among MZ twins revealed some evidence of differential parental treatment that

were the same as previous results with the combined sample of MZ and DZ twin pairs, however not all relationships remained significant in the MZ sample (see Table 8).

As in the MZ/DZ sample, differences in twin reports of M-EDI Total scores showed significant negative relationships with differential maternal and paternal involvement, suggesting that higher levels of disordered eating were associated with less maternal and paternal involvement in one twin relative to the other. General Disordered Eating, and Body Dissatisfaction in particular, showed significant negative relationships with maternal regard, indicating that higher levels of maternal regard in one twin relative to the other was associated with more disordered eating behaviors. However, this relationship was not found in mother reports. Parent reports showed differential parental treatment that was associated with Compensatory Behavior within twins, which was also found in the MZ/DZ analyses. Paternal conflict showed positive relationships to higher levels of Compensatory Behavior in one twin relative to the other, according to father reports. Less regard for mother by one twin in comparison to her co-twin, as reported by mothers, was associated with higher levels of Compensatory Behavior in that twin.

Taken together, these results suggest that according to twins, parental involvement and maternal regard may be specific forms of differential parental treatment that influence eating pathology. Based on parent reports, paternal conflict and regard for mother are forms of differential parental treatment that may be associated with eating pathology. These findings suggest that different aspects of the parental relationship are salient to twins and parents. Unlike previous findings in the combined sample of MZ and DZ twins, these identified factors within the parental relationship are completely

environmental in origin, as observed differences between MZ twins must be due to environment.

DISCUSSION

This study is one of the first to examine relationships between differential parental treatment and eating pathology. No relationships between differential parental treatment and disordered eating were found in the categorical analyses comparing AN and control twin pairs. However, analyses of continuous measures of eating pathology in a non-clinical sample showed that both between- and within-family parental treatment factors are associated with disordered eating characteristics. These findings suggest that parental relationships in general, and some forms of differential parental treatment, are associated with general disordered eating, but these findings do not appear to extend to clinical populations.

Relationships between AN and Differential Parental Treatment

As noted above, this study did not find evidence for differential parental treatment in AN women. No effects were found between or within families, indicating that no shared or nonshared parental relationship factors were found to be associated with AN.

Previous studies did find some relationships between differential parental treatment and AN. With the exception of parental involvement, this study did not measure the dimensions addressed in other studies directly, so it is difficult to make comparisons between them. Previous research showed evidence for higher parental expectations and maternal control in AN women, but did not find support for differential parental involvement (Kaurwautz et al., 2001, Murphy et al., 2000). The current study supports the lack of significant findings of differential parental treatment in the area of

parental involvement reported by Kaurwautz et al. (2001). However, this study did not include a measure of parental expectations or maternal control, so findings from this study cannot substantiate previous results in these areas. The maternal involvement scale on the PEQ focuses more on emotional closeness between parents and daughters, rather than focusing on control as delineated by the scale used in the study by Murphy el al. (2000).

There are many possible reasons for the general lack of significant findings in the AN sample. One possibility is that twins with threshold and subthreshold AN diagnoses and those who were ill and recovered, were all included in the AN group. There could be differences in parental relationships between these groups of AN women and their inclusion in the AN sample may have made it difficult to identify differential parental treatment effects. However, this likely did not affect the results because threshold and subthreshold AN twins were compared, as were ill and recovered AN twins, and differences in parental relationships were not found.

Another possible reason for nonsignificant effects is that participants have not passed through the period of risk for developing eating disorders, so control subjects and co-twins of AN women could develop AN or BN in the future. Therefore, twin pairs discordant for AN could become concordant for AN, if the co-twin develops AN. This would mean that attempts to measure factors associated with the differential development of eating pathology within twin pairs would be inaccurate, because there would not be a differential development of eating pathology. In addition, twins from the control group could develop AN or BN at some point in the future, because the period of risk for developing AN or BN lasts until age 25. However, twins were diagnosed with AN and

BN up to age 20, and AN typically begins between the ages of 14 and 18, while BN begins in late adolescence or early adulthood (DSM-IV; APA, 1994). Therefore, the potential for future development of eating disorders among control twins or co-twins of AN women is not likely to have had a substantial impact on the results.

