2015-07-27

Eating Pathology and Perceptions of the Parent-Child Relationship across Adolescence: Exploring Longitudinal Associations within a Monozygotic Twin Differences Design

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Eating Pathology and Perceptions of the Parent-Child Relationship across Adolescence: Exploring Longitudinal Associations within a Monozygotic Twin Differences Design

by

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A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE

DEGREE OF DOCTOR OF PHILOSOPHY

GRADUATE PROGRAM IN CLINICAL PSYCHOLOGY

CALGARY, ALBERTA

JULY, 2015

Abstract

The current study explored associations between eating pathology and the parent-child relationship (i.e., parent-child regard and involvement) across adolescence. Secondary data analyses were conducted of a population-based sample of 446 monozygotic (MZ) twins (i.e., 223 twin pairs), their mothers, and their fathers. The study used a MZ twin differences design that facilitated examination of the parent-child relationship as a nonshared environmental factor. Importantly, the MZ twin differences design has been labelled as the most powerful and direct approach for identifying nonshared environmental influences on development. Data from assessments when the twins were approximately age 11, 14, and 17 years were included. Part 1 of the study used cross-lagged models to consider longitudinal, bidirectional associations among variables, including the temporal order of variables associated across time. Specifically, analyses examined whether twin differences in mother-child and father-child relationship problems emerged as a risk factor for, or outcome of, twin differences in eating pathology; in other words, whether the twin who reported greater parent-child relationship problems later reported greater eating pathology, as compared to her co-twin, and/or vice versa. Parts 2-5 further clarified and elaborated on findings and addressed several methodological limitations of past research, including controlling for the influence of body mass index on eating pathology, examining whether findings generalized to other psychopathology, and considering multiple informants' ratings of the parent-child relationship. Overall, longitudinal associations of twin differences in eating pathology and twin differences in the parent-child relationship were generally neither strong nor robust. However, where longitudinal associations emerged, overall patterns indicated reciprocal associations between symptoms of eating pathology and parent-child relationship variables that shifted across developmental periods. Specifically, whereas twin differences in

parent-child relationship variables more commonly predicted later twin differences in eating pathology across early adolescence, twin differences in eating pathology more commonly predicted later twin differences in parent-child relationship variables across later adolescence. In particular, the twin who reported greater eating pathology later reported more negative perceptions of the father-child relationship, as compared to her co-twin. These associations between eating pathology and parent-child relationship variables were observed independent of any confounding effects of genetics or the shared environment. Findings raise questions for future research regarding parental – in particular, paternal – responses to adolescent eating pathology and suggest the potential importance of efforts to support the parent-child relationship within the context of adolescent eating pathology.

Acknowledgements

To my supervisor, Kristin von Ranson, thank you for your relentless encouragement and support throughout my graduate studies. Your passion for research and high standards with which you conduct your work, balanced with your supportive, thoughtful, and compassionate nature, have made my graduate studies both successful and enjoyable. You have supported me both as a student and a person, and I am forever grateful. I am so fortunate to have worked with you.

To my cohort of colleagues, I cannot imagine taking this journey with anyone else. Thank you for the support, the insight, and most importantly, the laughs. And to all of the members of the Eating Behaviors lab, I appreciate so much being a part of your team.

To my committee members, thank you for contributing your time, ideas, and knowledge throughout this process. I have greatly benefitted from your feedback and guidance.

To the Minnesota Twin Family Study, thank you for allowing me the opportunity to access both your remarkable database and the wealth of knowledge your research staff hold. Both have been critical to making this project possible.

And most importantly, to my family, thank you for your unwavering support. Thank you for standing by me, encouraging me to be me, and reminding me of the important things in life, whenever I need it most. Knowing you are always there, through the highs and the lows, is my greatest source of confidence. Thank you.

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AN	anorexia nervosa
BED	binge eating disorder
BMI	body mass index
BN	bulimia nervosa
CFI	comparative fit index
FIML	full information maximum likelihood
MAR	missing at random
MEBS	Minnesota Eating Behaviors Survey
MLR	maximum likelihood estimation with robust standard errors
MTFS	Minnesota Twin Family Study
MZ	monozygotic
PEQ	Parental Environment Questionnaire
RMSEA	root mean square error of approximation
STAI	State-Trait Anxiety Inventory

Eating Pathology and Perceptions of the Parent-Child Relationship across Adolescence: Exploring Longitudinal Associations within a Monozygotic Twin Differences Design

Eating disorders are associated with severe negative implications for social, emotional, and functional well-being, and are often accompanied by serious medical complications (Klump, Bulik, Kaye, Treasure, & Tyson, 2009). Based on DSM-5 criteria for eating disorders, approximately 13% of females may develop clinically significant eating pathology by young adulthood (Stice, Marti, & Rohde, 2012). In addition, subclinical eating pathology, including body dissatisfaction and disordered eating attitudes and behaviors that do not meet criteria for a clinical disorder, is even more prevalent. Research suggests 46%-80% of adolescent girls are dissatisfied with their weight, 26%-77% of adolescent girls have dieted, and 5%-16% of adolescent girls have engaged in compensatory behavior (e.g., vomiting, laxatives, or diuretics; Chamay-Weber, Narring, & Michaud, 2005). Furthermore, individuals who endorse subclinical eating pathology often endorse negative psychosocial and medical challenges that are similar to those observed among individuals with clinical eating disorders, which emphasizes the severity of such problems (Thomas, Vartanian, & Brownell, 2009). Recognizing the widespread prevalence of eating pathology (defined hereafter as including both clinical and subclinical symptoms of eating disorders) and the widespread implications for health and psychosocial well-being, understanding and ultimately reducing eating pathology is imperative.

It is widely accepted that eating pathology is a multiply determined problem involving complex interactions among genetics (e.g., heritability estimates of 59-82%; Klump, Suisman, Burt, McGue, & Iacono, 2009) and various environmental factors (e.g., Striegel-Moore & Bulik, 2007). Research continues to call for increased efforts to identify specific factors that affect

eating pathology and better understand the specific role these factors play in the development and maintenance of eating pathology.

One factor that has oft been considered in the development of eating pathology is the parent-child relationship. A better understanding of the specific nature of associations between eating pathology and the parent-child relationship may have implications for eating pathology prevention and/or treatment research, including the development of community and clinical programs to reduce eating pathology.

Eating Pathology and the Parent-Child Relationship

Although now controversial, families – in particular, parents – were historically theorized to be a primary cause of the development of eating pathology in children and adolescents. For example, early theorists such as Gull (1874) and Charcot (1889) described relatives of individuals with eating pathology as "generally the worst attendants" to assist individuals in their recovery from eating pathology and referred to parents' influence on the development of eating pathology as "particularly pernicious" (as cited in Le Grange & Eisler, 2008). However, perspectives regarding the role of parents in child and adolescent eating pathology began to shift around the 1960s. For example, Minuchin and colleagues (1978) emphasized that parents should not be to blame for adolescent eating pathology, yet continued to theorize that certain pathological interactive familial processes (e.g., enmeshment, rigidity, conflict avoidance, etc.) were fundamental to the pathogenesis of eating pathology, particularly AN. They maintained that families of children and adolescents with eating pathology were "psychosomatic," claiming that such families did not provide adequate emotional support for their children and instead transformed emotional conflicts into somatic symptoms.

Minuchin and colleagues' view that the family serves an important role in the development of eating pathology has subsequently been supported by research demonstrating that parent-child relationship problems and general family dysfunction, as measured by a range of variables, is associated with eating pathology in children and adolescents. For example, a large-scale review that examined putative risk factors for eating disorders (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004) indicated that various aspects of the family environment (e.g., interactions, communication, closeness, emotional expression) of individuals with eating disorders are more dysfunctional (e.g., disturbed, conflictual, pathological, etc.) than the family environment of individuals without eating disorders. More recent reviews of eating pathology and family functioning have largely supported these findings (e.g., Eisler, 2005; Holtom-Viesel & Allan, 2013), yet have emphasized that there is little evidence of a typical pattern of family dysfunction. This research is reviewed below, followed by discussion of methodological limitations of such research.

Low parent-child connectedness, often conceptualized as involving low closeness and communication, has been associated with eating pathology. For example, perceptions of low parental connectedness and communication have been associated with increased odds of adolescents engaging in unhealthy weight control behaviors, binge eating, and dieting in large-scale population-based studies (Ackard, Neumark-Sztainer, Story, & Perry, 2006; Berge et al., 2014). Likewise, a large, statewide survey on adolescent health indicated family connectedness and communication were protective against extreme dieting among adolescent girls (Fonseca, Ireland, & Resnick, 2002). Further, among samples of treatment-seeking adolescent and young adult patients with diagnosable eating disorders, both patients and their mothers reported difficulties with family cohesion and communication (Shisslak, McKeon, & Crago, 1990;

Vidovic, Juresa, Begovac, Mahnik, & Tocilj, 2005). In particular, impaired family communication involving affective expression has been reported to be associated with eating pathology (e.g., Stern et al., 1989; Topham et al., 2011).

Associations are also reported between eating pathology and parent-child attachment. For example, secure parent-child attachment during childhood and adolescence has been reported to protect against the development of eating pathology, whereas an insecure parent-child attachment has been reported to increase risk for eating pathology (Back, 2011; Goossens et al., 2012; Pace et al., 2012), particularly among adolescents who present with other risk factors (e.g., elevated pubertal weight gain, maternal negative affect; Milan et al., 2014). Associations have been demonstrated with both mother-child and father-child attachment relationships, although research suggests various parental attachment relationships may differentially affect various eating pathology symptoms (Goossens, Braet, Van Durme, Decaluwe, & Bosmans, 2012). Parent-child regard – a construct closely linked to parent-child attachment – has also demonstrated associations with eating pathology. Parent-child regard is often conceptualized as involving unconditional love, respect, and acceptance, qualities that are critical to facilitating the emotionally secure and nurturing base associated with a secure parent-child attachment (Allen & Land, 1999; Elkins, McGue, & Iacono, 1997; Lamis, Malone, Lansford, & Lochman, 2012). A large-scale, population-based study of adolescent eating pathology reported that adolescents who perceived their mothers did not regard them highly (e.g., cared very little or not at all about them) demonstrated particularly high prevalence of unhealthy weight control behaviours (e.g., fasting, dieting, self-induced vomiting, and laxative/diuretic misuse; Ackard et al., 2006). Similarly, another large-scale, population-based study indicated that adolescents who perceived

they were not loved enough by their mother were more likely to develop a diagnosable eating disorder (Beato-Fernandez, Rodriguez-Cano, Belmonte-Llario, & Martinez-Delgado, 2004).

Parental overprotection and high control have also been reported to be associated with eating pathology (Salafia, Gondoli, Corning, Bucchianeri, & Godinez, 2009). Specifically, parental overprotection and high control have been suggested to interfere with adolescents' ability to self-regulate emotions and behaviors and to increase their competence and independence. In turn, adolescents have been suggested to develop lowered perceptions of their self-competence and focus on changing their bodies and/or eating habits as a maladaptive means of attaining developmentally-normative independence and self-control. As a result, such adolescents may be at increased risk to become preoccupied with their body image, weight, and shape, and ultimately to engage in disordered eating behaviors (Salafia et al., 2009). In particular, parental psychological control (i.e., intrusive, manipulating, and/or coercive parenting behaviors) has demonstrated consistent associations with eating pathology, including increased emotional eating, dieting, and other disordered eating behaviors (e.g., Berge et al., 2014; Snoek, Engels, Janssens, & van Strien, 2007).

On the other hand, lack of parental monitoring and knowledge about their adolescent's whereabouts (e.g., who they are with, what they are doing, and where they are) have also been associated with eating pathology, emphasizing the importance of balanced parenting behaviors (e.g., maintain appropriate parental monitoring and knowledge within the context of low parental overprotection and psychological control) to reduce eating pathology risk (Berge et al., 2014; Mellin, Nuemark-Sztainer, Story, Ireland, & Resnick, 2002). Specifically, lower parental monitoring and knowledge have been associated with increased risk for adolescent dieting,

weight concerns, and binge eating (Berge et al., 2014; Krug, Sorabji, King, Letcher, & Olsson, 2014; May, Kim, McHale, & Crouter, 2006).

In addition, other parenting variables that are more conceptually relevant to eating, shape, and weight have also demonstrated associations with adolescent eating pathology. For example, a family focus on appearance and family weight-, food-, and appearance- related teasing have been associated with the development of eating pathology, including body dissatisfaction, thin-ideal internalization, binge eating, purging, and extreme weight control behaviors (Annus, Smith, Fischer, Hendricks, & Williams, 2007; Eisenberg, Berge, Fulkerson, & Neumark-Sztainer, 2012; Keery, Boutelle, van den Berg, & Thompson, 2005; Kluck, 2010; Neumark-Sztainer et al., 2007). Further, symptoms of maternal eating pathology – including maternal body dissatisfaction, drive for thinness, and eating disorder diagnosis – have been associated with child and adolescent eating pathology (Anschutz, Kanters, van Strien, Vermulst, & Engels, 2009; Canals, Sancho, & Arija, 2009; Jacobi, Schmitz, & Agras, 2008; Stein et al., 2006). Although maternal eating pathology is not a specific parenting practice or relationship factor, the association between parent and child eating pathology may be attributable, in part, to parenting behaviours and interactions that serve to model and/or reinforce eating pathology.

Methodological Limitations

Despite accumulating research suggesting associations between eating pathology and the parent-child relationship, as reviewed above, advancements in delineating and understanding the specific role of the parent-child relationship in the development and maintenance of eating pathology continue to be limited by methodological shortcomings of available research. As reviewed below, such methodological limitations interfere with a fine-tuned understanding of associations between eating pathology and the parent-child relationship.

Use of a single informant. Research regarding the parent-child relationship has often relied on data from only one informant – typically, the child or adolescent. A recent review of family functioning and parent-child relationships across eating disorder subtypes emphasized that different family members often provide discrepant reports regarding family functioning and relationships (Holtom-Viesel & Allan, 2013). This aligns with the concept of informant variability – i.e., the extent to which alternative sources of information vary in perceptions of a construct (Kazdin, 1994). Typically, adolescents with eating pathology have reported poorer perceived family functioning and parent-child relationships than their parents, including reporting poorer cohesion, communication, and affective expressiveness and greater conflict (Holtom-Viesel & Allan, 2013). Similar patterns of discrepant reports regarding family functioning and relationships between adolescents and parents have been reported in non-clinical samples, with indications that greater parent-child discrepancies in perceptions of family functioning are associated with greater difficulties with emotional adjustment (Ohannessian, Lerner, Lerner, & von Eye, 1995). Understanding differences in the perspectives of parents and adolescents and differential associations of each perspective with eating pathology has important implications for understanding the course of eating pathology, providing appropriate assessment, and identifying targets for treatment. Indeed, family systems perspectives suggest that information from one family member reflects only that individual's perception of the family and various relationships; multiple family member perspectives are necessary for a thorough understanding of the family environment and relationship dynamics (Carlson, Cooper, & Spradling, 1991; Dekovic & Buist, 2005). More research that includes the perspectives of both children/adolescents and their parents when exploring associations between the parent-child relationship and eating pathology is needed.

