ELUCIDATING FACTORS UNDERLYING PARENT-OFFSPRING SIMILARITY IN EATING PATHOLOGY IN PRE- AND EARLY PUBERTY: EXPLORING THE POSSIBILITY OF PASSIVE GENE-ENVIRONMENT CORRELATION

By

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ABSTRACT

ELUCIDATING FACTORS UNDERLYING PARENT-OFFSPRING SIMILARITY IN EATING PATHOLOGY IN PRE- AND EARLY PUBERTY: EXPLORING THE POSSIBILITY OF PASSIVE GENE-ENVIRONMENT CORRELATION

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Objective: Eating pathology has been found to aggregate in families. Typically, familial resemblance has been attributed to parents providing an environment that leads to the development of eating pathology. However, offspring raised by biological parents receive both their environment and genes from their parents, raising the possibility that genetic influences, environmental influences, and/or gene-environment interplay may account for familial resemblance. Past studies have not explored the possibility of parents' genes influencing the environment they provide (i.e., passive gene-environment correlations or "passive rGE"). If present, passive rGE is most likely to "hide" in estimates of shared environmental influences in classical twin models. The current study used a nuclear twin family design to explore the possibility of passive rGE during pre-/early puberty when past studies have demonstrated the importance of shared environmental influence. Additionally, the present study explored whether sibling-specific (i.e. influences specific to the twin generation) or family-specific (i.e., "cultural" influences within the home) shared environmental influences accounted for shared environmental influences found in past studies. **Methods:** Participants included pre-/early pubertal twins and their biological parents from the Minnesota Twin Family Study and the Michigan State University Twin Registry. Disordered eating (i.e., overall disordered eating, body dissatisfaction, weight preoccupation, binge eating) was assessed with self-report measures in the twins and parents. Pubertal status was determined using an established cut-off on a self-report measure. **Results:** Passive rGE was not indicated in pre-/early puberty. Instead, sibling-specific (not family-specific) shared environmental and non-shared environmental influences were most influential. **Conclusions**: Future research should explore parental influences that may impact the twin generation only (e.g., parenting style, parents' comments about weight/shape to their offspring, etc.), as this would be represented by sibling-specific environmental influences.

This dissertation is dedicated to coffee and my partner in life, Jacob.
Both have provided unwavering support, even on the toughest writing days.

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INTRODUCTION

Parents are commonly identified as an important source of influence for the development of eating disorders (i.e., anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED)) (Hill & Franklin, 1998; Moreno & Thelen, 1993; Pike & Rodin, 1991; Smolak, Levine, & Schermer, 1999; Thelen & Cormier, 1995). Given the higher prevalence of eating disorders in females (lifetime prevalence male to female ratio for AN 1:3-1:12, BN 1:3-1:18, BED 1:2-1:6; Bulik et al., 2006; Hudson, Hiripi, Pope, & Kessler, 2007; Woodside et al., 2001), the vast majority of studies exploring parent influences focus on the impact of mothers on their daughters rather than exploring the impact of fathers on their daughters or the impact of either parent on their sons (e.g., Benedikt, Wertheim, & Love, 1998; Benninghoven, Tetsch, Kunzendorf, & Jantschek, 2007; Elfhag & Linne, 2005; Fulkerson et al., 2002; Hill & Franklin, 1998; Pike & Rodin, 1991). However, it is important to note that the small number of studies that have explored the potential role of fathers have indicated that fathers are also influential in daughters' disordered eating development. Specifically, fathers' comments about his daughter's weight and/or encouragement to diet have been linked to daughters' disordered eating (Keery, Boutelle, van den Berg, & Thompson, 2005; Meesters, Muris, Hoefnagels, & van Gemert, 2007; Neumark-Sztainer et al., 2010; Vincent & McCabe, 2000).

To date, studies have consistently demonstrated mother-daughter similarity in eating pathology, whereas father-daughter similarity in eating pathology has yet to be explored. Garcia de Amusquibar & De Simone (2003) found that mothers of daughters with eating disorders (i.e., threshold and subthreshold AN, BN, BED) endorsed using more purging behaviors throughout their lifetime (e.g., diet pills, homeopathic medicines, laxatives, diuretics, or self-induced vomiting; 32% vs. 13.3%) and reported more binge eating episodes in the past three months (22% vs. 13%) than mothers in the control group. Similar levels of body dissatisfaction were also found between patients with bulimia nervosa and their mothers (r = .47-.50, p=.02-.049; Benninghoven et al., 2007). Additionally, even infants and young daughters of mothers with eating disorders have been shown to have elevated rates of eating disturbances (e.g., difficulty with self-regulation of food intake, extreme food selectivity, and food refusal) (Park, Senior, & Stein, 2003).

Importantly, mother-daughter similarity extends to disordered eating symptoms in the general population as well. Cross-sectional studies have demonstrated significant small-to-moderate positive correlations between mother's and daughter's level of many forms of disordered eating, including body dissatisfaction (Keery, Eisenber, Boutelle, Neumark-Sztainer, & Story, 2006; McKinley, 1999; Usmiani & Daniluk, 1993), body shape concerns

(Kichler & Crowther, 2001), drive for thinness (Elfhag & Linne, 2005), emotional eating (Elfhag & Linne, 2005), uncontrollable eating (Elfhag & Linne, 2005), disinhibition (Cutting, Fisher, Grimm-Thomas, & Birch, 1999), and extreme weight loss strategies (Benedikt et al., 1998) (range r =.16-.41, average r =.24, all p<.05). Thus, mother-daughter similarity is observed not only in clinical samples, but also across the spectrum of eating pathology.

Unfortunately, the vast majority of studies exploring associations between mothers' and daughters' disordered eating are cross-sectional in nature. However, a prospective cohort study that followed over 12,500 children over 7 years (ages 9 to 15 at baseline) found that girls whose mothers had a history of eating disorders were 1.5 to 3 times more likely than their peers to begin purging at least once per week (odds ratio range: 1.5- 2.9, 95% CI=0.7-6.1; Field et al., 2008). A second longitudinal study found that adolescent (ages 11-15) reports of parental (not specified if mother or father) encouragement to diet predicted the adolescent's level of body dissatisfaction a year later (β= .20, $T(8,225)$= 2.87, p<.01) (Helfert & Warschburger, 2011).

Thus, previous research has demonstrated evidence of significant mother-daughter resemblance in eating pathology. Most often, studies have attributed this resemblance to environmental factors, such that mothers provide an environment that leads to higher rates of disordered eating in daughters (Pike & Rodin, 1991; Rodgers & Chabrol, 2009; Smolak et al., 1999; Stice, 1998; Wertheim, Mee, & Paxton, 1999). This environmental transmission is thought to occur through mothers demonstrating a strong emphasis of the importance of body weight, shape, or appearance within the home (Stice, 1998), commenting on their daughter's weight/shape (Hanna & Bond, 2006; Smolak et al., 1999), encouraging their daughter to diet (Neumark-Sztainer et al., 2010), or acting as models of various disordered eating attitudes and behaviors that the daughter observes and imitates (Cooley, Toray, Wang, & Valdez, 2008; Pike & Rodin, 1991).

However, offspring raised by biological parents receive both their environment and their genes from their parents, raising the possibility that the association between mothers' eating pathology and daughters' eating pathology may not be simply environmental in origin. Indeed, family studies, which compare rates of eating disorders in relatives of individuals with eating disorders to relatives of non-disordered eating disorder controls, demonstrate higher rates of eating disorders in **both** first- and second-degree female relatives of girls/women with eating disorders. For example, Strober & Humphrey (1987) demonstrated that 10% of first-degree female relatives (i.e., mothers and sisters) of individuals with AN had some type of eating disorder (i.e., AN, BN, or subclinical AN) compared to 1.4% of first-degree female relatives in control families (p<.001). Similarly, higher rates of eating

disorders were found in second-degree female relatives (i.e., aunts) of individuals with AN (9.5%) than in second-degree female relatives of controls (2.4%) (Strober & Humphrey, 1987).

Thus, familial resemblance in eating disorders extends to biological family members that do not reside in the home with the individual. This extended familial aggregation suggests that similarities in eating attitudes and behaviors may be due to genetic similarities between relatives rather than environmental factors they may experience in the home. Consistent with this idea, twin studies have shown significant genetic influences (i.e., heritability ≥ 50%) on AN, BN, BED, and their symptoms (e.g., binge eating, weight preoccupation, body dissatisfaction) in women (Bulik, Sullivan, & Kendler, 1998; Klump et al., 2010; Klump, Miller, Keel, McGue, & Iacono, 2001; Klump, McGue, & Iacono, 2000). Thus, genetic influences may also contribute to familial resemblance of eating pathology.

The presence of genetic effects means that genetic factors, environmental factors, or their interplay may contribute to familial resemblance. Specifically, prior theories may be accurate in assuming that familial resemblance acts through environmental main effects, such that similarities in eating pathology that have been observed between mothers and their daughters are due to mothers providing an environment or modeling disordered eating attitudes and behaviors that are then picked up and imitated by the daughter. Alternatively, familial resemblance may act through genetic main effects, such that similarities observed between mothers' and daughters' eating pathology are due to mothers passing along genetic vulnerabilities that lead the daughter to develop disordered eating similar to that of her mother. In this case, disordered eating would develop in daughters whose mother passed along genetic risk, regardless of whether the mother comments on her daughter's weight and shape or models disordered eating behaviors in their home.