One other possible explanation for nonsignificant results is that the correlations between the M-EDI and PEQ scales were small in magnitude, indicating that effects in AN groups would likely be small as well. A power analysis was conducted to determine what power the analyses with the AN group had to detect these small effects. For the between-family analyses, the effect sizes for the correlations were low (d = .09-.27) and the power to detect these differences within the AN sample was also low (19 - 48%). For the within-family analyses, the effect sizes in the correlations were small (d = .13-.22), as was the power to detect these effects within the AN MZ/DZ group (15 - 28%). The power to detect small effects (d = .14-.21) within the AN MZ twin sample was even lower (10 – 16%). These power analyses reveal that while differential parental treatment may be an important and significant factor related to the development of disordered eating, its effects are small and difficult to detect in small sample sizes.

A final explanation is that differential parental treatment may be an important factor that is not completely captured by the PEQ. The PEQ does not measure elements of the parental relationship commonly investigated in relation to eating disorders, such as enmeshment (Minuchin, 1978). The inability of the PEQ to capture the relative constructs, in combination with small effect sizes, may explain the lack of significant results in the clinical sample. Previous studies using other measures of differential parental treatment have reported larger effect sizes than those found in analyses with the

AN sample in this study (Kaurwautz et al., 2001; Murphy et al., 2000). The PEQ may be less effective at identifying factors in the parental relationship that relate to eating disorders than other measures designed for this purpose.

Relationships between General Disordered Eating and Differential Parental Treatment

Between-Family Effects

Results from analyses using a continuous measure of disordered eating revealed that some variables of the parental relationship were associated with disordered eating characteristics. Although hypotheses predicting lower paternal involvement and parental regard were supported, the significant relationship between lower maternal involvement and more disordered eating behavior was contrary to original hypotheses. Individuals with more disordered eating actually appear to experience less close involvement with their mothers. In addition, structure was not found to have a relationship with disordered eating. Structure had low internal consistency as a construct, which may be one reason why it did not show relationships with the disordered eating variables. Findings that were not addressed by hypotheses emerged. Twin reports of all types of disordered eating were associated with lower regard from parent and greater conflict. Parent reports indicated that Body Dissatisfaction, Compensatory Behavior, and M-EDI Total scores were associated with lower regard for parents by their children. These results suggest that individuals with a range of disordered eating characteristics have more conflict with their parents, are less involved and experience less emotional intimacy with their parents, and have fewer positive feelings (e.g., pride, warmth) towards and from their parents.

Taken together, results from parent and twin reports support previous theory and research on differences between families with disordered eating and those without disordered eating. These families have been found to experience more conflict and problematic communication than other families (Lattimore et al., 2000; Minuchin, 1974). Associations between less paternal involvement and disordered eating also support anecdotal theories of a distant, unengaged father in families with disordered eating. These theories traditionally have not received much empirical support beyond individual case studies (Miller, 1997; Minuchin, 1978).

However, previous research and theory suggested the presence of maternal over-involvement and enmeshment in families with disordered eating (Foulkes, 1996; Horesh et al., 1996). The present study failed to find this association, but instead found that individuals with more disordered eating experienced less involvement with both parents. One possible reason for the discrepancy is that this study used the PEQ, which may not accurately measure variables of the parental relationship commonly associated with eating disorders. The PEQ involvement scale was not designed for eating disordered populations, and does not capture the enmeshment that was described by previous studies. Enmeshment describes an extreme form of proximity and intensity in family interactions, which is not completely covered in the involvement scale of the PEQ. The PEQ involvement scale does not include items referring to the weak boundaries, excessive togetherness, and lack of privacy of enmeshed families, but instead measures the extent to which parents interact with their children and are aware of their children's involvement in activities and hobbies. Therefore, the PEQ may not capture aspects of the parental relationship that are related to eating disorders.

Previous research also found evidence for more rigidity and structure in families with disordered eating, which was not supported by this study (Bruch, 1973; Goldstein, 1981; Garfinkle et al., 1983; Palazzoli, 1974). However, as discussed previously, the PEQ structure scale is less reliable than other PEQ scales. The items that comprise the structure scale are also not geared towards eating disordered families. The items have a conceptualization of structure that focuses on abiding by the law and rules about household chores. The rigidity that has been described in families with disordered eating refers to the difficulty in changing methods of interaction among family members, particularly during periods of change like puberty. Such a conceptualization of familial rigidity is not captured by the PEQ structure scale.