Overlooking the father-child relationship. Despite consistent findings that adolescents' relationships with mothers and fathers differ, associations of eating pathology – as well as other indicators of psychological well-being – with the father-child relationship have historically been overlooked. Similarly, some research has provided a "composite score" that averaged mother-child and father-child relationship variables (e.g., Spanos et al., 2010). As a result, an understanding of the differential associations of mother-child and father-child relationships on adolescent development is rarely obtained (Smetana, Campione-Barr, & Metzger, 2006).

However, research increasingly suggests the father-child relationship is important to understanding psychological well-being, including eating pathology (e.g., Dixon, Gill, & Adair, 2003; Videon, 2005; Williams & Kelly, 2005). In particular, recent research suggests opposite-sex parent-child relationships (i.e., father-daughter and mother-son relationships) may be particularly influential to adolescent weight-related behavior (Berge, Wall, Bauer, & Neumark-Sztainer, 2010; Berge, Wall, Loth, & Neumark-Sztainer, 2010). Only by separately examining both the mother-child and father-child relationship in research related to eating pathology and the parent-child relationship may we understand the extent to which each parent's unique relationship is differentially associated with eating pathology .

Lack of developmental perspective. Lack of inclusion of a developmentally appropriate perspective when designing studies and interpreting results constitutes another major methodological limitation of available research on associations between eating pathology and the parent-child relationship. Beginning assessment at pre-adolescence and extending assessment through adolescence is important, as adolescence is associated with high risk for the development of eating pathology (Herpertz-Dahlmann, 2008; Striegel-Moore & Bulik, 2007; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). For example, changes such as the

transition through puberty, physical maturation, development of greater self-identity, and shifting importance of interpersonal relationships all occur during adolescence and have all been observed to contribute to increased risk for eating pathology (von Ranson & Wallace, 2014). Likewise, causes and consequences of eating pathology change across adolescence. For example, significant changes have been reported from pre-adolescence to late adolescence in the proportion of genetic and shared environmental effects that account for variance in eating pathology, with genetic influences increasing and shared environmental influences decreasing (Klump, Burt, McGue, & Iacono, 2007).

In addition, adolescence is associated with significant changes in parent-child relationships and the role of the parent in promoting positive development. For example, as the child transitions through adolescence he/she seeks greater independence from family and relationships with peers assume greater importance peers (McCabe, Ricciardelli & Finemore, 2002; Steinberg, 2001), including at times choosing to seek emotional support and share personal experiences with peers rather than parents. The structure of the parent-child relationship begins to shift, moving from a more hierarchical structure in childhood to a more egalitarian structure in adolescence (De Goede, Branje, & Meeus, 2008; Markiewicz, Lawford, Doyle, & Haggart, 2006; Smetana, et al., 2006). Parent-child regard and involvement have been reported to decrease as conflict increases across adolescence, with these changes even greater for girls than boys (McGue, Elkins, Walden, & Iacono, 2005). However, research indicates that despite these changes, the parent-child relationship remains an important social and emotional resource throughout adolescence to support positive adolescent well-being (Hair, Moore, Garrett, Ling, & Cleveland, 2008; Smetana et al., 2006). With such changes in eating pathology and the parent-child relationship across adolescence, it is likely that associations between these variables may

also shift across this developmental period (e.g., Archibald, Linver, Graber, & Brooks-Gunn, 2002). Indeed, finding that associations between variables vary across different developmental periods would align with a “critical periods” theory of parent-child relationships and adolescent development, wherein parents are considered to have more influence over their children at certain ages than at other ages (Montemayor, Adams, & Gullotta, 1994).

Unfortunately, available research on eating pathology and the parent-child relationship often does not clearly define the specific age span examined in such research, so as to possibly identify associations between variables that may shift across developmental periods. In addition, such research tends to have not begun assessment early enough in the lifespan and/or not controlled for initial eating pathology and/or parent-child relationship problems. As symptoms of eating pathology can onset in childhood, it is important to begin assessment as early as possible. Otherwise, conclusions that can be drawn are limited, as it is not known whether difficulties related to the variables of interest already existed prior to assessment and contributed to any observed associations.

Lack of specificity. Another major methodological limitation of available research on associations between eating pathology and the parent-child relationship pertains to the fact that research often lacks appropriate controls and/or comparison groups and analyses to consider whether results are specific to eating pathology or general to other psychopathology (Le Grange et al., 2010). As parenting issues are oft implicated in numerous forms of psychiatric dysfunction (Weich, Patterson, Shaw, & Stewart-Brown, 2009), it is possible that parent-child relationship variables identified as being associated with eating pathology are also associated with risk for psychopathology in general. Indeed, research on risk and protective factors for adolescent emotional and physical health have indicated that strong parent-child relationships are critical for

protecting against a range of emotional and physical health problems (Boutelle, Eisenberg, Gregory, & Neumark-Sztainer, 2009; Resnick et al., 1997) – not just eating pathology. On the other hand, some research also suggests certain parent-child relationship variables are specific predictors of eating pathology. For example, a two-year longitudinal study of over 200 mother-child dyads that controlled for levels of child psychological distress indicated that certain variables related to the parent-child relationship and/or family functioning (e.g., maternal concern about child weight, child's level of family satisfaction, family exposure to stress) were specifically associated with early adolescent eating pathology and not general to other psychopathology (Allen, Gibson, McLean, Davis, & Byrne, 2014).

Not genetically informed. Most research in this area is not genetically informed, hence fails to account for the moderate to large heritability of eating pathology. The first adoption study to examine genetic and environmental effects for eating pathology indicated that genetic influences account for 59%-82% of the variance in eating pathology (including overall eating pathology, body dissatisfaction, weight preoccupation, and binge eating; Klump, Suisman, et al., 2009). Other twin research that has examined heritability estimates across development has indicated that, among females, genetic factors account for approximately 6% of variance in eating pathology during pre-adolescence and increase to account for approximately half of variance in eating pathology during later adolescence (Klump et al., 2007). This increase in genetic influence is suggested to be activated by sex-specific processes (e.g., estradiol levels) during puberty (Culbert, Burt, McGue, Iacono, & Klump, 2009; Klump et al., 2010; Klump et al., 2012). Thus, it is necessary to account for genetic effects to address the problem of confounded genetic and environmental sources of variance in eating pathology (Beaver, 2008; Deater-Deckard et al., 2001), and rule out possibilities such as (a) a genetically transmitted

liability explains both parent-child relationship variables and eating pathology, and/or (b) a genetically influenced disposition evokes certain parenting responses and thereby impacts the parent-child relationship.

Shared versus nonshared environment. Consideration and definition of family functioning in research related to eating pathology at times lacks conceptual clarity. In particular, research often fails to differentiate between overall family climate and issues specific to the parent-child relationship. An important distinction between overall family climate and specific parent-child relationship variables is that the former represents a shared environmental factor, whereas the latter represents a nonshared environmental factor. Shared environmental factors are environmental influences that are experienced by all children growing up in the same family and create similarities between these children (e.g., socioeconomic status, parental education, family structure). In contrast, nonshared environmental factors are environmental influences that are specific to each child growing up in the same family and create differences between these children (e.g., child-specific parenting, experiences unique to one child; Plomin, Asbury, & Dunn, 2001). Research consistently indicates that nonshared environmental factors are responsible for most environmental variation relevant to psychological development (Plomin et al., 2001; Plomin et al., 2011). In particular, research indicates that there are significant sibling differences in parental treatment and parent-child relationships within families (i.e., nonshared environmental factors) and these differences are associated with child and adolescent outcomes (e.g., Feinberg & Hetherington, 2001; Kowal, Krull, & Kramer, 2004; Plomin et al., 2011). In the field of eating disorders, research indicates that shared environmental factors account for approximately 40% of the variance in eating pathology during pre-adolescence yet account for only approximately 10% of the variance in eating pathology during middle and late adolescence

(e.g., Klump, et al., 2007). In comparison, nonshared environmental factors are suggested to have a significant and relatively stable effect on the development of eating pathology throughout adolescence, accounting for approximately 44%-54% of the variance in eating pathology across pre-adolescence to late adolescence. (The remaining variance is accounted for by genetic factors, which increases from approximately 6% of the variance in eating pathology during pre-adolescence to 46% of the variance during middle and late adolescence.)

In sum, these findings emphasize that across adolescence nonshared environmental factors account for more of the variance in eating pathology than shared environmental factors. Based on these findings, it is possible that examination of specific nonshared environmental factors of family functioning across adolescence, such as parent-child relationship variables, will prove more important and fruitful than examination of shared environmental factors of family functioning. Examination of nonshared environmental factors can help identify why siblings living within the same household differ from each other on eating pathology symptoms and severity. In addition, nonshared environmental family factors, as compared to shared environmental family factors, often reflect less entrenched family variables that are more amenable to change. As such, these factors can be more easily targeted for change via eating pathology prevention and/or treatment efforts.

Reliance on cross-sectional designs. Finally, a major methodological limitation of available research on associations between eating pathology and the parent-child relationship involves the use of cross-sectional designs. Cross-sectional designs have been used in the majority of research in the area of eating pathology and the parent-child relationship, including most of the research reviewed above. Cross-sectional research can identify correlates of eating pathology but cannot differentiate variables as risk factors, concomitants, or consequences of

eating pathology. Establishing correlates of an outcome is an important first step in identifying risk factors (Jacobi et al., 2004). However, establishing the precedence of a proposed risk factor to a proposed outcome is a crucial criterion for distinguishing risk factors from correlates (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001), which typically requires a longitudinal design. Of the available prospective research that has examined parent-child relationship variables as predictors of later onset of eating pathology, results are inconsistent. Whereas some longitudinal studies have found parenting variables to be significant predictors of subsequent eating pathology (Beato-Fernandez et al., 2004; Johnson, Cohen, Kasen, & Brook, 2002; Neumark-Sztainer et al., 2007; Salafia et al., 2009), others have not (Attie & Brooks-Gunn, 1989; McKnight Investigators, 2003; Nicholls & Viner, 2009).

Although findings are somewhat conflicting, overall trends suggest longitudinal studies that have examined more specific parenting behaviors (e.g., inadequate parental supervision, low parental affection; Johnson et al., 2002), rather than more general parenting styles (e.g., authoritarian parenting; Nicholls & Viner, 2009), have more consistently identified significant longitudinal associations between parenting variables and subsequent eating pathology. These findings align with recent shifts in the family and psychology field that have emphasized the importance of measuring specific parenting practices, rather than general parenting styles (e.g., Barber, Stolz, Olsen, Collins, & Burchinal, 2005; Berge et al., 2014). In addition, trends suggest longitudinal associations between parenting variables and subsequent eating pathology have more consistently emerged when assessments have been completed at specific age points (e.g., age 13 and again at age 15 [Beato et al., 2004], grade 6 and again at grade 8 [Salafia et al., 2009]), rather than when assessments have included a range of ages (e.g., grades 7-10 and again 2 years later [Attie & Brooks-Gunn, 1989], grades 6-9 and again 4 years later [McKnight

Investigators, 2009]). These findings highlight the importance of examining specific developmental periods and recognizing that associations may shift across development. Likely, parental associations with eating pathology are complex and interactive.

Conceptual Shift: Risk Factor or Outcome?

Such inconsistent findings across longitudinal studies have led to a recent conceptual shift regarding the role of the parent-child relationship in child and adolescent eating pathology, which provides the impetus for the current study. Specifically, rather than emphasizing parental etiological factors in the development of eating pathology, researchers have been moving toward emphasizing parental maintenance mechanisms in the progression of eating pathology. This shift aligns with the theory of Maudsley family-based therapy, which takes the perspective that parents can be resources for their child's recovery yet parents are not pathological and should not be blamed for the development of eating pathology (le Grange & Eisler, 2008). This manualized treatment has garnered accumulating empirical support for the treatment of AN and BN (Couturier, Kimber, & Szatmari, 2013). Many researchers have now hypothesized that parent-child relationship difficulties and overall family distress are likely outcomes, rather than causes, of living with a child with a condition that threatens their medical and mental health (Nilsson, Engstrom, & Hagglof, 2012; Sim et al., 2009). Possibly, parent-child relationship problems are both risk factors for *and* outcomes of eating pathology, with the nature of the relationship shifting depending on the specific parent-child relationship variables assessed and the specific developmental period examined. Unfortunately, among available prospective research, most studies tend to have examined parent-child relationship variables as predictors of the later development of eating pathology without considering the reverse possibility – i.e., that eating pathology may predict later parent-child relationship problems.

However, some exceptions exist. For example, strong empirical support for the perspective that eating pathology precedes parent-child relationship problems was provided by a recent longitudinal, population-based study by Spanos, Klump, Burt, McGue, and Iacono (2010). Spanos et al.'s (2010) study examined associations between eating pathology and parent-child conflict across adolescence and utilized the same sample as used in the current study. Notably, Spanos et al. used a particularly rigorous analytic approach, including cross-lagged models that controlled for pre-existing and within-age associations and considered bidirectional associations between variables (i.e., parent-child conflict could predict eating pathology and/or eating pathology could predict parent-child conflict). Contrary to hypotheses, findings indicated that parent-child conflict at age 17 years was a consequence of eating pathology (in particular, weight preoccupation) at age 14 years and was not a precipitant of eating pathology. Findings indicated no longitudinal associations between the variables assessed during early adolescence (i.e., age 11 to 14 years). Similarly, a longitudinal study that examined bidirectional associations between eating pathology (specifically, dieting and food preoccupation) and the overall quality of the parent-child relationship indicated that greater dieting and food preoccupation at approximately age 14 years predicted lower parent-child relationship quality at approximately age 16 years, and not the reverse (Archibald et al., 2002). Relatedly, longitudinal treatment research has demonstrated that improvements in family relationships (e.g., increased closeness, improved communication) reported by both adolescents and their parents are associated with recovery from adolescent onset eating disorders (Nilsson et al., 2012), regardless of treatment type (Ciao, Accurso, Fitzsimmons-Craft, Lock, & Le Grange, 2015). Based on these findings, some authors have cautioned against concluding that the parent-child relationship and family functioning are causal in the development of eating pathology, and instead suggested that negative family

dynamics are reactions to the adolescent's eating pathology that diminish as a result of the impact of the illness decreasing (Nilsson et al., 2012).

However, some research that has considered bidirectional associations between eating pathology and parent-child relationship variables is conflicting. For example, cross-lagged models that examined longitudinal associations between body satisfaction and family connectedness indicated reciprocal effects wherein adolescent girls' body satisfaction was both affected by and a predictor of perceived family connectedness (Crespo, Kielpikowski, Jose, & Pryor, 2010). Further, research with a similar sample of adolescent girls as described above (see Archibald et al., 2002) that was completed at an earlier point in development indicated that lower parent-child relationship quality at age 12 years predicted greater dieting at age 13 years, and not the reverse (Archibald, Graber, & Brooks-Gunn, 1999). A similar pattern of results was also observed between body image and the parent-child relationship, although results only trended toward significance. This direction of associations between variables was opposite to that observed within the sample assessed by the same authors at age 14 and 16 years (Archibald et al., 2002). In comparing results from both studies, the authors suggested that the parent-child relationship may have greater effects on the development of eating pathology in early adolescence, whereas eating pathology may weaken the parent-child relationship in later adolescence (Archibald et al., 2002). Further research in this area is needed to untangle and delineate associations. Notably, the direction of association between variables has significant implications for prevention and treatment research, as the target of any interventions will differ dependent on which variable predicts the other.