Finally, familial resemblance may occur through different types of gene-environment interplay, including passive gene-environment correlations ("passive rGE"). Passive rGE occur when parents with genetic risk for eating pathology create an environment that is correlated with their genotype (e.g., an environment that places a higher emphasis on weight or physical appearance of their children, is characterized by comments/criticisms about weight and shape, etc.). In this case, genetic and environmental influences are correlated and it is the combination of genetic risk for disordered eating and the fact that the parents who pass along genetic risk are also creating the rearing environment that leads to the development of eating pathology. Conceptually, in this case, risk for disordered eating would be decreased in daughters whose parents discuss weight or shape, but do not pass along genetic risk.

Similarly, risk for disordered eating would be attenuated in daughters whose parents passed along genetic risk, but who do not comment on their daughter's weight/shape or model disordered eating attitudes/behaviors in their home. Although father-daughter resemblance in eating pathology has yet to be explored, it is also possible that fathers could influence their daughters through environmental main effects, genetic main effects, or passive rGE.

Unfortunately, previous studies have not controlled for these different possibilities. For instance, prior studies that have interpreted correlations between mothers' and daughters' level of eating pathology as indicative of maternal modeling (i.e., daughters observing and mimicking her mothers' disordered eating behavior) (Abraczinskas, Fisak, & Barnes, 2012; Pike & Rodin, 1991; Smolak et al., 1999; Stice, 1998; Vincent & McCabe, 2000; Wertheim et al., 1999). However, these correlations may be due to shared genetic risk between mothers and daughters (i.e., genetic main effects) or the parents' genetic risk influencing the environment they provide which ultimately leads to similarities in eating pathology between family members (i.e., passive rGE). Similarly, prior studies have examined the effects of mother and father comments about their daughter's weight and shape on daughter's level of disordered eating (Hanna & Bond, 2006; Smolak et al., 1999) without taking into account the possibility that parents who comment on their daughter's weight and shape may also be passing on genetic risk for disordered eating (i.e., genetic main effects and/or passive rGE). Conversely, while twin studies have demonstrated significant heritability of disordered eating symptoms (Klump et al., 2010; Klump et al., 2001; Klump et al., 2000), these studies do not explore whether parents' genetic risk could also influence the environment they provide (i.e., passive rGE).

Indeed, classical twin studies are typically used to examine additive genetic main effects (A; i.e., genetic influences that add across genes), shared environmental main effects (C; i.e., environmental influences that are shared by reared together twins and are thus a source of their behavioral similarity) or dominant genetic main effects (D; i.e., non-additive interactions between alleles at a single genetic loci), and non-shared environmental main effects (E; i.e., environmental influences that are not shared by reared-together twins and are thus a source of their behavioral dissimilarity, this also includes measurement error). However, due to the fact that parents are not included in the model, classical twin designs are unable to estimate passive rGE effects (see below for how passive rGE can be estimated). Within these twin models, if passive rGE is present, it can inflate estimates of shared environmental main effects (Neiderhiser et al., 2004). Thus, it is possible when a trait or disorder demonstrates

shared environmental influences using classical twin models, that passive rGE could partially or completely account for these effects.

Past studies utilizing the classical twin design have provided important insights into which main effects may be present during specific developmental time periods. Specifically, data from classical twin studies have demonstrated significant genetic effects (~50%) and essentially no shared environmental effects (~0%) for eating pathology in adult women (Bulik et al., 1998; Bulik et al., 2006; Bulik et al., 2010; Kendler et al., 1991; Reichborn-Kjennerud et al., 2003; Rutherford, McGuffin, Katz, & Murray, 1993; Wade, Bulik, Sullivan, Neale, & Kendler, 2000). Given that un-modelled passive rGE would likely inflate estimates of shared environmental influence, it is unlikely that passive rGE is present in adulthood. Notably however, consistently using both cross-sectional (Klump et al., 2010; Klump, McGue, & Iacono, 2003; Klump et al., 2000) and longitudinal (Klump, Burt, McGue, & Iacono, 2007; Wade et al., 2012) designs, classical twin studies have demonstrated substantial shared environmental effects (~50%) and nominal influence of genetic effects (~0%) in early adolescence. Further, when exploring shared environmental effects for eating pathology in 11-year-olds, results showed substantial shared environmental effects (~50%) and nominal genetic effects (~0%) in pre-/early pubertal 11-year-olds and conversely, nominal shared environmental effects (~0%) and substantial genetic effects (<50%) in mid-/late pubertal 11-year-olds (Klump, McGue, & Iacono, 2003; Klump, Perkins, Burt, McGue, & Iacono, 2007). Indeed, past findings suggest differences in etiological influences may be better accounted for by changes in puberty rather than age (Culbert et al., 2009; Klump et al., 2007; Klump et al., 2012; Klump et al., 2013; Klump et al., 2017). Thus, past studies utilizing the classical twin design suggest genetic main effects in late adolescence/puberty/adulthood and shared environmental main effects in pre-adolescence/ pre-puberty and early puberty (Culbert et al., 2009; Klump et al., 2003; Klump 2007; Klump et al., 2012; Klump et al., 2017). If passive rGE underlies familial resemblance, it is likely that it is present in pre-/early puberty when prior studies have indicated the importance of shared environmental effects.

Clearly, in order to develop a more comprehensive and accurate understanding of the influence of parents on disordered eating development, studies are needed that model genetic main effects, environmental main effects, and passive rGE to assess which influences contribute to the parental influence, particularly in pre-/early puberty when passive rGE may be present. Fortunately, there is a model that includes both twins and their biological parents called the Nuclear Twin Family Model (NTFM), which allows for the simultaneous modeling of genetic main effects, environmental main effects, and passive rGE. Additionally, due to the addition of parents in the model, the

NTFM is also able to develop a more nuanced understanding of shared environmental influences (i.e., "C" in classical twin models), as it is able to break these influences down into family-specific ("F"; i.e., environmental influences that make parents and siblings similar to one another) and sibling-specific ("S"; i.e., environmental influences that make siblings similar to one another, but not to their parents) shared environmental influences. This differentiation between different types of shared environmental effects can provide a better indication of whether shared environmental influences observed within classical twin studies are due to family-level or "cultural" influences within the home (i.e., family-specific ("F"); e.g., familial emphasis on the importance of thin body weight/shapes) or influences specific to the twin generation (i.e., sibling-specific ("S"); e.g., influences of environment at school).

Past studies have suggested that correlations between mothers' disordered eating and daughters' disordering eating may be due to maternal modeling of disordered eating attitudes and behaviors (Abraczinskas, Fisak, & Barnes, 2012; Pike & Rodin, 1991; Smolak et al., 1999; Stice, 1998; Vincent & McCabe, 2000; Wertheim et al., 1999). Maternal modeling would occur if mother's eating pathology was observed and imitated by the offspring. In this case, the demonstration of parents' eating pathology would create a culture within the home that would make family members similar in their eating pathology. This increased similarity in eating pathology would be represented by family-specific shared environmental influences ("F") within the NTFM.

Notably, parents could also contribute to sibling-specific shared environment ("S") if the environment created by parents only affected the twin generation. For instance, how the twins experience their parents' parenting style or expectations could contribute to sibling-level shared environment, as parents do not "parent" themselves and may not hold the same expectations for themselves as their children. Indeed, if twins experienced their parents as placing a strong emphasis on being high achieving or on the thin-ideal but parents do not experience this same pressure and are not high-achieving or high in disordered eating themselves, this would create similarities between siblings and not between all families members. These similarities between siblings would be reflected in estimates of sibling-specific rather than family-specific shared environmental factors. Parental comments about their offspring's weight/shape and comments encouraging their offspring to diet has been suggested as an important contributing factor in the development of disordered eating (Gross & Nelson, 2000; Hanna & Bond, 2006; Helfert & Warschbuger, 2011; Keery et al., 2005; Meesters, Muris, Hoefnagels, & van Germet, 2007; Neumark-Sztainer et al., 2010; Smolak et al., 1999; Vincent & McCabe, 2000). If weight/shape comments made to the offspring are not

reflective of the parents' own level of disordered eating, these comments would contribute to sibling-specific shared environment (if both twins received these comments from their parents) or to non-shared environment (if only one twin received these comments from their parents).

The NTFM model has never been used to examine parent influence on eating disorders, because it is rare for eating disorder researchers to collect data from fathers. Consequently, the current study used the NTFM to explore the etiological pathways underlying familial resemblance during pre-/early puberty when past studies using the classical twin design have indicated that presence of shared environmental influences. Specifically, the present study explored the potential role of passive rGE while also modeling genetic and environmental main effects, as well as explored whether the shared environmental influences demonstrated in classical twin studies were attributed to sibling-specific or family-specific environmental influences.

In order to explore the full range of disordered eating symptoms, etiologic effects were explored across multiple disordered eating symptoms (e.g., weight preoccupation, binge eating, body dissatisfaction, and overall disordered eating). All of these disordered eating symptoms have shown parent-child similarity in past work (Elfhag & Linne, 2005; Keery et al., 2006; Kichler & Crowther, 2001; McKinley, 1999; Usmiani & Daniluk, 1993) and demonstrate shared environmental influences in pre-/early puberty (Culbert et al., 2009; Klump et al., 2007; Klump et al., 2012; Klump et al., 2013; Klump et al., 2017). Notably, however, body dissatisfaction has less reliably demonstrated shared environment in pre-/early puberty, as two studies have demonstrated the presence of genetic influence rather than shared environmental influence in pre-/early puberty (Klump, 2000; O'Connor, In prep). Importantly, weight preoccupation, binge eating, and body dissatisfaction are transdiagnostic factors that cut across all eating disorders and are significant risk factors for the eventual development of clinical eating disorders (Killen et al., 1996; E. Stice & Shaw, 2002). Exploring the etiologic influences underlying these disordered eating symptoms may assist in increasing understanding of clinical eating disorders.