In summary, the between-family analyses corroborate some previous findings. In disordered eating families, less paternal involvement and more conflict were found, which supports previous theory and research. Greater maternal involvement and parental structure, identified by the literature as characteristic of disordered eating families, were not found in this study. However, the analyses do expand upon previous research, which has primarily focused on the maternal relationship, by exploring the paternal relationship as well. Findings showed that parental treatment from both mothers and fathers were equally associated with disordered eating. This suggests that the paternal relationship may be as important as the maternal relationship in relation to the development of disordered eating, especially in the areas of involvement and conflict.

Despite this discovery of significant between-family effects in disordered eating, twin studies (Klump et al., 2001; Wade et al., 2000) suggest that shared environment is not important for predicting individual differences in risk for developing an eating

disorder. It is possible that these shared environmental variables can increase the overall risk of developing disordered eating and help explain why certain families have disordered eating. However, the shared environmental variables do not account for individual risk factors and cannot explain the differential development of disordered eating within families. In addition, the between-family effects measured in this study could be the result of genes, shared environment, or a combination of both genes and shared environment. Subsequent analyses of nonshared environment were done to help identify individual risk factors for disordered eating, and provide insights into the extent to which the effects are purely environmental.

Within-Family Effects

The within-family analyses assessed whether the shared variables that were identified as common risk factors for disordered eating between families also operated within families in a nonshared environmental manner. Correlations showed some evidence for relationships between differential parental treatment and differential disordered eating attitudes and behavior. Within the combined MZ/DZ group, twins who reported more conflict with both parents than their co-twins, also reported more disordered eating behavior than their co-twins. Twins who reported experiencing less involvement with their parents and less positive regard from their parents also experienced more disordered eating behavior than their co-twins. Therefore, twins who argue and fight more with their parents, feel that their parents are less involved in their lives, and feel like their parents are less proud of them, in comparison to their co-twins, report more disordered eating than their co-twins. Not all disordered eating variables showed these relationships, however. Differential Body Dissatisfaction only showed

relationships with differential regard for child and paternal involvement, while differential Binge Eating showed relationships with differential paternal conflict and regard for child. Differential Compensatory Behavior only showed relationships with differential maternal involvement and paternal regard.

As in the between-family analyses, it is unclear whether the effects described above are genetic or environmental effects, since both MZ and DZ twins were included in the analyses. To examine this and determine how much of the effect was truly environmental in origin, only MZ twins were included in the final analyses. These correlations showed some significant relationships between differential disordered eating and differential parental treatment, but few relationships identified in the MZ/DZ group remained significant. According to twin reports, maternal regard and parental involvement showed significant negative relationships with disordered eating characteristics. This suggests that MZ twins who experience less positive regard from their mother and feel that their parents are not involved in their lives, in comparison to their co-twins, experience more disordered eating than their co-twins. Parent reports identified regard for mother and paternal conflict to be differential parental treatment factors associated with differential levels of Compensatory Behavior.

However, the effect sizes for these few significant correlations were small ($\underline{d}$ = .14-.21), suggesting that associations are not highly robust. This is likely because the analyses with MZ and DZ twins contained both environmental and genetic effects. With the MZ twins, any genetic effect on differential parental treatment was excluded and only purely environmental effects remained. There is likely some genetic influence on the

MZ/DZ results, because effects within the MZ sample were smaller and fewer in number than those detected within the MZ/DZ sample.

Differences between Twin and Parent Reports

The use of parent reports allows for a more comprehensive assessment of the parental relationship, because it goes beyond the child's experience of the relationship. A common theme through all of the analyses was subtle differences in parent and twin perceptions. As indicated by some of the low to moderate correlations between parents and twin perceptions of their relationships, twins and their parents did not always experience their relationship similarly. Between-family analyses indicated that different types of parental treatment variables were significant across twins and parents. Both twin and parent reports indicated that conflict and involvement were related to disordered eating. However, twin reports suggested that regard from their parents was related to disordered eating, while parent reports of twins' regard for their parents showed relationships with disordered eating. This suggests that the child's perception of having less love and pride from her parents is related to disordered eating, while parents' perceptions of being held in low esteem by their children are related to disordered eating. This suggests that the parent or twin's perception of how they are regarded by each other is related to disordered eating. It is possible that individuals modify their behavior towards each other because of their perception of the relationship, which then influences disordered eating.