Purpose

As reviewed above, there are significant methodological limitations of past research regarding eating pathology and the parent-child relationship and outstanding questions regarding whether parent-child relationship problems are a risk factor for, or outcome of, eating pathology. Thus, researchers are calling for more methodologically rigorous research in this area to better understand associations between eating pathology and the parent-child relationship (Le Grange et al., 2010; Stice, South, & Shaw, 2012). The overarching goal of this exploratory study was to address this call. *Specifically, the purpose of the current study was to examine longitudinal, bidirectional associations among eating pathology and parent-child relationship variables across adolescence using a methodologically rigorous research design that overcame several of the methodological challenges that have limited previous study conclusions.* Specifically, to overcome each of the methodological limitations of past research reviewed above (see Methodological Limitations section), the current study used both the child and parents' perspectives of the parent-child relationship; separately examined the mother-child and father-child relationship; incorporated a developmental perspective that included and differentiated early and late adolescent periods; considered the specificity of findings by comparing findings to associations between the parent-child relationship and other psychopathology; used a MZ twin differences design to control for confounding genetic and shared environmental factors and isolate the parent-child relationship as a nonshared environmental factor; and included a longitudinal design that considered bidirectional associations among variables. Further details regarding how such methodological limitations were overcome are included below.

Extension of Spanos et al., 2010. Notably, this study extended previous work by Spanos et al. (2010), reviewed above, that examined longitudinal, bidirectional associations between

eating pathology and parent-child conflict. Specifically, following methodology used by Spanos et al., cross-lagged models were used in the current study to examine associations between eating pathology and parent-child relationship variables to consider the temporal order of variables associated across time: i.e., both the extent to which parent-child relationship variables predicted later eating pathology and eating pathology predicted later parent-child relationship variables. Whereas Spanos et al. examined parent-child conflict, parent-child regard and involvement were examined in the current study. To address other limitations of past research, the current study extended analyses beyond those used by Spanos et al. to consider differential associations between eating pathology and the mother-child versus father-child relationship; explore whether findings generalized to other psychopathology; and examine if findings differed based on whether the child or parent's perception of the relationship was considered.

A monozygotic (MZ) twin differences design was used in the current study, as used by Spanos et al. (2010). This design isolates the parent-child relationship as a nonshared environmental factor and controls for genetic and shared environmental confounds. Notably, the MZ twin differences design has been described as ideal for isolating specific nonshared environmental influences and labelled as the most powerful and direct approach for identifying nonshared environmental influences on development (Mullineaux, Deater-Deckard, Petrill, & Thompson, 2009; Vitaro, Brendgen, & Arsenault, 2009).

Recognizing the importance of understanding the MZ twin differences design to interpret study findings, a brief review of the logic and value of the MZ twin differences design is included below.

Logic and value of the MZ twin differences design. Individual differences in phenotypes are the result of a combination of genetic and environmental factors. The MZ twin

differences design involves examination of phenotypic differences (e.g., developmental outcomes) between MZ twins from the same pair and exploring associations between such phenotypic differences and differences in environmental experiences (Vitaro et al., 2009). Such methodology facilitates identification of nonshared environmental experiences that are associated with developmental outcomes, while controlling for genetic and shared environmental confounds. Specifically, in the MZ twin differences design the correlation between twin differences in developmental outcomes and twin differences in environmental experiences reflects the contribution of nonshared experiences to the creation of phenotypic differences, independent of either genetics or factors that differ between families (Rovine, 1994).

Why is this so? At its essence, the foundation for the MZ twin differences design lies in the fact that MZ twins are matched on their genetic background and exposure to numerous shared environmental factors, yet differ in their exposure to nonshared environmental factors. That is, MZ twins are genetically identical, sharing 100% of each other's genes. In addition, MZ twins reared in the same household are exposed to many of the same environmental factors (i.e., shared environmental factors), such as prenatal exposure to maternal stress, socioeconomic status, parental psychopathology, family composition, etc. However, MZ twins are also exposed to experiences that are unique to each twin (i.e., nonshared environmental factors). For example, each twin may develop unique relationships, suffer unique injuries, be exposed to unique substances, and experience unique stressful life events. These differences in nonshared environmental factors represent a major source of influence that can affect differences in developmental outcomes.

By examining within-twin pair differences in nonshared environmental factors and connecting these differences to within-twin pair differences in developmental outcomes,

associations can be identified between nonshared environmental factors and developmental outcomes. As MZ twins share 100% of their genes as well as certain aspects of their environment (as described above), genetic and shared environmental factors cannot account for differences in developmental outcomes between MZ twins from the same pair. In other words, the influence of genetic and shared environmental factors on developmental outcomes is the same for both twins thus is methodologically controlled in the MZ twin differences design.

In contrast, when other research methods are used that do not control for genetic and shared environmental factors, these genetic and shared environmental factors are confounded. That is, genetic and shared environmental factors may be confounded with nonshared environmental experiences via gene-environment correlations or environment-environment correlations. For example, a gene-environment correlation could occur if a child's genetic disposition to eating pathology increased the child's risk of developing a parent-child relationship characterized by low parental regard. In this case, any observed association between exposure to low parental regard and subsequent eating pathology may be accounted for, at least in part, by the child's genetic disposition to eating pathology, rather than to low parental regard itself. As another example, an environment-environment correlation could occur if high parental psychopathology in a family increased the risk of a child developing a parent-child relationship characterized by low parental involvement. In this case, any association observed between the experience of low parental involvement and subsequent eating pathology might be explained, at least in part, by living in a family with high parental psychopathology.

As these examples illustrate, genetic and shared environmental factors can often interfere with interpreting an observed association between a nonshared environmental factor and a developmental outcome. As a result, it often remains unclear to what extent an observed

association between a nonshared environmental factor and a developmental outcome may be attributable to genetic and/or shared environmental factors. Thus, it is important to control for genetic and shared environmental factors to be able to more clearly interpret and understand results, as can be done via the MZ twin differences design.

Of course, despite controlling for genetic and shared environmental factors, other factors (e.g., peer relationships, other psychopathology) may be unique to each twin within an MZ pair when analyzing the association between twin differences in a nonshared environmental factor and twin differences in a developmental outcome (Vitaro et al., 2009). It may also be important to identify and control for any such correlated factors that may operate as confounds with either the nonshared environmental factor or the developmental outcome of interest (e.g., via regressing such factors from scores on either the nonshared environmental factor or developmental outcome). Once an association between a nonshared environmental factor and a developmental outcome has been identified via the MZ twin differences design, genetic and shared environmental factors can also subsequently be examined in research that expands to consider the role of gene-environment interactions in predicting developmental outcomes.

Notably, the MZ twin differences design is often recognized as the “gold standard” for investigating the effects of nonshared environmental influences (Asbury, Dunn, Pike, & Plomin, 2003; Caspi et al., 2004; Pike et al., 1996; Rovine, 1994; Vitaro et al., 2009). Further, the MZ twin differences design has been widely used across the research literature, including in recent investigations of nonshared environmental influences in child, adolescent, and adult samples (Alemany et al., 2013; Asbury et al., 2003; Barnes & Meldrum, 2015; Beaver, 2008; Burt, McGue, Iacono, & Krueger, 2006; Burt, McGue, Krueger, & Iacono, 2005; Dirghangi et al., 2015; Huppertz et al., 2014; Kendler & Gardner, 2001; Lewis, Collishaw, Thapar, & Harold,

2014; Pike, Reiss, Hetherington, & Plomin, 1996; Ritchie, Bates, & Plomin, 2015; Vidal-Ribas et al., 2015).

Methodological details regarding the MZ twin differences design (e.g., details regarding calculation of within-pair twin difference scores) are described in the Methods section below.

The MZ twin differences design and eating pathology. Use of the MZ twin differences design in eating pathology research is particularly beneficial for two primary reasons. First, a large body of research indicates moderate to large heritability of eating pathology, including both subclinical eating pathology symptoms and clinical eating disorder diagnoses (e.g., approximately 50%-80%; Bulik, 2005; Bulik et al., 2006; Klump, Suisman, et al., 2009). As such, genetically informed analyses such as those facilitated via an MZ twin differences design are critical when examining the development of eating pathology.

Second, specific nonshared environmental factors – including within the family – have been understudied in the field of eating pathology (Klump, Wonderlich, Lehoux, Lilenfeld, & Bulik, 2002). Rather, research in this area has traditionally conceptualized and examined environmental factors, including family factors, more broadly – i.e., as shared environmental factors. Although examining family factors as shared environmental factors has been important for better understanding broad risk factors that contribute to eating pathology in one family compared to another, it has been less helpful for understanding why two siblings growing up within the same family may differ in expression of eating pathology. By considering the parent-child relationship as a nonshared environmental factor, the current study may identify specific and amenable targets for eating pathology prevention and/or treatment research. For example, whereas shared environmental family factors such as socioeconomic status, parental employment, family structure, cultural beliefs, etc. may reflect a more entrenched family context

that is more difficult to target via intervention efforts, specific parenting practices may be more easily targeted via intervention and more amenable to change.

Study Overview

The current study was divided into five parts. Part 1 included the primary analyses that provided the basis of the study. Specifically, Part 1 sought to answer the following question:

Part 1: *Are twin differences in the parent-child relationship a risk factor for, or outcome of, twin differences in eating pathology across early and late adolescence?*

Parts 2-5 expanded on Part 1 to clarify findings from Part 1 and address additional methodological limitations of past research in this area. Specifically, Parts 2-5 sought to answer the following questions:

Part 2: *Is the strength of longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship a function of the severity of twin differences in eating pathology?*

Part 3: *Are longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship accounted for by twin differences in body mass index (BMI)?*

Part 4: *Are longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship unique to eating pathology or generalizable to other psychopathology?*

Part 5: *Do longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship differ based on the informant (i.e., twin versus parent) who rates the parent-child relationship?*

More specific details regarding Parts 1-5 are described below.

Part 1: Are twin differences in parent-child relationship problems a risk factor for, or outcome of, twin differences in eating pathology across early and late adolescence?

Part 1 of the current study sought to examine longitudinal, bidirectional associations between twin differences in eating pathology and twin differences in twin perceptions of the parent-child relationship across early and late adolescence. The purpose of these analyses was to examine whether differences in parent-child relationship variables predicted later differences in eating pathology, differences in eating pathology predicted later differences in parent-child relationship variables, or both. This task was accomplished through use of methodologically rigorous cross-lagged path models, as used by Spanos et al. (2010) and described further in the Methods section below. Examination of bidirectionality in research related to parenting and child/adolescent well-being has received increasing research attention over the past decade as such can provide important insight into the transactional effects of behavior-problem development (e.g., Pettit & Arsiwalla, 2008).

Parent-child relationship variables examined in the current study included parental involvement, parent regard for child, and child regard for parent. Parental involvement was conceptualized to relate to communication and closeness as well as parental monitoring and knowledge. Parent-child regard was conceptualized to involve love, respect, and acceptance of one another. As reviewed above, these parent-child relationship variables have been associated with eating pathology in some previous research (e.g., Ackard et al., 2006; Beato-Fernandez et al., 2004; Berge et al., 2014; Krug et al., 2014), although conclusions have been limited by methodological issues. Importantly, in this study the parent-child relationship was rated separately for the mother-child and father-child relationship, facilitating identification of differential associations between eating pathology and each unique parental relationship.

Only twin ratings of the parent-child relationship were examined in Part 1; see Part 5 for examination of parental ratings of the parent-child relationship.

Part 2: Is the strength of longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship a function of the severity of twin differences in eating pathology?

In Part 2 of the current study, we sought to expand upon findings in Part 1 to examine whether the strength of longitudinal associations observed in the full sample differed when examined in the twin pairs most discordant for eating pathology. Specifically, we re-ran cross-lagged analyses from Part 1 using only the subset of twin pairs who demonstrated the most discordance on eating pathology. Some previous research that has examined associations between twin differences in parent-child relationship variables and adolescent psychosocial outcomes has indicated that the strength of associations between variables is a function of the severity of twin differences (Asbury et al., 2003; Burt et al., 2006). Specifically, more robust associations between variables have been observed among twin pairs most discordant on psychosocial outcomes.

Notably, MZ twins have high concordance for eating pathology, in large part due to strong heritability of eating disorders (Bulik, Sullivan, & Kendler, 2000; Kortegaard, Hoerder, Joergensen, Gillberg, & Kyvik, 2001). Thus, samples with greater discordance provide more power to detect effects of the nonshared environment (Burt et al., 2006). Specifically, because MZ twins are genetically identical and, in the current study's sample, brought up within the same household environment, analyzing twin pairs with greater discordance on eating pathology scores facilitates a greater focus on nonshared environmental contributions to the development of eating pathology. Further, by examining twin pairs with greater discordance, twins who

demonstrate smaller discordance in eating pathology scores that may be attributable to measurement error (e.g., respondent bias, processing errors, mood, etc.) do not distort findings.[1]

Part 3: Are longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship accounted for by BMI?

In Part 3 of the current study, analyses examined whether associations between twin differences in eating pathology and twin differences in the parent-child relationship may be accounted for by BMI. Specifically, analyses considered whether associations between variables observed in cross-lagged analyses in Part 1 remained when BMI was regressed from eating pathology scores.

Some – but not all – research with children and adolescents indicates that BMI is concurrently (Erermis et al., 2004; Neumark-Sztainer, Story, Hannan, Perry, & Irving, 2002; Tanofsky-Kraff et al., 2004) and prospectively (e.g., Thompson, Coovert, Richards, Johnson, & Cattarin, 1995; Wojtowicz & von Ranson, 2012) associated with risk for eating pathology. Research also suggests associations between BMI and parent-child relationship variables, although results are inconsistent and often demonstrate small effect sizes (Berge, 2009; Schuetzmann, Richter-Appelt, Schulte-Markowt, & Schimmelmann, 2008; Pinquart, 2014). Thus, controlling for BMI among eating pathology scores is important to determine to what extent any associations between eating pathology and twin ratings of the parent-child relationship may be explained by BMI.

[1] Analyses focused on twin pairs who demonstrated the most discordance in eating pathology rather than twin pairs who demonstrated the most discordance in parent-child relationship variables, for several reasons. In addition to the reasons aforementioned, high discordance on parent-child relationship variables may reflect differential parental treatment, which implicates a somewhat separate construct that may confound findings. For example, in some research differential parental treatment has been uniquely associated with child and adolescent outcomes above the specific level or quality of parenting (Feinberg & Hetherington, 2001; Tamrouti-Makkink, Semon Dubas, Gerris, & van Aken, 2004).

Part 4: Are longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship unique to eating pathology or generalizable to other psychopathology?

In Part 4 of the current study we considered whether another form of psychopathology other than eating pathology demonstrated similar longitudinal associations with parent-child relationship variables. Specifically, analyses examined whether associations between variables observed in cross-lagged analyses in Part 1 were also observed when analyses were re-run replacing eating pathology scores with a measure of another form of psychopathology – specifically, anxiety symptoms. If similar associations were observed in these analyses as in Part 1, findings would indicate that associations with parent-child relationship variables observed in Part 1 are generalizable to other psychopathology rather than unique to eating pathology.