METHODS

Participants

The present study used samples of pre-/early pubertal female twins and their biological parents from two twin registries: the Minnesota Twin Family Study (MTFS; Iacono & McGue, 2002; Iacono et al., 2006) and the Michigan State University Twin Registry (MSUTR; Burt & Klump, 2013; K. L. Klump & Burt, 2006). Details about each sample are described below.

Sample 1: MTFS

The MTFS is a population-based, longitudinal study of same-sex adolescent twins and their parents. Recruitment procedures for the MTFS are detailed elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999), and thus will only be described briefly. Using Minnesota birth certificates, the MTFS recruited two cohorts of twins: one age 11 at intake and one age 17 at intake. Follow-up assessments were conducted every three to four years following the intake assessment. The present study used data from the intake for the 11-year-old cohort, as fathers did not participate in follow-up data collections and all twins in the 17-year-old cohort were pubertal. Specifically, the present study included 268 female twin families (63.1% MZ, 36.9% DZ) from the 11-year old cohort. The vast majority of families had both biological parents' data (80.2% of families) with the majority of the remaining families missing biological father data and only having biological mothers (17.9% of the families). Only 1.9% of families had biological father data and no biological mother data. Participant mean ages were as followed: twins: $M = 11.62$, $SD = 0.45$, mothers: $M = 40.09$, $SD = 4.67$, fathers: $M = 42.78$, $SD = 5.16$.

Recruitment procedures allowed for locating over 90% of the twins in the state of Minnesota at the time of intake (Iacono et al., 2006). Approximately 83% of families participated, with the remaining families either not interested or not meeting eligibility criteria. Specifically, twins were excluded if the family lived more than a day's drive from the MTFS laboratory, if the twins had either severe intellectual or physical disabilities, or if the twins were adopted. The sample is representative of the Minnesota population, with the majority of participants being White (over 95%) (Iacono et al., 2006).

Sample 2: MSUTR

The MSUTR was modeled after the MTFS and thus, recruitment procedures and study assessments are very similar across twin registries. A recent study (i.e., ***A Twin Study of Mood, Behavior, and Hormones during Puberty*** or **MBHP**) from the MSUTR collected data on disordered eating symptoms from 500 (51.4% MZ, 48.6%

DZ) adolescent twin pairs ages 8 to 15 (M = 12.33, *SD* = 3.12) and their parents. Families were recruited through another MSUTR study, the Michigan Twins Project (MTP). The MTP is a population-based registry of all twins born in Michigan ages 3-25 and 30-55. The MTP recruits twins through birth records in collaboration with the Michigan Department of Health and Human Services (MDHHS). Response rates for participation in the overall MTP is 57%, while response rates for the MBHP were even higher at 66%. Both response rates are on par or better than those of other twin registries using similar recruitment methods (Burt & Klump, 2013; Iacono & McGue, 2002). The recruited MBHP was also highly representative of the MTP sample and the general population of Michigan in terms of ethnic/racial distribution, with 4.4% of pairs identifying as Hispanic (77.8% White/Hispanic, 22.2% more than one race) and with those identifying as non-Hispanic, 81.6% identified as White, 9.1% as African American, 0.7% as Asian, 0.2% as American Indian or Alaska Native, and 8.4% as multiple races. Finally, although the MBHP had several inclusion/exclusion criteria (e.g., no steroid medication use) specific to the study aims (i.e., to examine ovarian hormone effects on disordered eating), the MBHP twins were not significantly different from the larger sample of MTP twins in terms of disordered eating symptoms (assessed using a composite score that includes items taping body dissatisfaction, binge eating, compensatory behavior, over evaluation of weight and shape) ($t(391) = -0.95$, $p = .35$) or BMI ($t(375) = -0.84$, $p = .40$).

Notably, only one parent was required to participate in the MBHP study and thus, only a minority of families had both parents participate (N= 171/499 families, 34.3%); most commonly these families included both biological parents (N=157/171 families, 91.8%); however, in a small number of cases a biological and step-parent or long-term partner participated (N=14/171 families; 8.1%). Lack of participation was usually due to divorce/separation of the biological parents or scheduling issues (i.e., difficulties finding a time for both parents to attend the assessment). Given that the NTFM requires both biological parents, I led a follow-up data collection to recruit biological parents whose data were missing from the initial sample for the present study (63.6%, n= 318 biological fathers; 4.8%, n=24 biological mothers).

Procedures for follow-up data collection were very similar to the original data collection with the exception that all questionnaires were completed in the home rather than at the MSUTR lab. Specifically, families with missing parent data were contacted and if interested, sent a questionnaire packet with consent, voucher, and return envelope to complete in their home and send back to the MSUTR. Of the families missing parent data, a small number of families had a biological parent that was deceased (n= 11 biological fathers, n=1 biological mothers) or

was no longer in contact with the family (n= 17 biological fathers, n= 2 biological mothers). On average, the follow-up data collection was conducted 3.90 years (SD = 1.45; range = 1.73 - 6.58) from the twins' initial data collection. The biological father response rate for this follow up data collection was 61.7% (179/290). Of the biological fathers who responded with interest in this follow-up data collection, 92.2% (165/179) completed the data collection. The biological mother response rate for this follow up data collection was 76.2% (16/21). Of the biological mothers who responded with interest, 75% (12/16) completed the data collection.

Notably, because the present study focused on families with pre-/early pubertal twins, the present study included a final sample of 279 pre-/early pubertal female twin families (50.5% MZ, 49.5% DZ) from the MSUTR. The majority of families had both biological parent data (66.7% of families) with the remaining families having biological mother data only (30.8% of families), or biological father data only (2.5% of families). Participant mean ages within the MSUTR sample used in this study are as followed: twins: M = 10.46, SD = 1.25, mothers of twins: M = 41.98, SD = 5.15, fathers of twins: M = 45.99, SD = 6.64. Notably, age of the individual at the time he/she completed the study was used (e.g., age of parent at initial data collection if participated at initial data collection and age of parent at follow-up data collection if participated in the follow-up).

However, in order to increase power, the MTFS and MSUTR samples were combined. Combining the MTFS and MSUTR samples resulted in a total of 547 twin families (56.7% MZ, 43.3% DZ), including 407 families with both biological parent data (74.4% of families), 130 families with biological mother data only (23.8% of families) and 10 families with biological father data only (1.8% of families). Notably, while differences may emerge between families with both parents participating and families with one parent participating (e.g., family income, racial/ethnic breakdown, etc.), there were no significant differences in disordered eating symptoms (MEBS total score: $t(1073) = -1.50$, $p = .14$, d=.10; WP: $t(1071) = -1.10$, $p = .27$, d=.08; BE: $t(1073) = -1.13$, $p = .26$, d=.08; BD: $t(522.96) = -1.63$, $p = .10$, d=.11) or mothers (MEBS total score: $t(198.65) = .01$, $p = .99$, d<.01; WP: $t(532) = 1.85$, $p = .06$, d=.18; BE: $t(531) = -1.15$, $p = .25$; d=.11; BD: $t(532) = -.99$, $p = .32$, d=.09). Given the small number of families with missing mothers (n=10), comparisons between fathers of families with both parents and fathers of families without mothers were not calculated. Participant mean ages for the MTFS +MSUTR combined dataset are as followed: twins: M = 11.03, SD = 1.11, mothers of twins: M = 41.07, SD = 5.01, fathers of twins: M = 44.25, SD = 6.09. Notably, the average father's age was higher in the MSUTR sample (e.g., M = 45.99, SD = 6.64) than the average father's age in the MTFS sample (e.g., M=42.78, SD = 5.16), likely due to using father's age at the time of

father's participation and the follow-up (mainly) father data collection occurring on average 3.90 years (SD = 1.45; range = 1.73 - 6.58) after the twins data collection. Finally, 91.6% of families within the combined dataset identified as White, 2.8% as Black/African American, .9% as Asian, and 4.7% as multiracial.

Zygosity Determination

The MTFS and the MSUTR both determine zygosity using parents' report on a physical similarity questionnaire (Lykken, Bouchard Jr, McGue, & Tellegen, 1989; Peeters, Gestel, Vlietinck, Derom, & Derom, 1998) that are reported to have 95% accuracy or better when compared to genotyping (Peeters et al., 1998). Additionally, the MTFS uses staff opinion through comparison of physical similarities of face shape, ear shape, eye color and hair color and an algorithm which incorporates cephalic index (e.g. ratio of head width to length), finger–print ridge counts, and the ponderal index (i.e., a measurement of leanness calculated as height in inches/ $\sqrt[3]{}$weight in pounds). If there were discrepancies in the coding of zygosity between the three methods, serological samples were taken to determine zygosity (Iacono et al., 1999). Similarly, the MSUTR compares multiple ratings (i.e., parents' report and two trained research assistants) on the physical similarity questionnaire (Lykken et al., 1989; Peeters et al., 1998). Any discrepancies in reports are resolved through review of questionnaire data and twin photographs by the principal investigator (KLK) or by examination of DNA markers (Klump & Burt, 2006).

Measures

Disordered Eating Symptoms

The *Minnesota Eating Behavior Survey* (MEBS*;* von Rason, Klump, Iacono, & McGue, 2005) was used to assess disordered eating in twins and their parents. The MEBS is a 30-item questionnaire made up of true/false questions that aims to assess a spectrum of eating pathology on a continuum of severity. This measure was developed for use with children as young as 10-years-old as well as adults. Factor analysis produced four factors: body dissatisfaction (i.e., assessing discontent with body size and shape), compensatory behaviors (i.e., assessing the use of, and thoughts of using, self-induced vomiting and other inappropriate compensatory behaviors to control weight), binge eating (i.e., assessing thinking about binge eating as well as engaging in binge eating and/or secretive eating) and weight preoccupation (i.e., assessing preoccupation with weight, eating, and dieting) (von Rason et al., 2005). Notably, prior research has indicated that this factor structure is invariant across males and females, as well as across pubertal development (Luo, Donnellan, Burt, & Klump, 2016).