Parents reported very few differences in their treatment of children which supports previous studies comparing parent and child reports of parental relationships (Elkins, McGue, & Iacono, 1997; McGue, Walden, Elkins, & Iacono, in press). These

studies found that parents generally do not report treating their children differently, although children may perceive differential parental treatment. The within-family correlations between difference scores on the PEQ and M-EDI showed that twins perceived more differential parental treatment than their parents. By only examining one perspective, it cannot be concluded that differential parental treatment is occurring, because it could be the child's perception of differential parental treatment rather than an objective experience. However, it can be noted that although parents may or may not be treating their daughters differently, the daughters do perceive differential parental treatment, and these differences are associated with the differential development of disordered eating. Research has shown that the perception of differential parental treatment by children is related to greater adjustment difficulties and behavioral problems for the child who is less favored (Dunn, Stocker, & Plomin, 1990; McGuire, Dunn, & Plomin, 1995). Studies have also shown that the child's perception of differential parental treatment is more predictive of academic performance than parent reports of parental treatment (Paulson, 1994). Therefore, a child's perception of differential parental treatment may be related to behavior, even if parents do not report differential parental treatment. Whether or not differential parental treatment actually occurs, children's perceptions of differential parental treatment are associated with behavioral outcomes.

Directions for Future Research

Overall, this study identified between-family and nonshared environmental factors that are related to the development of general disordered eating attitudes and behavior. However, these factors were not found to have a relationship with the

development of clinical levels of disordered eating in the AN sample. This study points to future directions for research in nonshared environmental factors and their relationship to disordered eating. Previous research has focused on the maternal relationship, despite evidence that the paternal relationship may be equally important in BN (Wonderlich et al., 1994). Results from this study suggest that maternal and paternal relationships are both important in the development of disordered eating. Both maternal and paternal involvement were significantly associated with disordered eating in the MZ twin correlations, indicating that paternal involvement may be a purely environmental factor that is as important as maternal involvement in influencing the development of eating pathology. Future research should examine both the maternal and paternal relationship since the paternal relationship may be equally, if not more, important than the maternal relationship in the development of eating pathology (Wonderlich et al., 1994).

More research is needed to identify elements of differential parental treatment that affect eating pathology. This study provides some evidence for differential parental treatment as a nonshared environmental factor in disordered eating using continuous measures of eating pathology, but further research is needed to identify the specific elements of differential parental treatment that are most influential in the development of clinical eating disorders. Future studies that include appropriate family environment measures for eating disorders are needed to investigate whether findings from the non-clinical sample extend to clinical samples of AN women. In addition, future research should utilize larger samples of AN twins to identify potentially small nonshared environmental effects. Studies should also focus on identifying other nonshared environmental factors that may be more influential in the development of eating

pathology than differential parental treatment. Other potentially influential nonshared environmental factors include prenatal factors, differential peer and sibling relationships, and life events. Twins may have different experiences of these nonshared environmental factors, which may be related to the differential development of eating pathology. Finally, research should use genetically-informed designs in order to separate genetic from environmental effects and determine which effects are truly environmental in origin.

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Table 1

Internal Consistency Reliability Estimates for PEQ Scales.

PEQ Scales	Mother Report	Father Report	Twin Report	
			Mother	Father
Conflict	.86	.85	.90	.90
Involvement	.77	.83	.89	.90
Regard for Parent	.80	.82	.88	.91
Regard for Child	.77	.67	.81	.87
Structure	.45	.55	.46	.47

Note. PEQ = Parental Environment Questionnaire. Alpha coefficient estimates are internal consistency coefficients. Parent and twin reliabilities are based on self-reports. Twin reports address relationships with both of their parents.

Table 2

Correlations Between Parent and Twin Reports of PEQ Scales.