Available research inconsistently suggests associations between specific parent-child relationship variables and child and adolescent anxiety (Hudson, Doyle, & Gar, 2009; van Eijck, Branje, Hale III, & Meeus, 2012; Weich et al., 2009). For example, parental overcontrol has demonstrated some of the strongest associations with child and adolescent anxiety, whereas parent-child relationship variables such as parental warmth, criticism, and rejection have demonstrated weaker associations (Drake & Ginsburg, 2012).

Part 5: Do longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship differ based on the informant (i.e., twin versus parent) who rates the parent-child relationship?

Finally, in Part 5 of the current study we expanded upon Part 1 by examining the perspective of additional informants. Specifically, we re-ran analyses from Part 1 with maternal and paternal ratings of the parent-child relationship replacing twin ratings of the parent-child

relationship. The purpose of these analyses was to facilitate an understanding of differences and similarities between twin and parental perceptions of the parent-child relationship and the ways in which various perspectives are differentially associated with eating pathology.

Informant variability is consistently observed in psychological research, thus including multiple informants' perspectives is critical to more comprehensively understanding associations among variables. Multiple informants' perspectives is particularly critical to research regarding family relationships, as information from one family member reflects only that individual's perception of the family and various relationships. As aforementioned, multiple family member perspectives are necessary for a thorough understanding of the family environment and relationship dynamics (Carlson et al., 1991; Dekovic & Buist, 2005).

Methods

Participants

Participants included 446 female monozygotic (MZ) twins (i.e., 223 twin pairs) and their parents (i.e., 204 mothers, 99.5% [*n* = 203] of whom were biological mothers, and 170 fathers, 92.9% [*n* = 158] of whom were biological fathers) who participated in the Minnesota Twin Family Study (MTFS). Data were analysed from intake (approximately age 11 years) and two consecutive follow-up periods (approximately age 14 and 17 years).

Minnesota Twin Family Study. The MTFS is an ongoing population-based, longitudinal study that originated in 1991 and involves assessment at three-year intervals of same-sex twins from Minnesota and their parents (Iacono & McGue, 2002). Two age cohorts of female participants were recruited for the MTFS when twins were approximately age 11 and 17 years; only twins recruited in the younger cohort (i.e., age 11 years) were included in the current study to enable a focus on pre- to late adolescence.

Families were excluded from participation if the twins were adopted; if they did not live within a day's drive to the University of Minnesota's Twin Cities campus; or if one or more twins was deceased or had a mental or physical handicap that would impede completion of assessments. Twins were identified through birth records, and 90% of all twins born in Minnesota between 1971 and 1985 were successfully contacted by the MTFS. Of those contacted, 82.7% agreed to participate. Retention rates have subsequently been excellent: over 80% of the original sample was retained over an eight-year period.

At intake, there were no significant differences in rates of parental psychopathology or self-reported socioeconomic status between MTFS participants and other families in Minnesota with adolescent twins (Iacono, Carlson, Taylor, Elkins, & McGue, 1999). In addition, the ethnic composition of the MTFS sample is representative of the ethnic composition of Minnesota at the time of recruitment (over 95% Caucasian).

Zygosity determination. Participants were selected from a larger sample that included both MZ and dizygotic twins. Only data from MZ twins were used in the current study, to facilitate a monozygotic twin differences design that specifies nonshared environmental effects. Zygosity was determined by examining agreement among three independent measures. First, MTFS research staff provided ratings of the physical similarity between twins using face shape, ear shape, hair color, and eye color. Second, parents provided ratings of the physical similarity between twins using a zygosity questionnaire. Finally, data from the cephalic index, fingerprint ridge counts, and the ponderal index were aggregated by a specific algorithm. Any discrepancies among these three methods were resolved using serological procedures that examined 12 blood group antigens and protein polymorphisms (University of Minnesota, 2007).

Data Collection

Longitudinal data used in the current study were collected at assessments that extended across adolescence: i.e., at pre-adolescence (i.e., approximately age 11 years), middle adolescence (i.e., approximately age 14 years), and late adolescence (i.e., approximately age 17 years). As such, these data span two developmental periods – early adolescence (age 11-14 years) and later adolescence (age 14-17 years) – facilitating comparison of any changes in associations between variables across development.

Attrition and Missing Data

Of the 446 MZ twins included in the current study, self-report data were available for 86% and 79% of twins at age 14 and 17 years, respectively. Maternal reports of the mother-child relationship were available for 90% of twins at age 11 years, 82% of twins at age 14 years, and 74% of twins at age 17 years. Paternal reports of the parent-child relationship were available for 73% of twins at age 11 years and 48% of twins at age 17 years; no questionnaires relevant to the current study were administered to fathers at age 14 years. Researcher error contributed to some missing data, as some specific measures were inadvertently not administered to some participants at specific time points. In addition, some participants were missing responses for specific scale items. By convention, total scale and subscale scores were prorated if no more than 10% of the items were missing[2]; however, more sophisticated methods of handling missing data (e.g., maximum likelihood procedures, reviewed below in Data Analyses and Results) were used when more than 10% of items (including all items) were missing.

To explore any effect of attrition and missing data on findings, preliminary analyses used independent sample *t*-tests to compare variables of interest (i.e., eating pathology, parent-child

[2] The prorated score was computed by dividing the sum of scores for completed items by the number of completed items and subsequently multiplying the quotient by the total number of scale or subscale items.

relationship, and anxiety) at each time point between participants who had available data on the variable of interest at all three time points versus only one or two time points. Results indicated that there were almost no significant differences between groups on any variable measured at any time point. A few exceptions are described in Appendix A. Overall, analyses suggested that missing data associated with attrition were likely not a significant source of bias within the data set used in the current study.

Statistical Significance

Numerous statistical analyses were included across Parts 1-5 in the current study, as illustrated in the Data Analyses and Results section, below. Thus, the probability of type I error was elevated. However, the alpha level was retained at $p < .05$ for several reasons. For example, due to the exploratory nature of this research it was considered beneficial to be less conservative in interpreting results to avoid overlooking any possible associations. Moreover, cross-lagged models analysed in this research were already statistically rigorous (e.g., used twin difference scores, controlled for both stability and within-age associations, etc.). In addition, use of an alpha level of .05 is consistent with other studies that have performed cross-lagged models with twin difference scores (e.g., Burt et al., 2006; Spanos et al., 2010). However, the potential for type I error remains a limitation of this research that is elaborated further in the Discussion.

Measures

All measures are included in Appendix B. The internal consistency reliability (i.e., coefficient alpha) for each scale and subscale when administered to the current sample is listed in Appendix C.

Eating pathology. Eating pathology was assessed using the Minnesota Eating Behavior Survey (MEBS; von Ranson, Klump, Iacono, & McGue, 2005). The MEBS is a 30-item self-

report questionnaire developed to assess disordered eating attitudes and behaviours. It can be reliably administered to children as young as 10 years of age.

The MEBS provides a Total Score as well as four subscale scores. The subscales were extracted based on results from exploratory factor analyses and include: Body Dissatisfaction (i.e., discontent with body size and shape; 6 items), Weight Preoccupation (i.e., preoccupation with weight, eating, and dieting; 8 items), Binge Eating (i.e., binge eating, secretive eating, and preoccupation with food; 7 items), and Compensatory Behavior (i.e., use of, and thoughts of using, self-induced vomiting and other inappropriate compensatory behaviors to control weight; 6 items). Three items did not adequately load onto any of the four subscales thus only contributed to the Total Score. The MEBS does not specify a specific time frame, but all items are phrased in the present tense with the intent of assessing current pathology.

At age 11 years, the version of the MEBS administered included a two-point "true or false" response scale to provide a simple, dichotomous response format that was easy to understand. At age 14 and 17 years, the response format was expanded to a four-point scale (*Definitely True, Probably True, Probably False, Definitely False*) to capture a wider range of responses. However, to maintain consistency across assessment time points, the current study combined *Definitely True* and *Probably True* responses, and *Definitely False* and *Probably False* responses, at age 14 and 17 years. This method of data analysis is consistent with other studies that have examined MEBS data from the MTFS across these time points (e.g., Spanos et al., 2010; von Ranson et al., 2005).

Previous research with the larger MTFS sample indicates satisfactory internal consistency reliability of the MEBS. Internal consistency was lower among the behaviour-focused subscales – i.e., Binge Eating and Compensatory Behavior – than the attitude-focused subscales – i.e.,

Body Dissatisfaction and Weight Preoccupation (von Ranson et al., 2005). This finding is likely due the fact that behavioral indicators of eating pathology being are less prevalent than attitudinal indicators of eating pathology. The Compensatory Behavior subscale has demonstrated particularly low internal consistency (e.g., α = .40) and infrequent item endorsement in early adolescents (e.g., von Ranson et al., 2005; Klump, McGue, & Iacono, 2000). Thus, the Compensatory Behavior subscale was excluded from analyses in the current study.[3]

Three-year test-retest reliability of the MEBS has been described as modest. For example, from age 11 to 14 years, stability correlations suggest scores are reasonably stable over a three-year period: Total Score r = .59, Body Dissatisfaction r = .53, Weight Preoccupation r = .51, Binge Eating r = .32, and Compensatory Behavior r = .21 (all p's < .001; von Ranson et al., 2005). Adults report greater three-year stability on MEBS scores than adolescents, which aligns with the fact that adolescence presents a period of risk for eating pathology and susceptibility to triggers for developing eating pathology may vary across this developmental period. Both concurrent and criterion-related validity are supported (von Ranson et al., 2005).

MEBS scores were examined in Parts 1-3 and 5 of the current study.

Parent-child relationship. Perceptions of the parent-child relationship were measured via the Parental Environment Questionnaire (PEQ; Elkins et al., 1997). The PEQ is a 50-item self-report questionnaire that can be administered to children as young as 10 years of age. There are numerous informant variants of the PEQ: e.g., each twin reports on her relationship with each

[3] To support the decision to exclude the Compensatory Behavior subscale from analyses in the current study, preliminary analyses computed internal consistency within the current sample. Results supported previous findings: very low reliability was observed at age 11 years (i.e., α = .34), with somewhat higher, yet still questionable, reliability observed at age 14 (α =.70) and age 17 (α = .69) years.

parent separately and each parent reports on his/her relationship with each twin separately.[4] Items were essentially the same across the versions used for each informant, except wording was changed to suit the particular rater and the parent-child relationship being rated. For example, the twin rates "I don't feel close to my mother" when rating the mother-child relationship; the mother rates "This child doesn't seem to feel very close to me" when rating the mother-child relationship; etc. Each item is rated on a four-point Likert scale (*Definitely True, Probably True, Probably False, Definitely False*). The PEQ was not administered to fathers at the first follow-up assessment (i.e., age 14 years).[5] PEQ scales have been used in multiple studies that examined parent-child relationships during pre-adolescence and/or adolescence (e.g., Burt, Krueger, McGue, & Iacono, 2003; Shiner & Marmorstein, 1998; Wilson, Vaidyanathan, Miller, McGue, & Iacono, 2014).

The PEQ is composed of five scales: Conflict (i.e., the extent to which the parent-child relationship is characterized by disagreement, tension, and anger; 12 items), Involvement (i.e., the extent to which the parent-child relationship is characterized by communication [e.g., parental monitoring and knowledge], closeness, and support; 12 items), Regard for Parent (i.e., the extent to which the child is proud of and respects his/her parent; 8 items), Regard for

[4] As some families included biological parents and step-parents, twins rated the mother and father they lived with the most and the same parent provided ratings of the twin (I. Elkins, personal communication, 2014). For example, if a twin's biological parents were divorced and the twin lived most of the time with her biological mother and stepfather, the biological mother and stepfather would be rated by the twin and would likewise provide ratings of the twin. If a biological parent was deceased or otherwise unavailable (e.g., had lost contact with the twin), ratings for and from that parent were indicated as missing. In the current sample, of 204 mothers only one was a step-mother and the rest were biological mothers, and of 170 fathers only 12 were step-fathers and the rest were biological fathers.

[5] Although paternal ratings of his perception of the father-child relationship are not available at age 14, maternal ratings of her perception of the father-child relationship are available at this time-point (as well as at age 11 and 17 years). Preliminary analyses considered whether maternal ratings of the father-child relationship could be used in place of paternal ratings of the father-child relationship, to facilitate examination of the father-child relationship from a perspective other than the child. However, Pearson's correlations indicated weak and at times nonsignificant concurrent associations at age 11 and 17 years between maternal and paternal ratings of the father-child relationship. Such weak correlations imply poor convergent validity and suggest that maternal ratings of the father-child relationship are not an appropriate substitute for paternal ratings the father-child relationship. As such, the father's perception of the parent-child relationship at age 14 years was not examined in this study.

Daughter (i.e., the extent to which the parent is perceived to have high regard for the child; 5 items), and Structure (i.e., the extent to which the parent sets rules, monitors offspring compliance with rules, and believes that compliance is important; 5 items). Only the Involvement, Regard for Parent, and Regard for Daughter scales were used in the current study. These three scales have demonstrated adequate internal consistency across parent and child ratings (e.g., alpha = .62-.87; Elkins et al., 1997). Higher scores on these three scales indicate greater involvement, regard for parent, and regard for child, respectively. As PEQ scales relevant to both the mother-child relationship and father-child relationship are included in this study, to facilitate clarity the Involvement scale is hereafter referred to as either "Mother Involvement" or "Father Involvement," the Regard for Parent scale is hereafter referred to as either "Daughter Regard for Mother" or "Daughter Regard for Father," and the Regard for Daughter scale is hereafter referred to as either "Mother Regard for Daughter" or "Father Regard for Daughter."

The Conflict scale was not used in the current study because it has already been examined by Spanos et al. (2010) in a similar study with the MTFS sample that used cross-lagged analyses to explore associations between parent-child conflict and eating pathology, as aforementioned. In addition, the Structure scale was not used for several reasons. For example, the content was not perceived to be a good indicator of the parent-child relationship *quality* that the current study emphasized and some scale items (e.g., "It is important to me that this child obeys the law") appeared to reflect overall parental beliefs and family environment (i.e., shared environmental factors) more than the specific relationship unique to each parent-child dyad (i.e., nonshared environmental factors). In addition, and perhaps most critically, previous research has reported low internal consistency of this scale across age and informant ratings (i.e., alpha = .46-.58; Elkins et al., 1997). Due to poor reliability, this scale has also been excluded in other studies

that examined the PEQ (e.g., McGue et al., 2005). The MTFS eventually added another measure to better assess aspects of control and monitoring in the parent-child relationship (Stattin & Kerr, 2000). However, data for this measure are not available for the sample examined in the current study. Moreover, aspects of parental monitoring hypothesized to demonstrate associations with eating pathology were already examined by multiple items within the PEQ Involvement scale.

Whereas only twin ratings of the PEQ were examined in Parts 1-4 of the current study, parent ratings of the PEQ were additionally examined in Part 5.

Body mass index. BMI (i.e., one's body weight, measured in kilograms, divided by the square of one's height, measured in meters) was calculated from weight and height measurements taken by research staff using a level platform physician's scale.

BMI was only examined in Part 3 of the current study.