The current study examined the body dissatisfaction, binge eating, and weight preoccupation subscales, as well as the MEBS total score. Overall, the MEBS shows good three-year stability (adolescents: 0.32-0.59, mothers: 0.68-0.80, father: not available) with the total score being the most stable followed by the scales measuring attitudes (weight preoccupation and body dissatisfaction) and then behavior (binge eating) (von Rason et al., 2005). Notably, given the significant changes that occur during adolescence, these estimates of stability are quite remarkable. The MEBS shows good convergent validity with the Eating Disorder Examination Questionnaire in adolescent girls (EDE-Q; Fairburn & Beglin, 1994). The strongest correlation was between the MEBS total score and the EDE-Q total score (r = 0.83), however high correlations (r = 0.74-0.78) were also found between other subscales as well, including MEBS weight preoccupation with EDEQ weight concern (r = 0.78) and MEBS body dissatisfaction with EDEQ shape concerns (r = 0.74) (von Rason et al., 2005). The MEBS also shows good criterion-related validity, as demonstrated by girls with eating disorders (i.e., either anorexia nervosa or bulimia nervosa) having significantly higher scores on the body dissatisfaction subscale, weight preoccupation subscale, and total score on the MEBS than controls (von Rason et al., 2005). Additionally, participants with bulimia nervosa had significantly higher scores on the binge eating subscale than controls (von Rason et al., 2005). Internal consistency for the total score, weight preoccupation subscale, body dissatisfaction subscale, and binge eating subscale in previous studies have ranged from alphas of 0.65-0.89 in females (von Rason et al., 2005) and 0.65-0.84 in men (Klump et al., 2012; von Rason et al., 2005). Consistent with prior studies, adequate internal consistency was demonstrated within the present study for total score, body dissatisfaction, weight preoccupation, and binge eating scales (all alphas >.65; see **Table 1**).

Item endorsement on the compensatory behaviors subscale was explored to see if the compensatory behaviors scale could be used in analyses. Previous work has only inconsistently been able to examine this scale in younger subjects, as some studies have found that item endorsement is too low in younger age groups (e.g., O'Connor, Burt, VanHuysse, & Klump, 2016). Alphas for the compensatory behavior subcale in the pre-/early pubertal twins in the combined MSUTR and MTFS sample was .52. Additionally, alphas for both mothers and fathers were low in the combined MSUTR and MTFS sample (mother: α=.52, father: α=.44). Given the lack of internal consistency within the compensatory behaviors subscale, the current study conducted analyses using the MEBS total score, body dissatisfaction, weight preoccupation, and binge eating.

Pubertal Development

The *Pubertal Development Scale* (PDS; Petersen, Crockett, Richards, & Boxer, 1988) is a self-report questionnaire used by both twin registries to assess pubertal development. The PDS asks the participant to assess their pubertal development based on the physical signs of puberty (i.e., height spurts, body hair growth, skin changes, breast development, onset of menarche). Participants rated the development of these physical markers on a 4-point scale: (1) development has not yet begun; (2) development has barely started; (3) development is definitely underway; and (4) development seems completed. An exception to this 4-point scale is the coding for menses, which is coded dichotomously. The ratings of each physical marker were summed and averaged to obtain an overall PDS score, with higher score representing more advanced pubertal status. The PDS exhibits good psychometric properties and the PDS total score correlates highly (r = .61-.67) with physician ratings of pubertal development (Petersen et al., 1988). Due to the young age range in the MSUTR sample, maternal reports on the PDS were used for a subset of younger twins (n=16, 1% of sample) who were missing PDS scores due to marking they did not know if they had started their period.

Given the present study focused on exploring etiologic effects in pre-/early puberty, twin pairs used in these analyses were required to be concordant on pre-/early pubertal status (i.e., both co-twins must be in pre-/early puberty). The NTFM is unable to include both concordant and discordant twin pairs, as the addition of a moderator (e.g., pubertal status) would require even larger samples sizes due to the need for additional power. However, past developmental twin studies that have used twin models that can include both concordant and discordant pairs (e.g., moderation models, see Purcell 2002; Klump, Perkins, Burt, McGue, & Iacono, 2007) found very similar results to those that could only include concordant twin pairs (Culbert et al., 2009; Klump et al., 2003; Klump et al., 2012). Pre-/early pubertal status group was determined using a dichotomized PDS score (i.e., < 2.5 = pre-/early pubertal) that has been used in numerous previous developmental twin studies examining differences in etiologic effects in pre-/early puberty compared to mid-puberty (Culbert et al., 2009; K. L. Klump et al., 2003; K. L. Klump, Perkins, et al., 2007). Within the present study, only a small number of pairs (n = 60/607; 9.9% of families with pre-/early pubertal twins) were discordant for pubertal status, and there were no significant differences in disordered eating between concordant and discordant twin pairs (MEBS total score: $t(678) = .76$, $p = .45$; WP: $t(675) = 1.19$, $p = .23$; BE: $t(679) = -.94$, $p = .35$; BD: $t(152.86) = 1.23$, $p = .22$). Therefore, findings are unlikely to be significantly impacted by the exclusion of discordant pairs.

Body Mass Index

Body Mass Index (BMI) was calculated (weight in kg/ height in m^2) using height and weight measured by a trained research assistant at their assessment in both the MSUTR and MTFS samples.

Statistical Analyses

All analyses described below were run separately for each disordered eating symptom (i.e., MEBS total score, binge eating, weight preoccupation, body dissatisfaction). Disordered eating scores with skewness or kurtosis greater than 1.00 (i.e., all scores except mother total score, twin weight preoccupation, and mother weight preoccupation) were log-transformed to better approximate normality. Importantly, prior studies have demonstrated an association between BMI and disordered eating (Jones, Bennett, Olmsted, Lawson, & Rodin, 2001, Keel, Fulkerson, & Leon, 1997). Thus, prior to analyses, BMI was regressed out of the parents' and twins' MEBS subscale scores in order to ensure results highlighted etiological effects underlying disordered eating, rather than BMI. Additionally, age was regressed out of the twins' disordered eating scores to control for potential age effects given the age range of the twin sample was 8 to 14 years old[1].

Pearson correlations were used to examine initial phenotypic associations between mothers-daughters, fathers-daughters, and mothers-fathers in disordered eating symptoms (i.e., overall disordered eating, weight preoccupation, binge eating, body dissatisfaction). Significant correlations between parents and twins' disordered eating scores could be reflective of the presence of additive genetic influence (A) and/or familial environmental influence (F), but not sibling-specific shared environment (S) given that S reflects similarity between co-twins not between all family members.

Twin intraclass correlations (e.g., correlation between the MEBS total score of Twin 1 and the MEBS total score of Twin 2) were then calculated for each disordered eating symptom. Intraclass correlations calculate the similarity between co-twins separately in MZ and DZ twin pairs to provide an initial indication of genetic and environmental influences. Monozygotic (MZ) twins share 100% of their segregating genes with their co-twin, whereas dizygotic (DZ) twins share approximately 50%. Utilizing the genetic relatedness between twins, additive genetic influences (A) are suggested if MZ twin correlations are approximately two times greater than DZ twin

[1] To assess the impact of regressing age and BMI out of disordered eating scores, NTFMs were run without regressing out age and BMI. Best fitting models were identical (Supplemental Table 1) and parameter estimates similar (Supplemental Table 2) in NTFM models that did not regress out age and BMI as the models with these variables regressed out.

correlations. Dominant genetic influences (D: non-additive interactions between alleles at a single genetic loci) are suggested if MZ twin correlations are more than two times greater than DZ correlations. Shared environmental effects (C) are suggested if MZ and DZ twin correlations are similar. Twin correlations are unable to pull apart family-specific (F) and sibling-specific (S) shared environmental influences due to the fact that both types of shared environmental influences would make siblings similar to each other, thus contribute to C when parent similarity to their offspring is not taken into account. Importantly however, the combination of twin correlations and parent-offspring correlations can provide initial indication of the influence of A, F, S, and E. Non-shared environmental influences (E) are suggested if MZ twin correlation is less than 1.00. Importantly, non-shared environmental influences also include measurement error.

The etiologic structure underlying familial resemblance in eating pathology was examined by fitting NTFMs using the structural-equation modeling program, Mx (Neale, Mazzeo, & Bulik, 2003). Full-Information Maximum-Likelihood (FIML) raw data techniques were used in all models to account for any missing data. FIML allows for less biased estimates than pairwise or listwise deletion, if missing data is present (Little & Rubin, 1987). FIML assumes that any missing data is missing at random (i.e., the probability the data is missing is unrelated to the value). Importantly, the use of FIML allows for the retention of families in which one or more family members has missing data.

The NTFM is shown in Figure 1. This model uses information from the parents and twins to partition the variance within each disordered eating symptom into five components: additive genetic (A), dominant genetic (D), sibling-level shared environment (S), family-level shared environment (F) and non-shared environment (E). Thus, genetic main effects are indicated by A and D, whereas shared environmental main effects are indexed by S and F, and nonshared environmental main effects are indexed by E. Passive rGE (see "w" in the Figure 1) is estimated by calculating the covariance between the parents' A and F, as the covariance between A and F reflects the extent to which the family environment is correlated with the parents' genes. The NTFM also allows for modeling of assortative mating (i.e., spousal similarity on a trait; μ path in Figure 1). Assortative mating is included in the model to ensure that similarities in parents do not increase the proportion of genes shared by DZ twins (not MZ, since MZ twins are already genetically identical), which would inflate estimates of C.