PEQ Scales	Father-Daughter	Mother-Daughter	Father-Mother
Conflict	.40**	.51**	.59**
Parental Involvement	.43**	.43**	.64**
Regard for Parent	.34**	.45**	.55**
Regard for Child	.32**	.22**	.58**
Structure	.21**	.16**	.60**

Note. PEQ = Parental Environment Questionnaire. Father-Daughter describes correlations between father and daughter reports of their relationships. Mother-Daughter describes correlations between mother and daughter reports of their relationships. Father-Mother describes correlations between father and mother reports of their relationships with their daughters.

**$p<.01$

Table 3

Mean Differences in PEQ Scores Between AN and Control Twins.

PEQ Scales	AN n = 31	Control n = 598	T (df)	P
Mother Report:				
Conflict	21.52 (7.45)	20.88 (6.11)	-.50 (534)	.62
Parental Involvement	42.24 (4.03)	42.48 (4.03)	.28 (532)	.78
Regard for Parent	27.88 (3.94)	27.72 (2.89)	-.28 (533)	.78
Regard for Child	19.60 (.82)	19.55 (.99)	-.24 (538)	.81
Structure	17.36 (1.68)	17.16 (1.69)	-.58 (538)	.56
Father Report:				
Conflict	21.46 (5.17)	21.74 (6.35)	.22 (465)	.83
Parental Involvement	40.50 (4.23)	38.56 (5.77)	-1.63 (466)	.11
Regard for Parent	27.29 (2.29)	27.06 (3.33)	-.33 (465)	.74
Regard for Child	19.58 (1.10)	19.19 (1.43)	-1.32 (471)	.19
Structure	16.79 (2.09)	16.95 (1.99)	1.12 (466)	.27
Twin Report:				
Maternal Conflict	23.61 (7.56)	23.01 (7.29)	-.45 (623)	.65
Maternal Involvement	38.35 (7.05)	38.97 (6.55)	.51 (618)	.61
Regard for Mother	28.10 (3.94)	28.24 (3.73)	.21 (620)	.83
Mother's Regard for Child	18.00 (2.46)	18.45 (2.22)	1.09 (625)	.28
Maternal Structure	16.87 (2.45)	16.57 (1.99)	-.80 (622)	.42
Paternal Conflict	22.62 (7.05)	23.94 (7.23)	.15 (615)	.88

PEQ Scales	AN n = 31	Control n = 598	T (df)	P
Paternal Involvement	35.62 (8.02)	35.39 (7.79)	-.16 (611)	.87
Regard for Father	27.62 (4.55)	26.96 (4.79)	-.73 (612)	.46
Father's Regard for Child	17.97 (2.51)	18.09 (2.76)	.24 (618)	.81
Paternal Structure	16.62 (2.47)	16.19 (2.20)	-1.02 (612)	.31

Note. PEQ = Parental Environment Questionnaire. AN = anorexia nervosa.

Table 4

Within-pair Differences in Parental Treatment in AN and Control Twin Pairs.

PEQ Scales	AN Pairs[a] n = 23	Control Pairs n = 270	F (df = 1)	P
Mother Report:				
Conflict				
Twin 1	20.14 (5.95)	22.80 (7.29)	1.77	.19
Twin 2	20.19 (6.34)	20.84 (6.09)		
Parental Involvement				
Twin 1	42.19 (4.16)	39.25 (6.47)	3.45	.06
Twin 2	43.86 (4.11)	42.50 (4.31)		
Regard for Parent				
Twin 1	28.24 (3.21)	28.20 (3.79)	.48	.49
Twin 2	28.29 (2.94)	27.70 (2.89)		
Regard for Child				
Twin 1	19.57 (.87)	18.66 (2.11)	3.06	.08
Twin 2	19.67 (.66)	19.55 (.99)		
Structure				
Twin 1	17.19 (1.66)	16.52 (2.01)	1.42	.23
Twin 2	17.10 (1.61)	17.16 (1.69)		
Father Report:				
Conflict				
Twin 1	21.71 (6.44)	22.10 (6.16)	3.42	.07
Twin 2	22.67 (6.47)	21.45 (6.50)		
Parental Involvement				
Twin 1	36.67 (6.00)	38.51 (5.78)	2.97	.09
Twin 2	35.52 (6.82)	38.55 (5.79)		
Regard for Parent				
Twin 1	26.19 (3.08)	27.06 (3.09)	.00	.99
Twin 2	26.19 (3.60)	27.07 (3.53)		
Regard for Child				
Twin 1	18.71 (1.77)	19.22 (1.40)	1.93	.17
Twin 2	18.95 (1.43)	19.15 (1.46)		
Structure				
Twin 1	16.71 (2.17)	16.89 (1.92)	.12	.74
Twin 2	16.90 (1.67)	16.99 (2.06)		
Twin Report:				
Maternal Conflict				
Twin 1	23.78 (7.69)	22.41 (7.26)	.02	.90
Twin 2	24.30 (7.14)	23.13 (7.25)		