Anxiety. The State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) was administered as a measure of anxiety. The child version was administered at age 11 and 14 years, and the adult version was administered at age 17 years. The STAI is composed of two 20-item scales: the State Anxiety scale and the Trait Anxiety scale. Both scales are responded to via a Likert scale; the child version presents a three-point response scale, and the adult version presents a four-point response scale. Items and instructions on the child version were adapted from the adult version with adjustments to be developmentally appropriate.

The State Anxiety scale assesses state anxiety, which is conceptualized as a transitory emotional state or condition that is characterized by subjective, consciously perceived feelings of tension and apprehension, as well as a heightened autonomic nervous system. In contrast, the Trait Anxiety scale assesses trait anxiety, which is conceptualized as a relatively stable

individual difference in anxiousness or anxiety proneness. The two types of anxiety are believed to be related: trait anxiety is expressed through the tendency to regard situations as threatening and respond with state anxiety. In comparison to State Anxiety, Trait Anxiety assesses more chronic symptoms (Barnes, Harp, & Jung, 2002; Hedl Jr. & Papey, 1982; Spielberger et al., 1983).

Each participant completed the State Anxiety scale before a series of unfamiliar and potentially anxiety-provoking laboratory procedures that involved psychophysiological assessment. The Trait Anxiety scale was administered midway through the laboratory procedures. As the Trait Anxiety scale was not administered at age 14 years, only the State Anxiety scale was used in the current study. Previous research indicates the State scale can differentiate between children and adolescents who do and do not have an anxiety disorder (Seligman, Ollendick, Langley, & Baldacci, 2004) and can be used as a screener for concurrent anxiety disorders among adolescents (Hishinuma et al., 2001).

The STAI State Anxiety scale was only examined in Part 4 of the current study.

Within-Pair Twin Difference Scores

According to convention, within-pair twin difference scores were used in all cross-lagged analyses in the current study, creating an MZ twin differences design (Vitaro et al., 2009). Twin difference scores were used to isolate nonshared environmental effects from effects of genetic and shared environmental factors. To calculate twin difference scores, Twin B's score was subtracted from Twin A's score on each variable of interest (specifically, MEBS, PEQ, and STAI State scores at all ages). The sign (i.e., positive or negative) of twin difference scores was retained to establish the direction of differences within twin pairs (i.e., positive scores indicated Twin A scored higher on the variable of interest; negative scores indicated Twin B scored higher

on the variable of interest). It was necessary to retain the difference sign to confirm the direction of the association between differences in each variable.[6]

In an MZ twin differences design, the correlation between the two difference scores reflects the contribution of nonshared experiences to the creation of twin outcome differences, independent of either genetics or factors that differ between families (Rovine, 1994). This is because MZ twins are genetically identical and brought up within the same environment; thus, MZ difference scores reveal a direct index of nonshared environmental influence because they exclude genetic and shared environmental influence (Caspi et al., 200.4; Liang & Eley, 2005). Refer back to the Purpose section for further information regarding the MZ twin differences design.

Data Analyses and Results

Cross-lagged analyses were performed using Mplus version 7.2, a structural equation modelling program; all other analyses were performed using SPSS version 22.

PART 1:

Are twin differences in parent-child relationship problems a risk factor for, or outcome of, twin differences in eating pathology across early and late adolescence?

Part 1 Data Analyses

[6] For example, in one twin pair, Twin B may have greater eating pathology (e.g., MEBS = 5) and greater parent-child relationship quality (e.g., PEQ = 30) than Twin A (e.g., MEBS = 3 and PEQ = 25), creating a negative difference score for both eating pathology (i.e., MEBS = -2) and parent-child relationship quality (i.e., PEQ = -5). In contrast, in another twin pair, Twin B may have greater eating pathology (e.g., MEBS = 6) and lower parent-child relationship quality (e.g., PEQ = 23) than Twin A (e.g., MEBS = 2 and PEQ = 32), creating a negative difference score for eating pathology (i.e., MEBS = -4) and a positive difference score for the parent-child relationship quality (i.e., PEQ = 9). In this example, if the difference score sign is not retained, the data will falsely indicate a positive association between eating pathology and parent-child relationship quality in both twin pairs, despite the fact that the association is negative in the second twin pair.

Analyses in Part 1, the primary analyses of the current study, included (1) descriptive statistics, (2) cross-sectional and longitudinal Pearson's correlations, and (3) cross-lagged models.

Descriptive statistics. Descriptive statistics for twin ratings of PEQ (i.e., parental ratings were not included) and MEBS scores were computed. Both individual twin scores and twin difference scores were examined.

Paired-samples *t*-tests compared individuals' ratings of the mother-child relationship with ratings of the father-child relationship on each parenting variable at each time point, so as to examine whether the individual's perception of her relationship with her mother differed from her perception of her relationship with her father. All test assumptions were examined and addressed. For example, the assumption of independent cases was violated, as twin data are "clustered" due to familial-genetic characteristics that influence twins from the same pair to resemble each other. Analysing data from both members of a twin pair simultaneously would ignore twin correlations and thereby artificially reduce standard errors. Thus, only one twin from each pair was randomly selected to be included in each *t*-test.[7] In addition, the assumption of normality was examined by reviewing kurtosis and skewness values. The data were judged to be in violation of this assumption if the skewness was greater than +/- 2.0 and/or the kurtosis was greater than +/- 3. Variables that violated this assumption were transformed.[8]

[7] Although selecting only one twin per pair reduced the number of observations by half, an adequate number of observations (N = 233) remained that aligned with the number of observations included in twin difference score analyses. Further, the analyses (i.e., Pearson's correlations and paired samples *t*-tests) that used this analytic strategy of including only one twin per pair were considered descriptive and/or preliminary analyses that informed primary analyses (i.e., cross-lagged models), yet were not primary analyses themselves. Finally, this strategy of selecting one twin per pair to satisfy the assumption of independent cases has been used frequently throughout the literature, including within studies recently published in well-reputed, high-impact journals (e.g., Brown et al., 2014; Goldberg et al., 2013; Malenfant et al., 2012; Otowa et al., 2013; Vitaro et al., 2014).

[8] Data transformations were selected based on the type of skew reflected in each variable's distribution. To account for a negative skew, PEQ variables were transformed using log10(k-x), wherein k = maximum x value + 1. To account for a positive skew, MEBS variables were transformed using log10(x+1).

For all *t*-tests, a maximum likelihood estimator, the Expectation Maximization (EM) algorithm, was used to generate predicted values for the missing data. The EM algorithm attempts to predict the value that is the most likely to have been reported if the participant had responded, based on other data available (Graham, 2009). Further details regarding use of the EM algorithm are included in Appendix D.

Cross-sectional and longitudinal correlations. Exploratory Pearson's correlations were run using individual twin scores to examine within-individual cross-sectional and longitudinal associations between MEBS scores and twin ratings of PEQ scores, to confirm that a nonzero association existed between eating pathology and the twins' perception of the parent-child relationship. Prior to analyses, scatterplots were examined to ensure observed associations among variables analysed in Pearson's correlations followed a roughly linear, rather than curvilinear, relationship. Only one twin from each pair was randomly selected to be included in each analysis, so as to satisfy the assumption of independent cases (described above), and variables that violated the assumption of normality were transformed to approximate a normal distribution (using the same criteria described above).

Next, Pearson's correlations were repeated with twin difference scores replacing individual twin scores. These latter analyses were included to verify whether any associations between MEBS and PEQ individual twin scores remained when the parent-child relationship was isolated as a nonshared environmental factor (i.e., with both genetics and shared environment controlled). If associations were observed between variables only when individual twin scores were analysed, findings would suggest that associations were largely accounted for by genetic and/or shared environmental factors. For example, greater parent-child relationship problems may be associated with greater eating pathology due to factors such as genetic temperament,

overall family climate, cultural influences, etc., affecting both risk for parent-child relationship problems and risk for eating pathology. In contrast, if associations were also observed between variables when twin difference scores were analysed, findings would suggest that associations were, at least in part, accounted for by nonshared environmental factors. In other words, findings would suggest that twin differences in the parent-child relationship were associated with twin differences in eating pathology, regardless of any effects of genetic risk or shared environmental factors.

As difference score variables included positive, negative, and zero values, the sign (i.e., positive or negative) of difference scores was essential to retain to facilitate meaningful, interpretable correlations. Thus, any difference score variables that violated the assumption of normality were not transformed to approximate a normal distribution. As a result, analyses may include underestimated standard errors and statistical significance may be overestimated (i.e., increased likelihood of type I error).[9]

Cross-lagged models. Following methods implemented by Burt et al. (2006), cross-lagged models were fitted to the twin difference scores to explore prospective, reciprocal associations between eating pathology and twin ratings of the parent-child relationship (see Figure 1 [p. 44] for the model's structure). Specifically, separate cross-lagged models were performed to analyse associations among each PEQ scale (i.e., Mother Involvement, Father Involvement, Daughter Regard for Mother, Daughter Regard for Father, Mother Regard for Daughter, and Father Regard for Daughter) and each MEBS scale and subscale (i.e., Total, Body Dissatisfaction, Weight Preoccupation, and Binge Eating). Cross-lagged models have previously

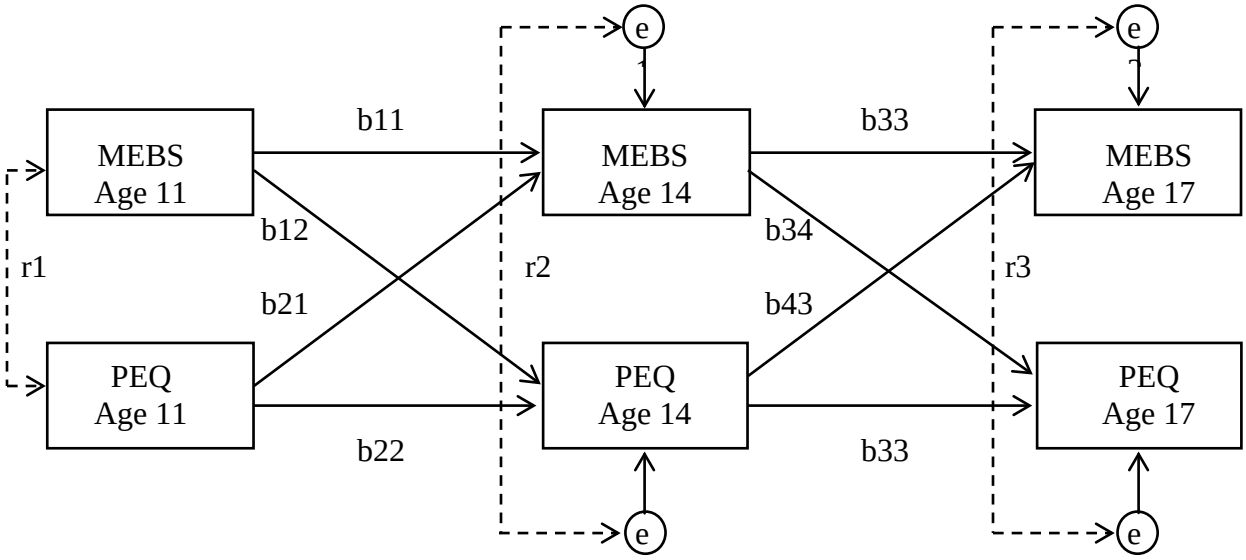
[9] Although violation of normality is not ideal, such violations have been reported to be less likely to cause major problems in drawing accurate and reliable results when large sample sizes are used (Pallant, 2007), such as in the current study. Further, examination of these Pearson's correlations was included as a preliminary analysis to support further analyses with more statistically rigorous cross-lagged models.

been used by other researchers to examine longitudinal associations among variables and to untangle both the direction of effects and changes in associations among variables across developmental periods (e.g., Marmorstein, Iacono, & Malone, 2010; Marmorstein, von Ranson, Iacono, & Malone, 2008; Spanos et al., 2010).

Cross-lagged models included each variable (i.e., PEQ and MEBS) at each time point (i.e., age 11, 14, and 17 years), with cross-lagged paths that indicated the association between one variable at an earlier time point and another variable at a later time point. In Figure 1, associations between eating pathology and parent-child relationship scores across time are indicated by the cross-lagged paths that connect MEBS at age 11 to PEQ at age 14 (b12), PEQ at age 11 to MEBS at age 14 (b21), MEBS at age 14 to PEQ at age 17 (b34), and PEQ at age 14 to MEBS at age 17 (b43). In addition, cross-lagged models included regression paths between the same variable across adjacent time points (e.g., PEQ at age 11, PEQ at age 14, and PEQ at age 17), which facilitated an estimate of the relative temporal stability (i.e., positional stability) of the construct across time (Burkholder & Harlow, 2003). In Figure 1, temporal stability of eating pathology and parent-child relationship scores across time is indicated by the regression paths that connect MEBS at age 11 to MEBS at age 14 (b11), PEQ at age 11 to PEQ at age 14 (b22), MEBS at age 14 to MEBS at age 17 (b33), and PEQ at age 14 to PEQ at age 17 (b44). Percentage of variance accounted for by each path can be obtained by squaring the path coefficient (i.e., standardized regression weight).

Further, cross-lagged models included a parameter to reflect associations between eating pathology and parent-child relationship scores within each time point. At age 11, this correlation is the Pearson correlation between the manifest variables (r1). Due to the inclusion of stability and cross-lagged coefficients between age 11 and 14 and age 14 and 17, these correlations

Figure 1. Structure of cross-lagged models examining longitudinal associations between eating pathology and the parent-child relationship.



Note. Partial regression coefficients are on the cross-age paths (b). Within-age correlations are illustrated by dotted lines (r). Due to the inclusion of stability and cross-lagged coefficients in the model, these within-age correlations function as residuals at ages 14 and 17 (e). Residuals reflect associations between MEBS and PEQ difference scores that are specific to the age at which they were measured and independent of any preexisting associations. MEBS = Minnesota Eating Behaviors Survey, PEQ = Parental Environment Questionnaire.

function as residuals or "innovations" associated with the manifest variables at age 14 (r2) and 17 (r3) – i.e., age-specific associations between eating pathology and parent-child relationship scores that were independent of any pre-existing association. Residual factors include effects not present at the earlier assessment point, other variables not assessed in the study, and error. Including correlated residuals of variables assessed concurrently at follow-up points is consistent with other research that has used cross-lagged models to examine associations among variables across time (e.g., Burt et al., 2006; Marmorstein et al., 2008; Spanos et al., 2010).

Importantly, this design facilitated examination of directional and reciprocal associations. In the current study, this means that two possibilities – i.e., (1) that the parent-child relationship prospectively predicted eating pathology, and (2) that eating pathology prospectively predicted the parent-child relationship – were examined. Cross-lagged models also controlled for pre-existing and within-age associations; thus, these models only examined associations between adjacent ages (i.e., 11 and 14 years, and 14 and 17 years) and ensured that results reflected across time associations only.

Mplus' MLR estimator was used in all cross-lagged analyses. This estimator provides maximum likelihood parameter estimates with standard errors and a chi-square test statistic that are robust to non-normality (Andreassen et al., 2006; Muthen & Muthen, 2010; Muthen & Asparouhov, 2002). Using MLR, standard errors are computed using a sandwich estimator.

A minimum of 10 cases to every parameter has been recommended to establish adequate power in path models (e.g., Kline, 2011). As such, the sample size was considered adequate in cross-lagged analyses of the full sample.