Importantly, due to the fact there is not enough information in the data to simultaneously estimate all 5 parameters (i.e., A, D, S, F, E), one parameter must always be fixed to zero in these models. Moreover, one

assumption of the NTFM is that A and E influence all traits to some extent; thus, A and E are always estimated within the model and D, S, F are differentially fixed to zero. Thus, the full models that can be compared are the ADSE, ADFE, and ASFE. Further, nested submodels of these full models can be fit to the data, including ADE, AFE, ASE, and AE.

The series of nested models, including the full models (ADSE, ADFE, ASFE) and submodels (ADE, ASE, AFE, AE) were fit to each disordered eating symptom. Model fit statistics were used to compare these full and sub-models to determine the best fitting model for each disordered eating symptom. Four information criteria indices that balance overall fit with model parsimony were used to determine fit: the Akaike's Information Criterion (AIC; Akaike, 1987), the Basyesian Information Criterion (BIC; Raftery, 1995), the sample-size adjusted Bayesian Information Criterion (SABIC; Sclove, 1987), and the Deviance Information Criterion (DIC; Spiegelhalter, Best, Carlin, & Van Der Linde, 2002). For all four indices, the lowest most negative values indicate the best fitting model. Additionally, comparisons of fit between different models were made by taking the difference in minus twice the log-likelihood (-2lnL) (for nested models). Large (statistically significant) differences in -2lnL values led to a rejection of the nested model in favor of the full model. Due to the fact that each these indices place different values on parsimony and overall fit, the fit indices may not agree. In this case, the best fitting model was indicated best fit for the most indices (as seen in Burt, Larsson, Lichtenstein, & Klump, 2012 and Hicks, South, DiRago, Iacono, & McGue, 2009). [2]

As mentioned above, the current study utilized two twin family samples: one from the MTFS and the other from the MSUTR. Given prior studies have combined samples from the MSUTR and MTFS (e.g., Klump et al., 2010; Suisman, Burt, McGue, Iacono, & Klump, 2011), it was expected that these samples are similar enough to combine for analyses. However, prior to combining samples from the MSUTR and MTFS, differences in means/variances and etiologic effects were compared across the two samples. Due to the large sample size, multiple independent samples t-test indicated significant phenotypic differences between samples (see **Table 1)**. However, effects sizes indicated only small mean differences between the MSUTR and MTFS samples (i.e., *d's* <.30; see **Table 1**) for all scales except for mothers' weight preoccupation scores that showed a moderate difference (*d* =.48).

[2]Due to the fact divorced/separated parents may not share their environment with their twins or may only share their environment with their children part time, NTFM models were run excluding divorced/separated families and anyone missing parental marriage status. Overall, there were no differences in the best-fitting models (Supplemental Tables 3) or the parameter estimates (Supplemental Table 4) in the samples excluding divorced parents and samples including divorced parents.

Fortunately, twin model-fitting analyses confirmed no significant differences in the etiologic structure of the MEBS scales across the two samples. These analyses compared the fit of the fully unconstrained models (i.e., models that allow parameters to vary in the MSUTR and MTFS samples) to the fully constrained models (i.e., models that force all parameters to be equal across the MSUTR and MTFS sample) for each of the base models (i.e., ADSE, ADFE, AFSE). In all cases, the majority (3 or more out of 5) of fit indices showed that the fully constrained model did not significantly worsen fit,, suggesting no significant differences in etiologic influences on MEBS scale scores between the MSUTR and MTFS samples (see **Table 2**). Weight preoccupation was the only scale the exhibited some variability between fit indices. Specifically, for two of the Weight Preoccupation base models (e.g., ADSE and AFSE), there was a significant change in chi-square, suggesting that parameter fit worsened when parameters were constrained across samples. However, the BIC, SABIC, and DIC indicated that the fully constrained model did not significantly worsen fitting. To ensure that the samples could be combined for this subscale, the NTFMs were run separately in the MSUTR and MTFS samples, and results were nearly identical (see Supplemental Tables 5 and 6). Thus, the combined MSUTR+MTFS sample was used for all analyses.

RESULTS

Twin, mother, and father means, standard deviations, and score ranges are presented in **Table 1**. Mothers' scores were larger than both twins' and fathers' mean scores (twins: *d*s= .35-1.41; fathers: *d*s=.25-.98). Fathers' endorsed more body dissatisfaction and binge eating than twins (BD: *d*=.45; BE: *d*=.11). Twins endorsed more weight preoccupation than fathers (*d*=.26).

In general, Pearson correlations demonstrated significant mother-daughter similarity with smaller father-daughter associations (see **Table 3**). Specifically, small, significant mother-daughter correlations were observed for total score, body dissatisfaction, and weight preoccupation (*r*=.13-.16, *p*<.001*)*. No significant mother-daughter association was demonstrated for binge eating (*r*=.06, *p*>.05). Small, significant father-daughter correlations were found for total score, body dissatisfaction, and binge eating (*r*=.08-.10, *p*<.05). No significant father-daughter association was demonstrated for weight preoccupation (*r*=.06, *p*>.05).

Intraclass correlations (i.e., correlations between co-twins calculated separately in MZ and DZ twin pairs) indicated significant shared environmental influences for total score, weight preoccupation, and binge eating, as MZ and DZ correlations were similar in magnitude (see **Table 3**). Notably, given parents' scores are not incorporated, intraclass correlations are unable to determine whether shared environmental influences are due to sibling-specific or family-specific influences. Significant additive genetic and nonshared environmental influences were suggested for body dissatisfaction, as MZ correlations were approximately twice as large as the DZ correlations and less than 1.0. Shared environmental influences were not suggested for body dissatisfaction, as there was a significant difference between MZ and DZ correlations (see **Table 3**).

Model fit statistics for each series of nested nuclear twin family models are reported in **Table 4**. Unstandardized and standardized parameter estimates from the best fitting models are provided in **Table 5.** In cases where the best-fitting model did not include F, the base model with the parameters from the best fitting model and F were provided (e.g., if ASE was best fitting, AFSE was also provided; see **Table 5**) due to the present study's specific interest in passive rGE and the need for F to calculate passive rGE.

For all of the disordered eating symptoms except body dissatisfaction, family-level influences (i.e., F) were not included in the best fitting models and thus, passive rGE was not indicated. Additionally, base models including F demonstrate non-significant parameter estimates for F and non-significant estimates of passive rGE (see Table 5), confirming they can be excluded from the model. Instead, results demonstrated the importance of sibling-level

shared environment and non-shared environmental influence and, to a lesser extent, additive genetic influences. Specifically, the ASE model was best fitting for total score, weight preoccupation, and binge eating as demonstrated by the lowest AIC, BIC, SABIC, and DIC values and non-significant changes in chi-square. Variance in these disordered eating symptoms was primarily attributed to environmental influences, specifically sibling-level shared environment (total score: 32.1%, weight preoccupation: 28.9%, binge eating: 21.5%) and non-shared environmental influences (total score: 54.1%, weight preoccupation: 61.4%, binge eating: 67.4%). Variance attributable to additive genetic influences was much lower (< 14%) for all three symptoms, and was non-significant for weight preoccupation.

For body dissatisfaction, the nuclear twin family model indicated quite different etiologic effects compared to the other disordered eating symptoms. Specifically, results indicated the presence of family-level shared environmental influences and a small amount of passive rGE. The AFE model was best-fitting with the lowest AIC, BIC, SABIC, DIC values, and a non-significant change in chi-square. The majority of variance in body dissatisfaction was attributed to additive genetic influence (45.0%) and non-shared environmental influences (61.9%). However, a significant but small proportion of variance was attributed to family-level shared environmental influences (4.6%). The presence of family-specific shared environment allowed for the calculation of passive rGE. The magnitude of the variance attributed to passive rGE was small, but significant (unstandardized estimate: -.09).

Importantly, assortative mating was explored to assess whether shared environmental influences may be inflated. However, estimates of assortative mating were small and non-significant for MEBS total score, binge eating, and body dissatisfaction (parameter estimates = .01-.09). Weight preoccupation exhibited a significant estimate of assortative mating, but the effect was small in magnitude (parameter estimate=.11; 95% CI=.01-.21). Thus, it is unlikely that assortative mating is inflating estimates of shared environment influences.

Additionally, analyses were conducted to ensure that the inclusion of the follow-up data collection of missing parents in the MSUTR sample did not decrease family member similarity (and thus, decrease F) due to data collection occurring at different times. NTFMs were run removing parents who did not complete the data collection at the same time as their family. Best fitting models were identical and parameter estimates were similar as compared to the full dataset that included parents from the follow up data collection (See Supplemental Table 7 and Supplemental Table 8). Thus, it does not appear that the follow-up data collection decreased estimates of F.

DISCUSSION

The present study is the first to explore the possibility of parents' genes influencing the home environment during a period when past studies have indicated the importance of environmental influences. Specifically, the present study explored the possibility of passive rGE during pre-/early puberty, as well as decomposed shared environmental influences demonstrated in past studies into family-specific or sibling-specific shared environment. Overall, the nuclear twin family models did not suggest the importance of passive rGE or family-specific shared environmental influences in pre-/early puberty. Instead, sibling-specific shared environmental influences and non-shared environmental influences were important.