PEQ Scales	AN[a] n = 23	Control n = 270	F (df = 1)	P
Maternal Involvement				
Twin 1	37.65 (7.35)	39.46 (6.22)	3.45	.06
Twin 2	39.65 (5.88)	38.85 (14.72)		
Regard for Mother				
Twin 1	27.87 (4.07)	28.47 (3.50)	1.29	.26
Twin 2	28.39 (3.39)	28.11 (3.88)		
Mother's Regard for Child				
Twin 1	17.61 (2.71)	18.40 (2.20)	1.02	.31
Twin 2	18.35 (1.90)	18.61 (2.16)		
Maternal Structure				
Twin 1	16.87 (2.28)	16.61 (1.92)	.02	.89
Twin 2	16.74 (2.20)	16.55 (2.05)		
Paternal Conflict				
Twin 1	22.74 (7.67)	22.02 (6.84)	.01	.92
Twin 2	23.52 (6.47)	22.96 (7.39)		
Paternal Involvement				
Twin 1	34.26 (8.23)	35.34 (7.81)	2.15	.14
Twin 2	37.00 (6.82)	35.87 (7.71)		
Regard for Father				
Twin 1	27.00 (4.81)	27.23 (4.53)	1.26	.26
Twin 2	27.61 (3.45)	26.88 (4.88)		
Father's Regard for Child				
Twin 1	17.61 (2.69)	18.08 (2.64)	1.66	.20
Twin 2	18.57 (1.73)	18.27 (2.73)		
Paternal Structure				
Twin 1	16.39 (2.33)	16.20 (2.20)	.00	.99
Twin 2	16.48 (2.09)	16.28 (2.12)		

Note. PEQ = Parental Environment Questionnaire. AN = anorexia nervosa.

[a]For the group of AN women, Twin 1 represents the AN twin, while Twin 2 represents the co-twin without AN.

Table 5

Within-pair Differences in Parental Treatment in MZ Twin Pairs Among AN and Control Women.

PEQ Scales	AN Pairs[a] n = 11	Control Pairs n = 270	F (df = 1)	P
Mother Report:				
Conflict				
Twin 1	20.45 (4.91)	22.80 (7.29)	.66	.42
Twin 2	20.18 (5.69)	20.84 (6.09)		
Parental Involvement				
Twin 1	42.55 (2.54)	39.29 (6.39)	1.19	.28
Twin 2	43.82 (2.79)	42.52 (4.29)		
Regard for Parent				
Twin 1	28.55 (2.30)	28.24 (3.71)	.33	.57
Twin 2	28.64 (2.34)	27.71 (2.88)		
Regard for Child				
Twin 1	19.55 (.82)	18.66 (2.11)	1.63	.20
Twin 2	19.64 (.67)	19.55 (.99)		
Structure				
Twin 1	17.27 (1.56)	16.52 (2.01)	.55	.46
Twin 2	17.36 (1.80)	17.16 (1.69)		
Father Report:				
Conflict				
Twin 1	21.82 (6.45)	22.10 (6.16)	2.19	.14
Twin 2	22.91 (6.66)	21.45 (6.50)		
Parental Involvement				
Twin 1	35.73 (6.13)	38.51 (5.78)	.99	.32
Twin 2	34.91 (6.77)	38.55 (5.79)		
Regard for Parent				
Twin 1	26.27 (2.53)	27.06 (3.09)	.24	.62
Twin 2	26.55 (2.84)	27.07 (3.53)		
Regard for Child				
Twin 1	18.64 (1.50)	19.22 (1.40)	.32	.57
Twin 2	18.73 (1.56)	19.15 (1.46)		
Structure				
Twin 1	16.82 (1.99)	16.89 (1.92)	.00	.97
Twin 2	16.91 (1.70)	16.99 (2.06)		
Twin Report:				
Maternal Conflict				
Twin 1	21.55 (5.40)	22.41 (7.26)	.01	.93
Twin 2	22.45 (5.79)	23.13 (7.25)		