Missing data. Full information maximum likelihood (FIML) was used to accommodate missing data in analyses of cross-lagged models. FIML is a modern method of missing data

analysis that enables all available data points to be used in analyses, including for cases with some missing data. Thus, analyses were not restricted to completed cases, nor were missing data values arbitrarily replaced or imputed. This approach is more appropriate than deleting data casewise when only a minority of data is missing (e.g., less than 25%), as casewise deletion may lead to severe population selection bias.

As a result of using FIML when only a minority of data is missing, there is less bias than would be caused by analyzing data only for participants who completed all assessments; thus there can be greater confidence in the generalizability of results. The use of FIML for missing data is well-supported as a strong statistical method, including with structural and regression models (Enders, 2001; Enders & Bandalos, 2001). Simulation studies have indicated that FIML is appropriate when up to 25% of data is missing (Enders & Banadalos, 2001; Schlomer, Bauman, & Card, 2010). At 50% missing data, one simulation study indicated FIML produced regression coefficients that were biased by approximately 12% (Schlomer et al., 2010).

Model fit. Adequate model fit was determined by multiple test statistics: a non-significant chi-square value, a root mean square error of approximation (RMSEA) value of .05 or less, and a comparative fit index (CFI) value of .90 or greater (Byrne, 2012; Hu & Bentler, 1999; Kline, 2011). The chi-square test is considered the most basic fit index and is the traditional measure for evaluating overall model fit. It assesses the magnitude of discrepancy between the sample and fitted covariance matrices. A non-significant chi-square value indicates good model fit. The chi-square test is sensitive to sample size, such that the model may fit the data reasonably well yet the chi-square test may reject the model due to large sample size. Thus, alternative goodness-of-fit indices are important to supplement the chi-square statistic. RMSEA is an absolute fit index that measures the discrepancy between the hypothesized model and sample data while taking

into account the number of parameters. Finally, the CFI is an incremental fit index that measures the proportionate improvement in fit of the hypothesized model compared with a baseline model that assumes zero population covariances among observed variables (Byrne, 2012).

Part 1 Results

Descriptive statistics. Descriptive statistics for MEBS and twin ratings of PEQ scores (including individual and twin difference scores) at all ages are presented in Table 1 (p. 49). Absolute values of difference scores are presented to reflect the magnitude of differences within twin pairs.[10] Almost all subsequent statistical analyses were conducted on signed difference scores, so as to indicate the direction of any significant associations between variables.

As illustrated in Table 1 (p. 49), a range of severity of eating pathology was observed at each time point. Comparison of MEBS data to clinical cutoffs for eating pathology suggested a portion of the sample likely demonstrated clinical levels of eating problems. Clinical cutoffs were previously identified by von Ranson and colleagues (2005) by calculating the mean MEBS scores for subsamples of female adolescents who had met DSM-IV criteria for lifetime anorexia nervosa or bulimia nervosa (see Appendix E). These mean scores have been used as clinical cutoffs in previous research (e.g., Klump, Keel, Sisk, & Burt, 2010; Spanos et al., 2010), although it is possible that mean scores reflect an underestimate of participants with clinical pathology. In the current sample, 4% of twins scored at or above the clinical cutoff on the MEBS Total scale at age 11 years, whereas 8% of twins met this cutoff at age 14 and 17 years. In regards to MEBS subscales, 10%, 24%, and 28% of twins met the clinical cutoff on the Body Dissatisfaction subscale at age 11, 14, and 17 years, respectively; 7%, 8%, and 8% of twins met

[10] When descriptive statistics for signed difference scores were computed, the mean typically approached zero and lacked interpretive meaning. This is because, by chance, Twin A had the higher score in approximately half of twin pairs and Twin B had the higher score in approximately half of twin pairs, resulting in difference scores that were positive in approximately half of twin pairs and negative in approximately half of twin pairs.

the clinical cutoff on the Weight Preoccupation subscale at age 11, 14, and 17 years, respectively; and 14%, 11%, and 13% of twins met the clinical cutoff on the Binge Eating subscale at age 11, 14, and 17 years, respectively.

Paired samples *t*-tests indicated that, across adolescence, twins tended to rate the mother-child relationship as stronger than the father-child relationship. Specifically, Mother Involvement was rated as higher than Father Involvement at age 11 years ($t(222) = -7.43$, $p < .001$), age 14 years ($t(222 = -10.15$, $p < .001$), and age 17 years ($t(222) = -9.88$, $p < .001$); Daughter Regard for Mother was higher than Daughter Regard for Father at age 11 years ($t(222) = -4.64$, $p < .001$), age 14 years ($t(222) = -5.51$, $p < .001$), and age 17 years ($t(222) = -5.17$, $p < .001$), and Mother Regard for Daughter was higher than Father Regard for Daughter at age 14 years ($t(222) = -2.63$, $p < .01$) and age 17 years ($t(222) = -3.59$, $p < .001$). Mother Regard for Daughter was not rated significantly differently than Father Regard for Daughter at age 11 years, suggesting perceived differences in maternal and paternal regard may not emerge until middle adolescence. See Table 1 (p. 49) for mean scores.

Cross-sectional and longitudinal correlations. Within- and across-age correlations between MEBS and PEQ individual twin scores are presented in Table 2 (p. 53). As the overall pattern of results largely remained consistent regardless of whether or not data that violated the assumption of normality were transformed to approximate normality, results from analyses with untransformed data are presented to assist with interpretation. However, results from analyses with transformed data are presented in Appendix F. Within- and across-age correlations between MEBS and PEQ twin difference scores are presented in Table 3 (p. 55).

Table 1. Descriptive statistics for eating pathology and twin ratings of the parent-child relationship.

	Age 11					Age 14					Age 17				
	M	SD	Min.	Max.	*N*	M	SD	Min.	Max.	*N*	M	SD	Min.	Max.	*N*
Individual Twin Scores															
MEBS Total	5.43	4.73	0	21	414	5.63	5.64	0	23	385	6.20	5.59	0	24	352
MEBS Body Dissatisfaction	1.09	1.66	0	6	416	1.84	2.11	0	6	385	2.06	2.15	0	6	352
MEBS Weight Preoccupation	2.45	2.27	0	8	415	2.23	2.40	0	8	385	2.39	2.36	0	8	352
MEBS Binge Eating	1.05	1.32	0	7	415	.84	1.30	0	7	385	1.00	1.35	0	7	352
PEQ Mother Regard for Daughter	19.30	1.24	14	20	440	18.84	1.67	11	20	342	18.68	2.09	7	20	373
PEQ Father Regard for Daughter	19.12	1.67	5	20	437	18.68	2.02	6	20	336	18.42	2.39	6	20	366
PEQ Daughter Regard for Mother	30.06	2.05	23	32	439	28.93	3.23	14	32	342	28.89	3.44	13	32	372

PEQ Daughter Regard for Father	29.38	3.08	8	32	435	28.06	4.00	8	32	335	27.78	4.54	8	32	365
PEQ Mother Involvement	42.18	4.52	26	48	440	40.48	5.33	22	48	342	40.41	5.93	19	48	372
PEQ Father Involvement	40.32	5.56	12	48	436	37.70	6.40	15	48	335	36.86	7.91	12	48	365
Twin Difference Scores															
MEBS Total	3.40	2.94	0	16	198	3.76	3.85	0	19	183	3.87	4.12	0	24	162
MEBS Body Dissatisfaction	.93	1.24	0	6	200	1.44	1.52	0	6	183	1.30	1.51	0	6	162
MEBS Weight Preoccupation	1.74	1.59	0	7	199	1.66	1.72	0	7	183	1.88	1.90	0	8	162
MEBS Binge Eating	1.02	1.15	0	7	199	.91	1.18	0	5	183	.94	1.13	0	6	162
PEQ Mother Regard for Daughter	.97	1.29	0	6	217	1.17	1.46	0	7	166	1.45	1.91	0	11	176
PEQ Father Regard for Daughter	1.07	1.47	0	9	215	1.39	1.99	0	14	163	1.61	2.06	0	11	173
PEQ Daughter Regard for	1.97	1.61	0	8	216	2.17	2.09	0	8	166	2.24	2.53	0	17	175

Mother															
PEQ Daughter Regard for Father	2.14	2.16	0	14	213	2.79	3.18	0	22	162	2.58	2.79	0	17	172
PEQ Mother Involvement	4.12	3.44	0	15	217	3.67	3.12	0	16	166	4.21	3.82	0	22	175
PEQ Father Involvement	4.71	3.73	0	20	215	4.68	4.11	0	24	162	5.18	4.56	0	24	172

Note. The absolute values of twin difference scores are presented. MEBS = Minnesota Eating Behaviors Survey, PEQ = Parental Environment Questionnaire.

Effect sizes were small to medium. Individual twin correlations were generally stronger and more consistently observed within-age, although some significant across-age correlations were observed. In addition, the aspect of the parent-child relationship that tended to demonstrate the strongest and most consistent associations with eating pathology was the twin's rating of Mother/Father Regard for the Twin. Thus, specific indicators of eating pathology appeared to be associated with specific parent-child relationship problems, although associations tended to be concurrent and were not robust.

There were fewer significant correlations observed when twin difference scores were examined, suggesting that genetics and the shared environment contribute to associations between eating pathology and the parent-child relationship. The direction of significant associations was negative, indicating that the twin who reported lower parental regard for self, regard for parent, and involvement with parent was also more likely to report greater severity of eating pathology than her co-twin.

Almost all significant across-age correlations for both individual and twin difference scores were between adjacent ages (i.e., age 11 and 14 years, or age 14 and 17 years). There were no significant correlations between twin difference scores on the MEBS at age 11 and PEQ at age 17 or between the PEQ at age 11 and MEBS at age 17. These findings support the structure used in the cross-lagged models, wherein no cross-lagged path is included between variables at age 11 and age 17 years.

Cross-lagged models. Overall results from analyses of cross-lagged models for MEBS and twin ratings of PEQ difference scores are described below. Detailed results for these cross-lagged models can be reviewed in Appendix G. For cross-lagged models that included one or

Table 2. Within-twin Pearson correlations for eating pathology and twin ratings of the parent-child relationship (N = 223).

	Age 11				Age 14				Age 17			
	Total	BD	WP	Binge	Total	BD	WP	Binge	Total	BD	WP	Binge
PEQ Mother Regard for Daughter												
Age 11	-.25**	-.24**	-.17*	-.24**	-.20**	-.22**	-.15*	-.10	-.19*	-.19*	-.17*	-.08
Age 14	-.09	-.11	-.05	.02	-.16*	-.18*	-.07	-.15	-.05	-.07	-.04	.08
Age 17	-.04	.01	-.03	-.02	-.11	-.11	-.06	-.08	-.11	-.11	-.02	-.13
PEQ Father Regard for Daughter												
Age 11	-.25**	-.17*	-.17*	-.21**	-.20**	-.18*	-.18*	-.09	-.12	-.12	-.13	-.06
Age 14	-.04	-.05	-.03	.01	-.15	-.12	-.06	-.17*	.02	.04	-.00	.05
Age 17	-.05	-.05	-.01	-.09	-.19*	-.18*	-.20*	-.06	-.27**	-.18*	-.22**	-.24**
PEQ Daughter Regard for Mother												
Age 11	-.21**	-.14	-.17*	-.23**	-.20**	-.10	-.13	-.23**	-.24**	-.06	-.24**	-.27**
Age 14	-.02	-.03	.02	-.02	-.03	-.06	.05	-.02	-.04	-.14	-.04	.13
Age 17	-.01	.04	.01	-.01	-.04	-.05	.04	-.11	.03	.02	.08	-.04

PEQ Daughter Regard for Father												
Age 11	-.26**	-.14*	-.19**	-.22**	-.19**	-.12	-.14	-.18*	-.10	-.03	-.11	-.15
Age 14	-.01	-.02	-.03	.00	-.06	-.09	.01	.00	.08	.03	.06	.13
Age 17	-.01	.01	-.01	-.04	-.05	-.06	-.06	.00	-.05	-.01	-.07	-.04
PEQ Mother Involvement												
Age 11	-.10	-.09	-.06	-.15*	-.07	-.03	-.02	-.12	-10	.02	-.09	-.21**
Age 14	-.04	-.05	.00	.01	-.06	-.09	.06	-.13	-.04	-.08	.01	-.03
Age 17	-.05	-.01	-.02	-.05	-.14	-.12	-.04	-.19*	-.10	-.07	-.01	-.20**
PEQ Father Involvement												
Age 11	-.20**	-.12	-.16*	-.17*	-.13	-.11	.08	-.12	-.06	.00	-.05	-.16*
Age 14	-.08	-.06	-.08	-.01	-.19*	-.18*	-.08	-.21*	-04	.02	-.03	-.04
Age 17	-.05	-.01	-.06	-.07	-.19*	-.13	-.16*	-.14	-.15	-.04	-.13	-.22**

Note. Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, PEQ = Parental Environment Questionnaire. *$p < .05$, **$p < .01$.

Table 3. Within-pair twin difference score Pearson correlations for eating pathology and twin ratings of the parent-child relationship (N = 223).

	Age 11				Age 14				Age 17			
	Total	BD	WP	Binge	Total	BD	WP	Binge	Total	BD	WP	Binge
PEQ Mother Regard for Daughter												
Age 11	-.15*	-.20**	-.09	-.05	-.11	-.05	-.12	-.06	-.07	-.06	-.09	.05
Age 14	-.10	-.11	-.08	-.02	-.19*	-.19*	-.12	.00	-.04	-.02	-.07	.11
Age 17	-.04	-.01	-.02	-.01	-.14	-.11	-.11	-.03	-.23**	-.26**	-.18*	-.09
PEQ Father Regard for Daughter												
Age 11	-.14	-.20**	-.05	-.03	-.14	-.04	-.17*	-.08	-.07	-.06	-.12	.06
Age 14	.02	-.07	.01	.05	-.13	-.12	-.03	-.10	-.03	-.07	-.08	-.03
Age 17	-.05	-.06	-.03	-.02	-.16	-.02	-.12	-.12	-.28**	-.20*	-.20*	-.23**
PEQ Daughter Regard for Mother												
Age 11	-.21**	-.05	-.18*	-.23**	-.11	-.05	-.02	-.16*	-.03	.03	-.03	-.02
Age 14	-.06	.03	-.09	-.10	-.14	-.21**	-.08	-.11	-.04	-.13	-.09	.01
Age 17	-.09	-.01	.00	-.11	-.11	-.11	-.05	-.10	-.07	-.09	-.06	.00

PEQ Daughter Regard for Father												
Age 11	-.22**	-.09	-.15*	-.25**	-.10	-.04	-.01	-.16*	-.02	.01	-.03	-.02
Age 14	.00	-.06	-.04	.00	-.09	-.14	.04	-.08	-.06	-.08	-.07	.00
Age 17	-.07	-.06	.02	-.04	-.14	-.05	-.12	-.11	-.06	-.06	-.02	-.05
PEQ Mother Involvement												
Age 11	-.07	.03	-.13	-.06	-.09	-.05	-.04	-.01	-.02	.03	-.03	.03
Age 14	-.04	-.03	-.05	-.02	-.07	-.17*	.02	.04	.01	-.07	.01	.07
Age 17	.00	-.01	.02	.00	-.12	-.09	-.02	-.11	-.19*	-.17*	-.14	-.14
PEQ Father Involvement												
Age 11	-.14	-.03	-.15*	-.09	-.10	-.06	-.02	-.07	-.02	-.02	-.04	.03
Age 14	-.05	-.11	-.04	-.02	-.11	-.15	-.01	-.13	.01	.04	-.05	.03
Age 17	-.06	-.09	-.02	-.05	-.19*	-.01	-.11	-.24**	-.20*	-.08	-.14	-.24**

Note. Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, PEQ = Parental Environment Questionnaire. *$p < .05$, **$p < .01$.

more significant cross-lagged path coefficient, the results are additionally displayed in Figures 2-4 below (p. 64-66). All paths referenced below are labelled in Figure 1 (p. 44).