Sibling-specific environmental influences are any factors that create similarities between twins, but not their parents. These influences could be related to any environmental factor that the co-twins share. Specific factors could be the importance of the thin-ideal within the twins' school or neighborhood (Thompson & Stice, 2001), shared experience in a weight-focused sport (Kong & Harris, 2015), or consumption of similar media emphasizing the thin ideal (Harrison, 2000). Indeed, the shared experience of these environmental risk factors by the twins could increase co-twin similarity, independent of the influence of the parents. Additionally, as stated previously, the importance of sibling-specific shared environment does not rule out the possibility that parents may influence their offspring's disordered eating. Indeed, twins' *experience* of their parents' attitudes and expectations about their body weight/shape/eating habits could influence the twins. Numerous studies have suggested that parental comments about offspring's weight or shape or encouragement to diet is associated with disordered eating in the offspring (Gross & Nelson, 2000; Hanna & Bond, 2006; Helfert & Warschbuger, 2011; Keery et al., 2005; Meesters, Muris, Hoefnagels, & van Germet, 2007; Neumark-Sztainer et al., 2010; Smolak et al., 1999; Vincent & McCabe, 2000). Interestingly, the majority of these past studies used the offspring's self-report of their parents' comments about the offspring's weight/shape or encouragement of the offspring to diet (Gross & Nelson, 2000; Hanna & Bond, 2006; Helfert & Warschbuger, 2011; Keery et al., 2005; Meesters, Muris, Hoefnagels, & van Germet, 2007; Neumark-Sztainer et al., 2010; Vincent & McCabe, 2000), which may more accurately reflect the offspring's perception of their parents' attitudes and expectations about their weight and shape, rather than the parents' own disordered eating attitudes and behaviors. Indeed, only a minority of studies explored the association between parents' self-reported comments about their offspring's weight/shape (Baker et al., 1999; Smolak et al., 1999). One such study explored both parental-reported and offspring-reported parental criticism of eating and appearance. Stronger correlations were

demonstrated between offspring- perceived parental criticism and the offspring's disordered eating than both mothers' and fathers' self-report of their comments about their offspring's eating or appearance and their offspring's disordered eating (Baker et al. 1999). Thus, a gap in perception of the importance of weight/shape within the home environment between parents and offspring (i.e., offspring perceiving their parents as valuing thinner body weights/shapes) may contribute to sibling-specific, rather than family-specific, shared environmental influences. Clinically, it may be important for parents of offspring with eating disorders to understand that offspring may perceive their home environment and/or comments from their parents as more critical than their parents.

Another possible explanation for the presence of sibling-specific rather than family specific shared environment influences could be that the phenotype used in the present study is too narrowly defined to capture the parent-offspring resemblance. Indeed, given the small, and in some cases non-significant, correlations between daughters' disordered eating and their parents' disordered eating, the lack of family-specific environmental influence or additive genetic influence within the NTFM was somewhat predicted. It is possible that parents' influence, either environmentally or genetically, on daughters' disordered eating could be better reflected by a different phenotype of the parents. This may be particularly true for fathers given disordered eating generally has lower endorsement by men than women. For instance, perfectionism and obsessive compulsive traits have been significantly linked to eating pathology both phenotypically (e.g., Wade, O'Shea, & Shafran, 2016; Bardone-Cone et al., 2007; Brecelj Aderluch, Tchanturia, Rabe-Hesketh, & Treasure, 2003) and etiologically (Hsu, Kaye, & Weltzin, 1993; Wade & Builk, 2007). Perhaps perfectionistic parents or parents' with obsessive compulsive traits contribute genes or an environment that lead to development of disordered eating in their daughters'. In this case, parents' self-reported disordered eating attitudes and behaviors may not specifically correlate with their daughters disordered eating; however, parents' perfectionism or obsessive compulsive traits may contribute to a home environment with overall high standards that then is interpreted by the daughter to perfectionism around her body or weight. Future studies should explore whether familial resemblance in broader traits may demonstrate more resemblance between parents and their offspring.

Notably, body dissatisfaction seems to exhibit slightly different etiological influences than total score, weight preoccupation, and binge eating. Additive genetic influences and non-shared environmental influences explained the majority of the variance in body dissatisfaction. This finding is consistent with past studies that have as demonstrated that genetic influence increases earlier in pubertal development for body dissatisfaction than other

disordered eating symptoms (Klump, McGue, & Iacono, 2000; O'Connor, Culbert, Burt, & Klump, In prep). Body changes begin in early puberty, which may explain the initial increase in additive genetic influence in body dissatisfaction relatively to binge eating, weight preoccupation, and overall disordered eating which may be more linked to the heavy influx of ovarian hormones at mid-puberty (Klump, 2013). More importantly for the current study, body dissatisfaction was the only disordered eating symptom that demonstrated the presence of passive rGE and family-specific shared environment. These influences were small in magnitude, but it is it possible that body dissatisfaction is modeled more within a family culture as oppose to weight preoccupation or binge eating. Indeed, researchers have demonstrated that body dissatisfaction is so prevalent among women that it is normative (e.g., Mazzeo, 1999; Striegel-Moore, Silberstein, & Rodin, 1986). Additionally, the observable behavior of fat-talk (i.e., negative body-related conversations) has been shown to predict body dissatisfaction within the individual (Warren, Holland, Billings, & Paker, 2012). It is possible that given the prevalence and acceptability of body dissatisfaction within the boarder culture (Fallon, Harris, & Johnson, 2014) and the link between body dissatisfaction and "fat talk" (Shape, Naumann, Treasure, & Schmidt, 2013), body dissatisfaction (rather than weight preoccupation or binge eating) is modeled more within the home. Another possibility is that parents' are not modeling body dissatisfaction within the home; instead the similarity between family members is due to exposure to the same broader culture in which body dissatisfaction is quite prevalent (Fallon, Harris, & Johnson, 2014).

While the present study significantly advances our understanding of parental transmission of eating pathology by exploring the possible influence of passive rGE, family-specific and sibling-specific shared environment, it is notable that there is a fourth possible source of influence that this study did not account for: gene-environment interactions. Gene-environment interactions (i.e., GxE) occur when environmental influences activate genetic risk (Moffitt, Caspi, & Rutter, 2005; Purcell, 2002). Indeed, it is possible that parents could provide an environment that activates that genetic risk in their offspring. The key difference between GxE and passive rGE is that with GxE, the genetic risk for disordered eating is not leading to the environment provided by the parents – the genes for disordered eating and the environment provided are independent. For instance, if parental comments about offspring weight and shape is <u>not</u> a result of parents' genetic risk for disordered eating **<u>and</u>** this environmental influence activates genetic risk in the offspring, disordered eating would have developed due to GxE processes. It is also possible that both rGE and GxE are present in the etiology of a disorder, as genes and environment may correlate **and** genetic risk may make an individual more susceptible to environmental risk that is not associated with

their genes. It is possible that then both these processes lead to development of disordered eating. Unfortunately, the nuclear twin family model is not able to model potential GxE effects, as GxE requires twin reports of parents' disordered eating or parental influence (e.g., parental encouragement to diet or comments about daughters' weight/shape). Future studies should explore this possibility.

Despite the many strengths of this study (i.e., the examination of multiple disordered eating symptoms, exploration of passive rGE and different types of shared environmental influences, etc.), this study was not without limitations. One such limitation is since this study was conducted in a non-clinical sample, it is unknown if the findings generalize to a clinical population. However, the disordered eating symptoms that were examined are precursors to full clinical eating disorders (Killen et al., 1996; Stice & Shaw, 2002) and recent data suggest that disordered eating exists on a continuum with clinical eating disorders (Luo et al., 2016). Additionally, the heritabilities of disordered eating attitudes and behaviors and clinical eating disorders are similar in adulthood (~50%; Bulik, Sullivan, Tozzi, Furberg, Lichtenstein, & Pedersen, 2006; Bulik et al. 2010; Culbert et al. 2009; Kendler et al., 1991; Klump, Burt, McGue, & Iacono, 2007; Klump, McGue, & Iacono, 2000; Klump, McGue, & Iacono, 2003; Klump, Miller, Keel, McGue & Iacono, 2001; Klump, Suisman, Burt, McGue, & Iacono, 2009; Rutherford, McGuffin, Katz & Murray, 1993; Slof-Op't Landt et al., 2008; Wade, Bulik, Neale, & Kendler, 2000;), suggesting it is likely that similar results might be found in a clinical sample. One challenge in conducting the present study using a clinical sample would be finding a sufficient number of twins with clinical eating disorders in pre-/early puberty to conduct a well-powered twin analysis. Nonetheless, future research is needed to assess whether findings translate to individuals with clinical eating disorders.

In conclusion, the present study advanced our understanding of the influences underlying parent-offspring resemblance in pre-/early puberty by being the first to explore the possibility of passive rGE and decomposing shared environmental influences found in previous studies. The present study's findings suggest the importance of environmental factors specifically shared by co-twins, which may include the twins' experience or interpretation of their parents' expectations. Future research should work to identify sibling-specific environmental factors driving disordered eating development. Advancing our understanding of specific environmental influences important in pre-/early puberty may assist in pinpointing important early targets for eating disorder prevention and intervention efforts.