PEQ Scales	AN[a] n = 23	Control n = 270	F (df = 1)	P
Maternal Involvement				
Twin 1	37.36 (5.92)	39.46 (6.22)	1.26	.26
Twin 2	38.91 (6.66)	38.85 (6.70)		
Regard for Mother				
Twin 1	28.36 (4.25)	28.47 (3.50)	.85	.36
Twin 2	29.00 (3.69)	28.11 (3.88)		
Mother's Regard for Child				
Twin 1	18.00 (2.28)	18.40 (2.20)	.03	.86
Twin 2	18.09 (1.87)	18.61 (2.16)		
Maternal Structure				
Twin 1	17.09 (2.17)	16.61 (1.92)	.65	.42
Twin 2	16.45 (2.34)	16.55 (2.05)		
Paternal Conflict				
Twin 1	23.36 (8.99)	22.02 (6.84)	.75	.39
Twin 2	22.45 (6.30)	22.96 (7.39)		
Paternal Involvement				
Twin 1	33.91 (8.54)	35.34 (7.81)	1.30	.26
Twin 2	36.82 (7.52)	35.87 (7.71)		
Regard for Father				
Twin 1	27.27 (5.29)	27.23 (4.53)	2.31	.13
Twin 2	28.73 (3.72)	26.88 (4.88)		
Father's Regard for Child				
Twin 1	17.73 (2.76)	18.08 (2.64)	.10	.75
Twin 2	18.18 (1.72)	18.27 (2.73)		
Paternal Structure				
Twin 1	16.55 (2.30)	16.20 (2.20)	.06	.82
Twin 2	16.45 (2.34)	16.28 (2.12)		

Note. PEQ = Parental Environment Questionnaire. AN = anorexia nervosa.

[a]For the group of AN women, Twin 1 represents the AN twin, while Twin 2 represents the co-twin without AN.

Table 6

Correlations between M-EDI and PEQ Scales for Twin Relationships with their Parents

	M-EDI Scales				
PEQ Scales	BD	CB	BE	WP	Total
Mother Report:					
Conflict	.19**	.23**	.18**	.21**	.25**
Parental Involvement	-.15**	-.20**	-.13*	-.14*	-.18**
Regard for Parent	-.10	-.09	-.16**	-.07	-.11**
Regard for Child	-.02	-.03	-.01	-.02	-.01
Structure	-.01	-.02	-.04	-.04	-.04
Father Report:					
Conflict	.19**	.26**	.21**	.12**	.23**
Parental Involvement	-.18**	-.17**	-.15**	-.12*	-.18**
Regard for Parent	-.15**	-.06	-.11*	-.08	-.13*
Regard for Child	-.10	-.14*	-.06	.02	-.09
Structure	.06	.05	.06	.01	.02
Twin Report:					
Maternal Conflict	.19**	.20**	.21**	.25**	.27**
Maternal Involvement	-.14*	-.24**	-.16**	-.18**	-.21**
Regard for Mother	-.08	-.09	-.16**	-.08	-.11*
Mother's Regard for Child	-.22**	-.22**	-.25**	-.20**	-.26**

	M-EDI Scales				
PEQ Scales	BD	CB	BE	WP	Total
Maternal Structure	-.09	-.05	.03	-.04	-.03
Paternal Conflict	.25**	.23**	.24**	.25**	.31**
Paternal Involvement	-.19**	-.20**	-.18**	-.17**	-.23**
Regard for Father	-.15**	-.10	-.17**	-.10	-.16**
Father's Regard for Child	-.17**	-.22**	-.22**	-.17*	-.25**
Paternal Structure	-.10	-.05	.02	-.10	.06

Note. PEQ = Parental Environment Questionnaire. M-EDI = Body Dissatisfaction (BD), Compensatory Behavior (CB), Binge Eating (BE), Weight Preoccupation (WP), Total Score (Total). To adjust for nonindependence between twins, the number of pairs (n = 321) was used as the degrees of freedom for significance testing of the correlations (Kashy & Snyder, 1995).