Model fit. Almost all of the cross-lagged models provided adequate fit to the data, as evidenced by non-significant chi-square values, RMSEA values equal or close to zero, and CFI values of .90 or higher. However, there were a few exceptions. In particular, cross-lagged models that included MEBS Body Dissatisfaction scores demonstrated generally poor fit. In addition, a specific model that examined associations between Binge Eating and Father Regard for Daughter demonstrated poor fit. A few factors may have affected such poor fit. For example, Body Dissatisfaction and Binge Eating demonstrated the smallest variability in twin difference scores of any of the MEBS subscales and Mother/Father Regard for Daughter demonstrated the smallest variability in difference scores of any of the PEQ scales (see Table 1, p. 49). Thus, it is possible that model fit was limited by lack of variability within subscale/scale twin difference scores. In addition, as previous research indicated the Body Dissatisfaction subscale is the most stable MEBS subscale across time (e.g., von Ranson et al., 2005), it is possible that model fit was affected by exclusion of longitudinal paths that accounted for stability of Body Dissatisfaction from age 11 to 17 years. Such stability paths were included when examining body dissatisfaction across three time points in a similar longitudinal, cross-lagged model in another study, wherein adequate model fit was reported (Crespo et al., 2010).

As such, additional analyses were run to explore poor model fit in a subset of cross-lagged models. Specifically, to examine the latter hypothesis, analyses were re-run on cross-lagged models that included MEBS Body Dissatisfaction, wherein an additional longitudinal path was included between Body Dissatisfaction at age 11 years and Body Dissatisfaction at age

17 years. Results indicated the additional longitudinal path was significant in all models (*p*s < .05); improved model fit in all models; and did not change the statistical significance of any cross-lagged path in any model. These findings suggest the association between Body Dissatisfaction at pre- and late-adolescence was not completely mediated by Body Dissatisfaction at middle adolescence; rather, pre-adolescence Body Dissatisfaction predicts important variance in late-adolescence Body Dissatisfaction. Thus, the extent to which Body Dissatisfaction at pre-adolescence predicted Body Dissatisfaction at late adolescence, irrespective of Body Dissatisfaction at middle adolescence, appeared important to recognize to best understand associations between eating pathology and the parent-child relationship.

Stability. For both the MEBS and PEQ, twin difference scores were more stable during later adolescence (paths b33 and b44) than early adolescence (paths b11 and b22). Specifically, MEBS Total difference scores at age 11 and 14 accounted for up to 17% and 22%, respectively, of the variance in the same construct three years later; Body Dissatisfaction difference scores at age 11 and 14 accounted for up to 6% and 14%, respectively, of the variance in the same construct three years later; Weight Preoccupation difference scores at age 11 and 14 accounted for up to 9% and 21%, respectively, of the variance in the same construct three years later; and Binge Eating difference scores at age 11 and 14 accounted for up to 3% and 7%, respectively, of the variance in the same construct three years later. By comparison, Mother Regard for Daughter difference scores at age 11 and 14 accounted for 0% and up to 7%, respectively, of the variance in the same construct three years later; Father Regard for Daughter difference scores at age 11 and 14 accounted for up to 1% and 5%, respectively, of the variance in the same construct three years later; Daughter Regard for Mother difference scores at age 11 and 14 accounted for up to 1% and 8%, respectively, of the variance in the same construct three years later; Daughter

Regard for Father difference scores at age 11 and 14 accounted for up to 1% and 14%, respectively, of the variance in the same construct three years later; Mother Involvement difference scores at age 11 and 14 accounted for up to 3% and 14%, respectively, of the variance in the same construct three years later; and Father Involvement difference scores at age 11 and 14 accounted for up to 3% and 8%, respectively, of the variance in the same construct three years later. Overall, comparison of MEBS and PEQ stability coefficients emphasize that differences within twin pairs in eating pathology (particularly, overall levels of eating pathology) are more likely to persist into later adolescence, as compared to differences within twin pairs in ratings of the parent-child relationship. However, overall low percentage of variance accounted for across time suggests that many other factors are associated with change in twin differences in these constructs across time, particularly during the early adolescent period.

Concurrent associations. Overall, significant within-age correlations were strongest between MEBS and PEQ difference scores at age 11 years (r1) and age 17 years (r3). .[11] Associations indicated an inverse association, wherein the twin who reported greater eating pathology also reported lower parent-child relationship quality. Thus, new sources of influences specific to middle adolescence did not appear to strengthen the initial association observed between eating pathology and the parent-child relationship at pre-adolescence. In contrast, new

[11] Cross-lagged models in Part 1 were re-run without including correlated residuals at age 14 and 17 years. As aforementioned, correlated residuals reflected age-specific associations between eating pathology and parent-child relationship scores that were independent of any pre-exiting association. Overall, the pattern of significant findings and model fit remained the same when these residuals were specified to not be correlated. The fit of models that examined Body Dissatisfaction continued to be poorer than most other models and improved when a longitudinal path was included between Body Dissatisfaction at age 11 and 17 years. However, in models that examined associations between Father Regard for Daughter and either Total eating pathology or Binge Eating, model fit declined compared to models with these variables that included correlated residuals. Such decreased fit was likely due to significant correlations between residuals at age 17, emphasizing the importance of recognizing new sources of influence specific to late adolescence that contributed to inverse concurrent associations between eating pathology and the parent-child relationship.

sources of influences specific to late adolescence did appear to contribute to inverse associations between eating pathology and the parent-child relationship as adolescents grew older

Longitudinal associations. No significant cross-lagged paths were observed in any of the models that examined associations between any of the MEBS difference scores and Daughter Regard for Mother/Father difference scores. However, there were some significant cross-lagged paths in models that examined various MEBS difference scores and either Mother/Father Regard for Daughter or Mother/Father Involvement difference scores. Specifically, twin differences in MEBS Total (see Figure 2, p. 64) and Binge Eating (see Figure 3, p. 65) at age 14 both predicted twin differences in Father Involvement at age 17, with the direction of associations indicating that the twin who reported greater total eating pathology or greater binge eating later reported lower father involvement. In addition, twin differences in Father Regard for Daughter at age 11 predicted twin differences in Weight Preoccupation at age 14 (see Figure 4, p. 66), such that the twin who reported lower perceived father regard later reported greater preoccupation with her weight.

The three cross-lagged models that indicated a significant cross-lagged path (see Figures 2-4) were rerun excluding any twin pair wherein the rearing father was someone other than the biological father (e.g., stepfather) at any time-point. 31 cases were removed and 192 twin pairs remained. In these revised analyses, the cross-lagged path between Binge Eating at age 14 and Father Involvement at age 17 remained significant ($p < .05$). Neither the cross-lagged path between MEBS Total at age 14 and Father Involvement at age 17 ($p = .19$) nor the cross-lagged path between Father Regard for Daughter at age 11 and Weight Preoccupation at age 14 ($p = .11$) reached statistical significance, although results trended in the same direction. The latter results likely reflect the reduced sample size; however, they may also suggest associations among

variables differ dependent on whether one's relationship with a biological father or non-biological father is rated.

Summary. Overall, cross-lagged models indicated that longitudinal associations between twin differences in eating pathology and twin differences in parent-child relationship variables were neither strong nor robust. In particular, neither twin ratings of their regard for their mother nor twin ratings of their regard for their father were associated with eating pathology across time. However, where significant cross-lagged paths emerged, results suggested that the direction of association between variables shifted between early adolescence and later adolescence. Specifically, lower perceived father regard at pre-adolescence predicted greater eating pathology – specifically, greater weight preoccupation – at middle adolescence. In contrast, greater eating pathology – in particular, binge eating – at middle adolescence predicted lower father involvement at late adolescence. However, some associations no longer met statistical significance when twin ratings of non-biological fathers were excluded from analyses.

Part 2 expanded on these findings to consider whether any differences would be observed in the strength of longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship when examined within the subsample of twin pairs most discordant on eating pathology. As aforementioned, some previous research that has examined twin differences in parent-child relationship variables and other adolescent psychosocial outcomes have indicated that associations between variables are a function of the severity of twin differences. Specifically, more robust associations were observed among twin pairs most discordant on psychosocial outcomes (Asbury et al., 2003; Burt et al., 2006).

PART 2:

Is the strength of longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship a function of the severity of twin differences in eating pathology?

Part 2 Data Analyses

After analysing cross-lagged models with the full sample in Part 1, analyses in Part 2 involved re-running cross-lagged analyses using only the twin pairs most discordant on eating pathology.

Discordant subsamples. To select the subsamples of twin pairs most discordant on eating pathology, the mean amount of discordance for each twin pair across the three time points (i.e., age 11, 14, and 17 years) was calculated for each MEBS scale. Subsequently, a median split was performed. Only twin pairs that scored at or above the median for amount of discordance on each MEBS scale were included in analyses of that scale. This cutoff point was selected to balance selection of a subsample that was large enough to facilitate statistical testing and also demonstrated ample magnitude of discordance.

Cross-lagged models. Cross-lagged models examined in Part 1 were re-run using only the discordant samples (as defined above). Analyses followed the same procedure outlined in Part 1: the model is presented in Figure 1 (p. 44); the MLR estimator was used to provide maximum likelihood parameter estimates with standard errors and a chi-square test statistic that are robust to non-normality; FIML was used to accommodate missing data; and adequate model fit was determined by a non-significant chi-square value, RMSEA value of .05 or less, and CFI values of .90 or greater.

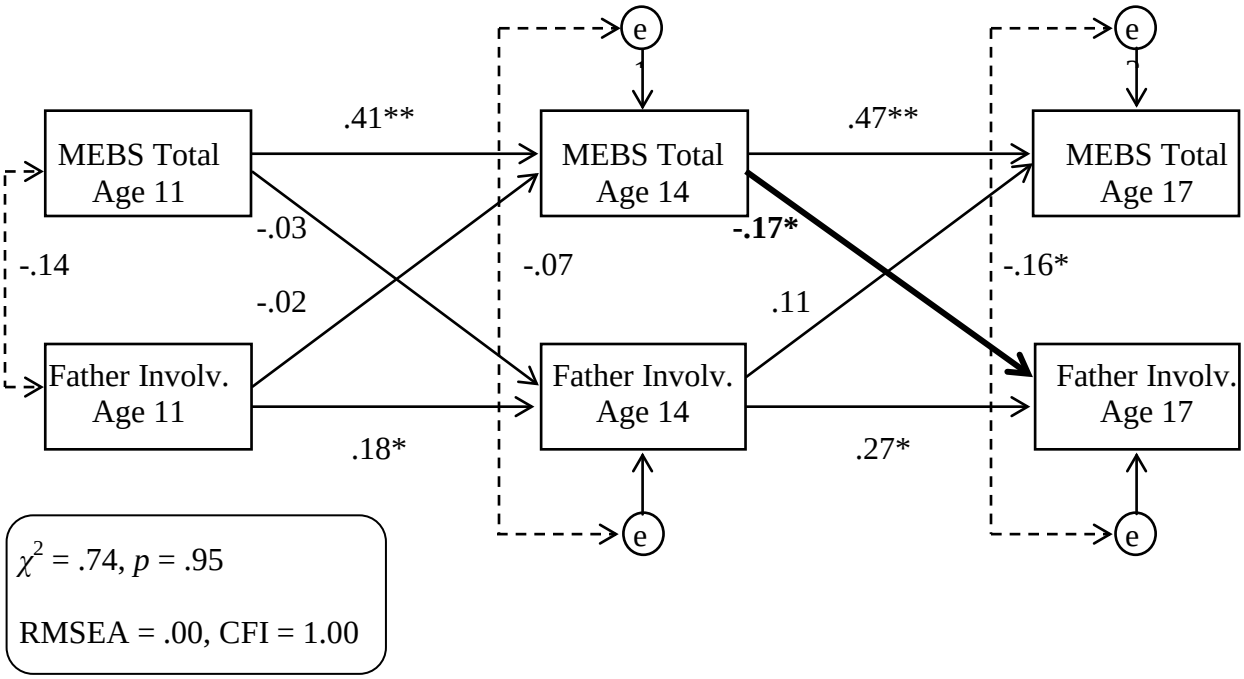
Part 2 Results

Discordant subsamples. Each discordant subsample included approximately half of the total sample (e.g., 111 twin pairs) by selecting only twin pairs that scored at or above the median for discordance on each MEBS scale. Based on the number of parameters in the models (i.e., 17), the size of these discordant subsamples was less than recommended for adequate statistical power (i.e., 10 cases to every parameter, for a total of 170 cases; Kline, 2011). However, as the size of these subsamples was similar to the size of discordant samples used in other cross-lagged analyses that identified significant effects (e.g., Burt et al., 2006), and use of discordant twin pairs was considered to increase power to detect nonshared environmental effects, analyses were run with these subsamples. However, results should be interpreted with caution, recognizing that the analyses may be underpowered.

Cross-lagged models. Overall results from analyses of cross-lagged models for MEBS and twin ratings of PEQ difference scores using the discordant subsamples are described below. Detailed results for these cross-lagged models can be reviewed in Appendix H. For cross-lagged models that included one or more significant cross-lagged path coefficient, the results are additionally displayed in Figures 5-11 (p. 72-78) below. All paths referenced below are labelled in Figure 1 (p. 44).