APPENDICES

APPENDIX A

Tables

Table 1. Mean, Standard Deviation, Range, and Alphas for Minnesota Eating Behavior Survey Subscale Score Separately and Combined MTFS and MSUTR Samples (Combined Sample N= 547 twin families, MSUTR n=279 twin families, MTFS n=268 twin families)

		Combined Sample M (SD) Range	MSUTR M (SD) Range	MTFS M (SD) Range	Difference between MSUTR and MTFS	d
Total Score	Twins	4.56 (4.46) 0-25 α =.85	3.89 (4.09) 0-25	5.26 (4.73) 0-22	***t*(1032.10)=-5.07 *p*<.01**	.23
	Mom	8.49 (5.58) 0-24 α =.88	9.40 (5.46) 0-23	7.56 (5.57) 0-24	***t*(535)=3.85, *p*<.01**	.25
	Dad	4.56 (4.41) 0-24 α =.86	4.73 (4.36) 0-20	4.41 (4.45) 0-24	*t*(415)=.74, *p*=.46	.05
WP	Twins	1.97 (2.13) 0-8 α =.79	1.58 (1.90) 0-8	2.38 (2.27) 0-8	***t*(1020.89)= -6.22 *p*<.01**	.27
	Mom	2.91 (2.35) 0-8 α =.79	3.51 (2.29) 0-8	2.29 (2.24) 0-8	***t*(534)=6.22, *p*<.01**	.48
	Dad	1.46 (1.71) 0-8 α =.71	1.62 (1.70) 0-8	1.32 (1.72) 0-8	*t*(414)=1.81, *p*=.07	.17
BE	Twins	.92 (1.31) 0-7 α =.65	.84 (1.26) 0-7	1.00 (1.35) 0-7	***t*(1073)= -2.12 *p*=.03**	.09
	Mom	1.47 (1.77) 0-7 α =.77	1.68 (1.83) 0-7	1.26 (1.69) 0-7	***t*(533)=2.76, *p*=.01**	.20
	Dad	1.07 (1.38) 0-7 α =.69	1.07 (1.36) 0-7	1.07 (1.40) 0-7	*t*(414)=.01, *p*=.99	.10
BD	Twins	.86 (1.41) 0-6 α=.77	.68 (1.16) 0-6	1.05 (1.62) 0-6	***t*(948.67)= -4.27 *p*<.01**	.09
	Mom	3.49 (2.22) 0-6 α =.85	3.54 (2.19) 0-6	3.44 (2.25) 0-6	*t*(534)=.51, *p*=.61	.06
	Dad	1.56 (1.70) 0-6 α =.77	1.56 (1.71) 0-6	1.55 (1.69) 0-6	*t*(415)=.07, *p*=.95	.15

Note: Total Score= Minnesota Eating Behavior Survey Total Score; BD = Body Dissatisfaction; WP = Weight Preoccupation; BE = Binge Eating; BMI= Body Mass Index; M= mean; SD = standard deviation, d= Cohen's d. Independent sample t-test and Cohen's d were used to explore differences between the two samples.

Table 2. Constraint Models Constraining MTFS and MSUTR Parameter Estimates for all Full Nuclear Twin Family Models (N=547 twin families)

Sx	Model		-2lnL	df	**Δ** -2lnL (df)	*p*	AIC	BIC	SABIC	DIC
Total Score	ADSE	full uncon	5585.48	1999			1587.48	-3508.56	-335.74	-1671.60
		full con	**5588.26**	**2005**	**2.78 (6)**	**0.84**	**1578.26**	**-3526.08**	**-343.74**	**-1683.61**
	ADFE	full uncon	5600.58	1999			1602.58	-3501.01	-328.19	-1664.05
		full con	**5603.39**	**2005**	**2.81 (6)**	**0.83**	**1593.39**	**-3518.52**	**-336.18**	**1676.04**
	AFSE	full uncon	5584.57	1999			1586.57	-3509.01	-336.20	-1672.05
		full con	**5587.77**	**2005**	**3.20 (6)**	**0.78**	**1577.77**	**-3526.33**	**-343.99**	**-1683.86**
WP	ADSE	full uncon	5591.44	1995			**1601.44**	-3492.97	-326.50	-1659.69
		full con	**5605.27**	**2001**	**13.83 (6)**	**0.03**	**1603.27**	**-3504.97**	**-328.98**	**-1666.17**
	ADFE	full uncon	5605.10	1995			1615.10	-3486.14	-319.67	-1652.85
		full con	**5615.84**	**2001**	**10.74 (6)**	**0.10**	**1613.84**	**-3499.68**	**-323.69**	**-1660.88**
	AFSE	full uncon	5590.47	1995			**1600.47**	-3493.45	-326.98	-1660.17
		full con	**5604.67**	**2001**	**14.19 (6)**	**0.03**	**1602.67**	**-3505.27**	**-329.28**	**-1666.47**
BE	ADSE	full uncon	5623.19	1996			1631.19	-3480.25	-312.19	-1646.05
		full con	**5635.15**	**2002**	**11.97 (6)**	**0.06**	**1631.15**	**-3493.18**	**-315.60**	**-1653.46**
	ADFE	full uncon	5627.93	1996			1635.93	-3477.88	-309.82	-1643.68
		full con	**5638.87**	**2002**	**10.95 (6)**	**0.09**	**1634.87**	**-3491.32**	**-313.74**	**-1651.60**
	AFSE	full uncon	5623.07	1996			1631.07	-3480.31	-312.25	-1646.11
		full con	**5635.15**	**2002**	**12.09 (6)**	**0.06**	**1631.15**	**-3493.18**	**-315.60**	**-1653.46**
BD	ADSE	full uncon	5624.07	1996			1632.07	-3479.80	-311.75	-1645.60
		full con	**5634.04**	**2002**	**9.97 (6)**	**0.13**	**1630.04**	**-3493.73**	**-316.15**	**-1654.02**
	ADFE	full uncon	5625.42	1996			1633.42	-3479.13	-311.08	-1644.93
		full con	**5634.25**	**2002**	**8.83 (6)**	**0.18**	**1630.25**	**-3493.63**	**-316.05**	**-1653.92**
	AFSE	full uncon	5624.35	1996			1632.35	-3479.67	-311.61	-1645.47
		full con	**5634.04**	**2002**	**9.70 (6)**	**0.14**	**1630.04**	**-3493.73**	**-316.15**	**-1654.02**

Note: Total Score= Minnesota Eating Behavior Survey Total Score, BD= Body Dissatisfaction, WP= Weight Preoccupation; BE= Binge Eating; A= additive genetic, D= dominant genetic, S= environmental influences shared by siblings, F= environmental influences shared by all family members, and E= non-shared environmental influences. full uncon= fully unconstrained (i.e., allowing parameter estimates to vary between MSUTR and MTFS samples), full con= fully constrained (i.e., constraining all equivalent parameter estimates to be equal between the MSUTR and MTFS samples). The best fitting model for each sample is highlighted in bold font, and is indicated by lowest AIC = Akaike's Information Criterion, BIC = Bayesian Information Criterion, SABIC= sample size adjusted Bayesian Information Criterion, and DIC = Deviance Information Criterion values and a non-significant change in -2lnL. If fit statistics identify more than one model as best fitting, the model with the most model fit statistic indicating best fit was selected.

Table 3. Pearson Correlations Between Family Members' Disordered Eating Scores and Intraclass Correlations Between Cotwins' Disordered Eating Scores (n= 537 mothers, n=417 fathers, n=547 twin pairs, including 310 MZ and 237 DZ twin pairs)

	Pearson Correlations			Intraclass Correlations				
	Twin-Mom	Twin-Dad	Mom-Dad	MZ	DZ	z	p	q
Total Score	.16***	.10**	.08*	.45***	.41***	.78	.22	.05
WP	.13***	.05	.11**	.37***	.36***	.18	.43	.01
BE	.06	.10**	.03	.32***	.26***	1.04	.15	.07
BD	.14***	.08*	.07	.38***	.17***	3.63	<.01	.23
BMI	.34***	.20***	.22***	-	-	-	-	-

Note: Total Score= Minnesota Eating Behavior Survey Total Score; BD= Body Dissatisfaction; WP= Weight Preoccupation; BE = Binge Eating. M= mean; SD = standard deviation The "Test of Equality (z)" tests for the differences between the MZ and DZ twin correlations. Z indexes the magnitude of the difference between MZ and DZ twin correlations with the p-value indicating whether that difference is significant. Cohen's q is a calculation of the effect size with <.1 = no effect; .1 to .3 = small effect; .3 to .5 intermediate effect; >.5 = large effect.

*p <.05, **p<.01, ***p<.001. The correlation is greater than zero.