* $p<.05$, ** $p<.01$

Table 7

Correlations between PEQ and M-EDI Difference Scores for MZ and DZ Twin Relationships with their Parents

	M-EDI Scales				
PEQ Scales	BD	CB	BE	WP	Total
Mother Report:					
Conflict	.04	.10	.02	.02	.05
Parental Involvement	-.02	-.15*	.02	.01	-.02
Regard for Parent	-.03	-.15*	.03	-.03	-.04
Regard for Child	.05	-.01	.06	.06	.06
Structure	.05	.01	.01	.03	.04
Father Report:					
Conflict	.08	.16*	.01	.03	.08
Parental Involvement	.02	-.08	.08	-.03	.02
Regard for Parent	.06	.00	.08	.04	.09
Regard for Child	.03	.06	.16*	.07	.10
Structure	-.04	-.01	.07	-.07	-.03
Twin Report:					
Maternal Conflict	.06	.04	.11	.17**	.15*
Maternal Involvement	-.06	-.13*	-.03	-.17**	-.14*
Regard for Mother	-.04	-.09	-.10	-.03	-.08
Mother's Regard for Child	-.14*	-.12	-.10	-.13*	-.17**

	M-EDI Scales				
PEQ Scales	BD	CB	BE	WP	Total
Maternal Structure	-.05	-.02	.05	.04	.02
Paternal Conflict	.11	.10	.15*	.17**	.19**
Paternal Involvement	-.12*	-.10	-.05	-.22**	-.20**
Regard for Father	-.11	-.06	-.09	-.07	-.11
Father's Regard for Child	-.13*	-.12*	-.14*	-.13*	-.18**
Paternal Structure	-.06	-.04	.05	.03	.01

Note. PEQ = Parental Environment Questionnaire. M-EDI = Body Dissatisfaction (BD), Compensatory Behavior (CB), Binge Eating (BE), Weight Preoccupation (WP), Total Score (Total).

* $p<.05$, ** $p<.01$

Table 8

Correlations between PEQ and M-EDI Difference Scores for MZ Twin Relationships with their Parents

	M-EDI Scales				
PEQ Scales	BD	CB	BE	WP	Total
Mother Report:					
Conflict	.06	.07	.14	.06	.13
Parental Involvement	.00	-.14	-.04	-.03	-.06
Regard for Parent	-.05	-.19*	-.03	-.03	-.08
Regard for Child	.15	.09	.00	.08	.10
Structure	-.01	-.01	-.03	.03	.01
Father Report:					
Conflict	.02	.21*	.03	-.03	.06
Parental Involvement	-.01	-.11	-.03	-.10	-.07
Regard for Parent	.06	-.02	.10	.07	.12
Regard for Child	-.02	.12	.10	.01	.04
Structure	-.09	.02	-.04	-.09	-.08
Twin Report:					
Maternal Conflict	.10	-.02	.12	.07	.12
Maternal Involvement	-.11	-.07	-.14	-.11	-.17**
Regard for Mother	-.02	-.08	-.12	.02	-.08
Mother's Regard for Child	-.14*	-.12	-.10	-.05	-.17**

	M-EDI Scales				
PEQ Scales	BD	CB	BE	WP	Total
Maternal Structure	-.06	-.02	.05	-.06	.03
Paternal Conflict	.12	-.03	.08	.08	.12
Paternal Involvement	-.15*	-.01	-.12	-.11	-.18*
Regard for Father	-.09	-.03	-.05	.02	-.04
Father's Regard for Child	-.12	.01	-.12	-.08	-.14
Paternal Structure	-.07	.01	.04	.06	.03

Note. PEQ = Parental Environment Questionnaire. M-EDI = Body Dissatisfaction (BD), Compensatory Behavior (CB), Binge Eating (BE), Weight Preoccupation (WP), Total Score (Total).

* $p<.05$


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