Table 4. NTFM Model Fit Statistics for Combined MSUTR + MTFS Sample by Disordered Eating Subscale (N=547 twin families)

Sx	Model	-2lnL	df	Δ -2lnL (Δdf)	p	AIC	BIC	SABIC	DIC
Total Score	Baseline	5561.91	1983						
	ADSE	5588.26	2005	26.34 (22)	0.24	1578.26	-3526.08	-343.74	-1683.61
	ADFE	5606.61	2005	44.70 (22)	<0.01	1596.61	-3516.90	-334.56	-1674.43
	AFSE	5587.77	2005	25.85 (22)	0.26	1577.77	-3526.33	-343.99	-1683.86
	ASE	**5588.26**	**2006**	**26.35 (23)**	**0.29**	**1576.26**	**-3529.23**	**-345.31**	**-1685.84**
	ADE	5606.61	2006	44.70 (23)	<0.01	1594.61	-3520.06	-336.13	-1676.67
	AFE	5603.39	2006	41.48 (23)	0.01	1591.39	-3521.67	-337.74	-1678.28
	AE	5631.65	2007	69.73 (24)	<0.01	1617.65	-3510.69	-325.18	-1666.38
WP	Baseline	5581.62	1979						
	ADSE	5605.27	2001	23.65 (22)	0.37	1603.27	-3504.97	-328.98	-1666.17
	ADFE	5615.84	2001	34.22 (22)	0.05	1613.84	-3499.68	-323.69	-1660.88
	AFSE	5604.67	2001	23.04 (22)	0.40	1602.67	-3505.27	-329.28	-1666.47
	ASE	**5605.27**	**2002**	**23.65 (23)**	**0.42**	**1601.27**	**-3508.12**	**-330.54**	**-1668.40**
	ADE	5619.22	2002	37.60 (23)	0.03	1615.22	-3501.14	-323.57	-1661.43
	AFE	5615.84	2002	34.22 (23)	0.06	1611.84	-3502.83	-325.25	-1663.12
	AE	5637.41	2003	55.79 (24)	<0.01	1631.41	-3495.20	-316.04	-1654.57
BE	Baseline	5618.14	1980						
	ADSE	5635.15	2002	17.02 (22)	0.76	1631.15	-3493.18	-315.60	-1653.46
	ADFE	5638.87	2002	20.74 (22)	0.54	1634.87	-3491.32	-313.74	-1651.60
	AFSE	5635.15	2002	17.02 (22)	0.76	1631.15	-3493.18	-315.60	-1653.46
	ASE	**5635.17**	**2003**	**17.04 (23)**	**0.81**	**1629.17**	**-3496.32**	**-317.15**	**-1655.69**
	ADE	5640.74	2003	22.61 (23)	0.48	1634.74	-3493.53	-314.37	-1652.90
	AFE	5638.87	2003	20.74 (23)	0.60	1632.87	-3494.47	-315.30	-1653.84
	AE	5652.42	2004	34.28 (24)	0.08	1644.42	-3490.85	-310.10	-1649.30
BD	Baseline	5608.83	1980						
	ADSE	5634.04	2002	25.22 (22)	0.29	1630.04	-3493.73	-316.15	-1654.02
	ADFE	5634.25	2002	25.42 (22)	0.28	1630.25	-3493.63	-316.05	-1653.92
	AFSE	5634.04	2002	25.22 (22)	0.29	1630.04	-3493.73	-316.15	-1654.02
	ASE	5637.00	2003	28.17 (23)	0.21	1631.00	-3495.41	-316.24	-1654.77
	ADE	5634.97	2003	26.14 (23)	0.29	1628.97	-3496.42	-317.26	-1655.79
	AFE	**5634.25**	**2003**	**25.42 (23)**	**0.33**	**1628.25**	**-3496.78**	**-317.62**	**-1656.15**
	AE	5653.60	2004	44.78 (24)	0.01	1645.60	-3490.26	-309.50	-1648.70

Note: Total= Minnesota Eating Behaviors Survey Total Score; WP= Weight Preoccupation; BE= Binge Eating: BD= Body Dissatisfaction;, A= additive genetic, D= dominant genetic, S= environmental influences shared by siblings, F= environmental influences shared by all family members, and E= non-shared environmental influences. The best fitting model for each sample is highlighted in bold font, and is indicated by lowest AIC = Akaike's Information Criterion, BIC = Bayesian Information Criterion, SABIC= sample size adjusted Bayesian Information Criterion, and DIC = Deviance Information Criterion values and a non-significant change in -2lnL. If fit statistics identify more than one model as best fitting, the model with the most model fit statistic indicating best fit was selected.

Table 5. Standardized and Unstandardized Parameter Estimates for the Best-Fitting NTFM and Full Model Including Family-Specific Shared Environment for each Disordered Eating Symptom (N= 547 twin families)

	Model		A	E	D	S	F	passrGE
Total	AFSE (full)	Std.	.048 (.000, .325)	**.557 (.479, .638)**	-	**.384 (.186, .500)**	.005 (.000, .008)	-
		Unstd.	.218 (-.563, .563)	**.747 (.692, .799)**	-	**.620 (.432, .707)**	.050 (-.083, .082)	.013 (-.028, .028)
	ASE (best)	Std.	**.137 (.042, .234)**	**.541 (.477-.614)**	-	**.321 (.221-.429)**	-	-
		Unsd.	**.368 (.204, .479)**	**.735 (.690, .784)**	-	**.567 (.470-.656)**	-	-
WP	AFSE (full)	Std.	.000 (.000, .285)	**.631 (.548, .710)**	-	**.360 (.146, .442)**	.006 (.000, .023)	-
		Unstd.	.000 (-.526, .526)	**.794 (.740, .842)**	-	**.600 (.381, .665)**	.053 (-.085, .053)	.000 (-.020, .020)
	ASE (best)	Std.	.093 (.000, .188)	**.614 (.543, .694)**	-	**.289 (.185, .400)**	-	-
		Unstd.	.303 (-.429, .429)	**.783 (.737, .833)**	-	**.538 (.430, .633)**	-	-
BE	AFSE (full)	Std.	.125 (.000, .439)	**.670 (.578, .775)**	-	.202 (.000, .368)	.000 (.000, .044)	-
		Unstd.	.354 (-.661, .661)	**.819 (.760, .881)**	-	.449 (-.606, .606)	-.010 (-.147, .098)	.004 (-.085, .019)
	ASE (best)	Std.	**.106 (.009, .203)**	**.674 (.597, .762)**	-	**.215 (.111, .325)**	-	-
		Unstd.	**.325 (.096, .451)**	**.821 (.773, .873)**	-	**.464 (.334, .570)**	-	-
BD	AFE (best)	Std.	**.450 (.317, .588)**	**.619 (.535, .716)**	-	-	**.046 (.015, .092)**	-
		Unstd.	**.668 (.561, .763)**	**.787 (.732, .846)**	-	-	**-.150 (-.210, -.085)**	**-.090 (-.135, -.047)**

Note: Total= Minnesota Eating Behavior Survey Total Score; WP= Weight Preoccupation; BE= Binge Eating; BD= Body Dissatisfaction; Std.= Standardized; Unstd. = Unstandardized; A= additive genetic, E= non-shared environmental influences, D= dominant genetic, S= environmental influences shared by siblings, F= environmental influences shared by all family members, passrGE = passive rGE, 95% Confidence intervals are present below each estimate. Any intervals that include zero are non-significant. Significant parameter estimates are in bold. The best-fitting models are indicated by "best" under the model name. If the best fitting model does not include the "F" parameter and thus, does not compute passive rGE, the full model was also included (i.e., "full" listed under the model name).

APPENDIX B

Figures

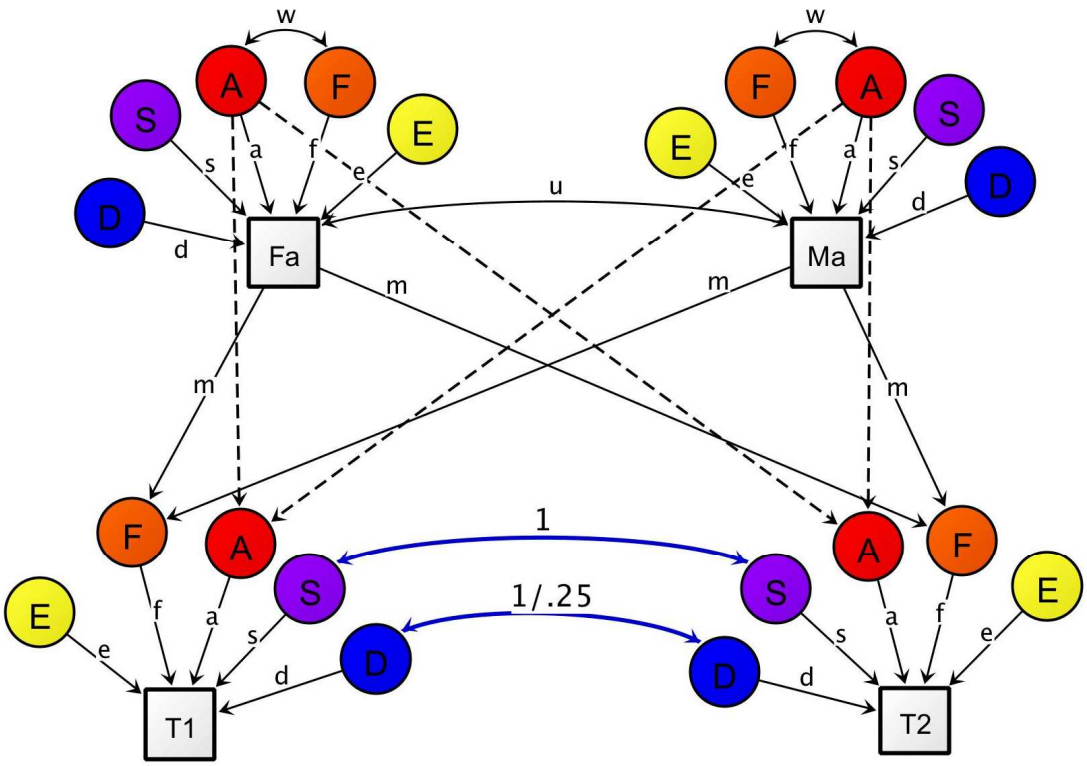

Figure 1. Path diagram of a univariate nuclear twin family model. The variance in a phenotype in fathers (Fa), mothers (Ma), and twins (T1 and T2) is divided into estimates of additive genetic effects (A), dominant genetic effects (D), sibling environmental influences (S), familial environmental influences (F), and non-shared environmental influences (E). u reflects assortative mating between the twins' parents. w indicates the covariance between A and F (i.e., passive rGE). Squaring the path estimates indicates the proportion of variance accounted for. Only two of the three sources of variance (D, S, F) can be estimated at one time (along with A and E, that are always assumed to contribute to the phenotype).

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