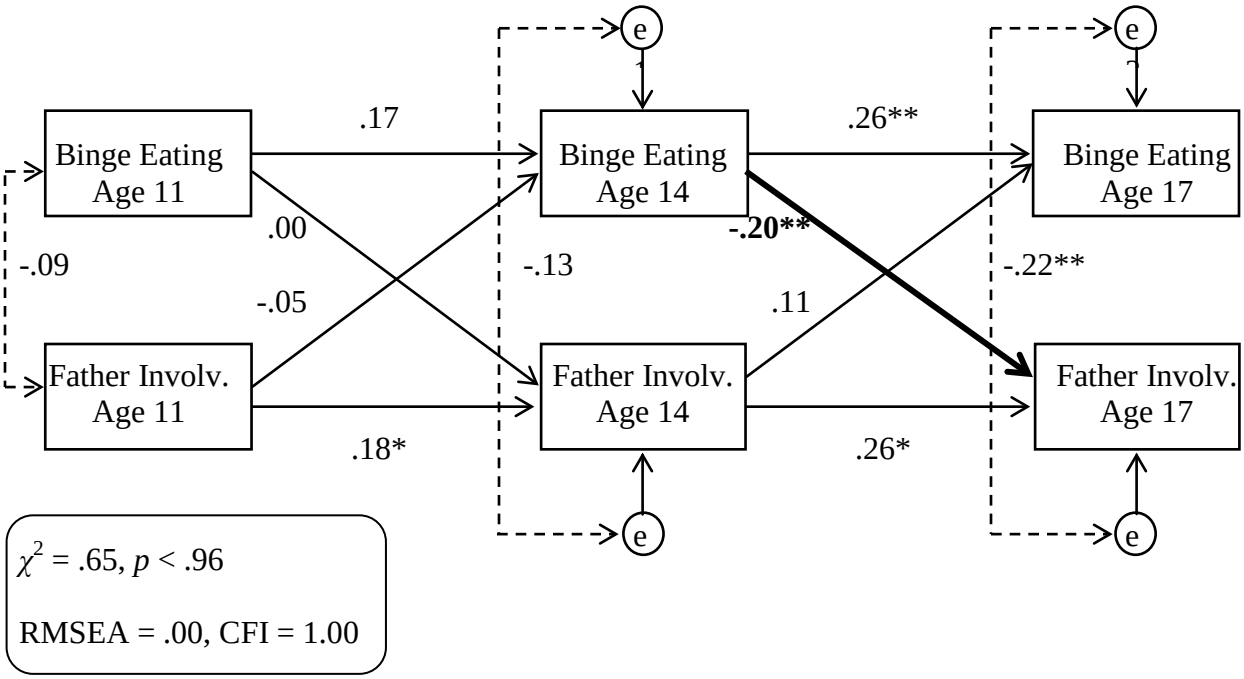
Model fit. As in analyses of the full sample, model fit tended to be adequate, with some exceptions. In particular, models that included the Body Dissatisfaction subscale tended to demonstrate poor fit. Thus, as described above with the full sample, analyses of models that included the Body Dissatisfaction subscale were re-run with an additional longitudinal path included between Body Dissatisfaction at age 11 years and Body Dissatisfaction at age 17 years. The additional longitudinal path was significant in all models ($ps < .05$); improved model fit

Figure 2. Cross-lagged model of Total eating pathology and twin ratings of Father Involvement.



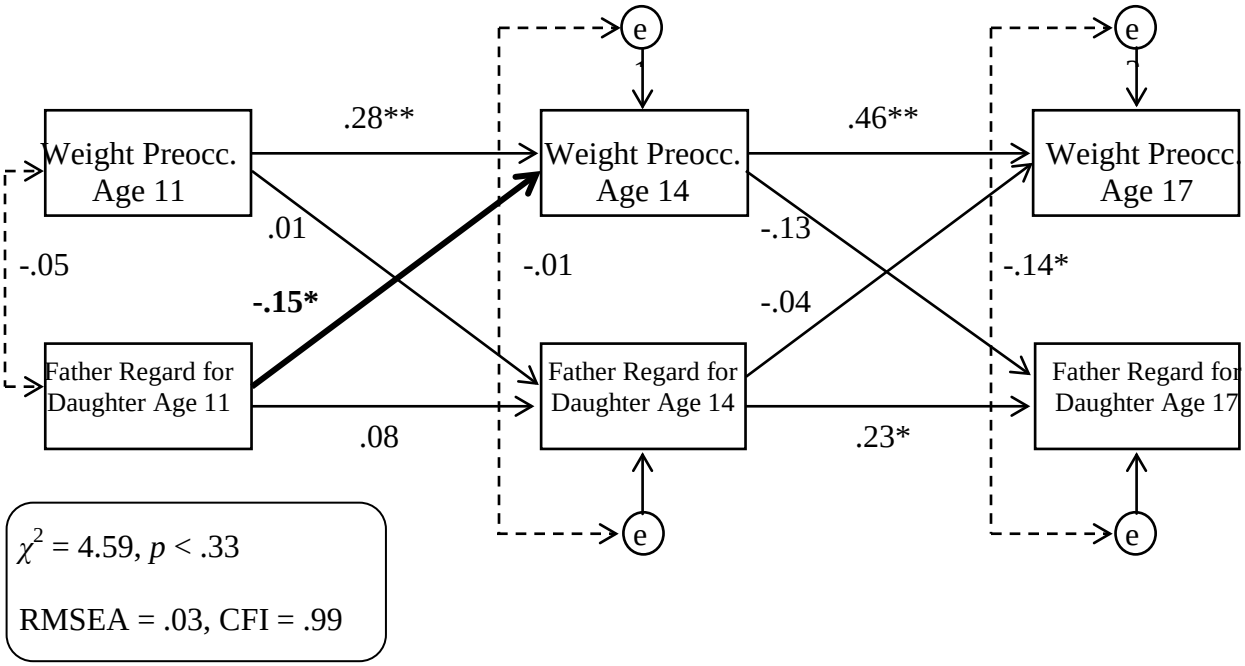
Note. The above model indicates that twin differences in MEBS Total at age 14 predicted twin differences in twin ratings of Father Involvement at age 17 years. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. MEBS = Minnesota Eating Behaviors Survey, Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 3. Cross-lagged model of Binge Eating and twin ratings of Father Involvement.



Note. The above model indicates that twin differences in Binge Eating at age 14 predicted twin differences in twin ratings of Father Involvement at age 17. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 4. Cross-lagged model of Weight Preoccupation and twin ratings of Father Regard for Daughter.



Note. The above model indicates that twin differences in twin ratings of Father Regard for Daughter at age 11 predicted twin differences in Weight Preoccupation at age 14. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Preocc. = Preoccupation, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

across all models; and did not change the statistical significance of any cross-lagged path in any model.

Stability. As in analyses of the full sample, analyses of the discordant sample indicated both MEBS and PEQ difference scores were more stable during later adolescence (paths b33 and b44) than early adolescence (paths b11 and b22). In regards to MEBS scores, MEBS Total difference scores at age 11 and 14 accounted for up to 19% and 26%, respectively, of the variance in the same construct three years later; Body Dissatisfaction difference scores at age 11 and 14 accounted for up to 15% and 16%, respectively, of the variance in the same construct three years later; Weight Preoccupation difference scores at age 11 and 14 accounted for up to 11% and 25%, respectively, of the variance in the same construct three years later; and Binge Eating difference scores at age 11 and 14 accounted for up to 5% and 7%, respectively, of the variance in the same construct three years later. In regards to PEQ scores, Mother Regard for Daughter difference scores at age 11 and 14 accounted for up to 3% and 9%, respectively, of the variance in the same construct three years later; Father Regard for Daughter difference scores at age 11 and 14 accounted for up to 1% and 10%, respectively, of the variance in the same construct three years later; Daughter Regard for Mother difference scores at age 11 and 14 accounted for up to 6% and 15%, respectively, of the variance in the same construct three years later; Daughter Regard for Father difference scores at age 11 and 14 accounted for up to 1% and 30%, respectively, of the variance in the same construct three years later; Mother Involvement difference scores at age 11 and 14 accounted for up to 5% and 17%, respectively, of the variance in the same construct three years later; and Father Involvement difference scores at age 11 and 14 accounted for up to 3% and 17%, respectively, of the variance in the same construct three years later.

Concurrent associations. The strength of within-age correlations in the discordant sample was strongest between STAI State and PEQ difference scores at age 11 years (r1) and age 17 years (r3), and weakest at age 14 years (r2). Observed associations indicated the twin who reported greater eating pathology also reported lower parent-child relationship quality. Thus, as in analyses of the full sample in Part 1, new sources of influences specific to middle adolescence did not appear to strengthen the initial association observed between eating pathology and the parent-child relationship at pre-adolescence, although new sources of influences specific to late adolescence did appear to contribute to inverse associations between eating pathology and the parent-child relationship as adolescents grew older.

Longitudinal associations. In analyses of cross-lagged models using the discordant sample, all significant cross-lagged paths observed within the full sample (described above and displayed in Figures 2-4, p. 64-66) remained significant (see Figures 5-7, p. 72-74). That is, MEBS Total and Binge Eating at age 14 predicted twin ratings of Father Involvement at age 17, and twin ratings of Father Regard for Daughter at age 11 predicted Weight Preoccupation at age 14. In addition, the strength of associations tended to be stronger and some additional cross-lagged paths became significant. Specifically, whereas in the full sample twin differences in MEBS Total at age 14 only predicted twin differences in Father Involvement at age 17, in the discordant sample twin differences in MEBS Total at age 14 additionally predicted twin differences in Father Regard for Daughter (see Figure 8, p. 75) and Daughter Regard for Father (see Figure 9, p. 76) at age 17. The direction of association remained the same: i.e., the twin who reported greater eating pathology later reported lower father-child regard. Likewise, whereas in the full sample twin differences in Binge Eating at age 14 only predicted twin differences in Father Involvement at age 17, in the discordant sample twin differences in Binge Eating at age

14 additionally predicted twin differences in Mother Involvement at age 17 (see Figure 10, p. 77). Again, the direction of association remained the same: i.e., the twin who reported greater binge eating later reported lower mother involvement. In addition, in the discordant sample twin differences in MEBS Total at age 11 predicted twin differences in Mother Regard for Daughter at age 14 (see Figure 11, p. 78) and twin differences in Weight Preoccupation at age 14 predicted twin differences in Father Regard for Daughter at age 17 (see Figure 7, p. 74). The direction of associations indicated that the twin who reported greater eating pathology later reported lower perceived mother regard and the twin who reported greater weight preoccupation later reported lower perceived father regard.

Overall, associations between twin differences in eating pathology and twin differences in their ratings of the parent-child relationship were stronger and observed across more parenting constructs when examined within the discordant sample, as compared to the full sample. These findings suggest that larger, more clinically significant differences in eating pathology – as compared to smaller, less clinically significant differences in eating pathology – are more likely to predict differences in the parent-child relationship.

Summary. Associations between twin differences in eating pathology and twin differences in ratings of the parent-child relationship were more robust within the discordant sample than the full sample. In particular, consistent negative associations were observed within the discordant subsample between overall levels of eating pathology at middle adolescence and various aspects of the father-child relationship at late adolescence. In addition, negative associations were observed between eating pathology and daughters' later perceptions of mother regard and involvement. These finding suggest associations between twin differences in eating

pathology and the parent-child relationship may be a function of the severity of twin differences in eating pathology.

Having uncovered some associations between twin differences in eating pathology and twin differences in specific parent-child relationship variables across Parts 1 and 2, Part 3 expanded on these findings to consider whether associations between variables could be accounted for by BMI.

PART 3:

Are longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship accounted for by body mass index?

Part 3 Data Analyses

Analyses in Part 3 involved re-running cross-lagged models from Part 1 with BMI regressed out from eating pathology scores. The full sample was included in analyses.

Controlling for BMI. Revised MEBS scores were computed that controlled for any association between eating pathology and BMI. Specifically, linear regression analyses were used to regress out BMI from each twin's MEBS scores at all ages. Standardized residuals were then retained as an indicator of eating pathology that was not accounted for by BMI. Revised MEBS twin difference scores were then calculated by subtracting Twin B's revised MEBS score from Twin A's revised MEBS score at all ages.

Cross-lagged models. To examine to what extent, if any, associations between twin ratings of the parent-child relationship and eating pathology demonstrated in Part 1 were accounted for by BMI, cross-lagged models examined in Part 1 were run using these revised MEBS scores that controlled for associations with BMI (see Figure 1 [p. 44] for the model's structure). Specifically, analyses followed the same procedure outlined in Part 1, with one

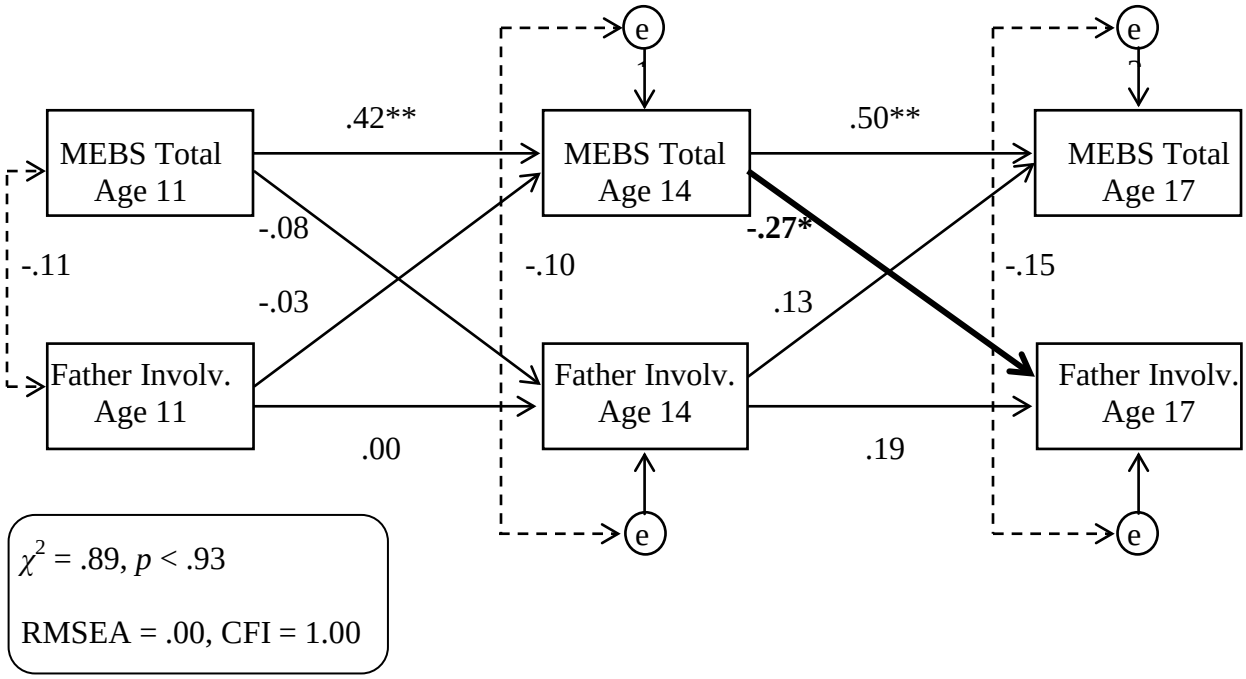
exception: revised MEBS scores that controlled for associations with BMI replaced original MEBS scores. The MLR estimator was used to provide maximum likelihood parameter estimates with standard errors and a chi-square test statistic that are robust to non-normality; FIML was used to accommodate missing data; and adequate model fit was determined by a non-significant chi-square value, RMSEA value of .05 or less, and CFI values of .90 or greater.

Part 3 Results

Cross-lagged models. Detailed results from analyses of cross-lagged models that included MEBS differences scores (calculated after BMI was regressed from each twin's score) and twin ratings of PEQ difference scores can be reviewed in Appendix I. Comparison of these analyses to those in Part 1 indicated whether cross-lagged associations between eating pathology and the parent-child relationship reported in Part 1 may be accounted for by BMI. Results indicated the models adequately fit the data and the pattern of significant cross-lagged paths was unchanged from Part 1. Specifically, only models that examined associations between MEBS Total and twin ratings of Father Involvement, Binge Eating and twin ratings of Father Involvement, and Weight Preoccupation and twin ratings of Father Regard for Daughter demonstrated a significant cross-lagged path coefficient. These results are displayed in Figures 12-14 (p. 80-82).

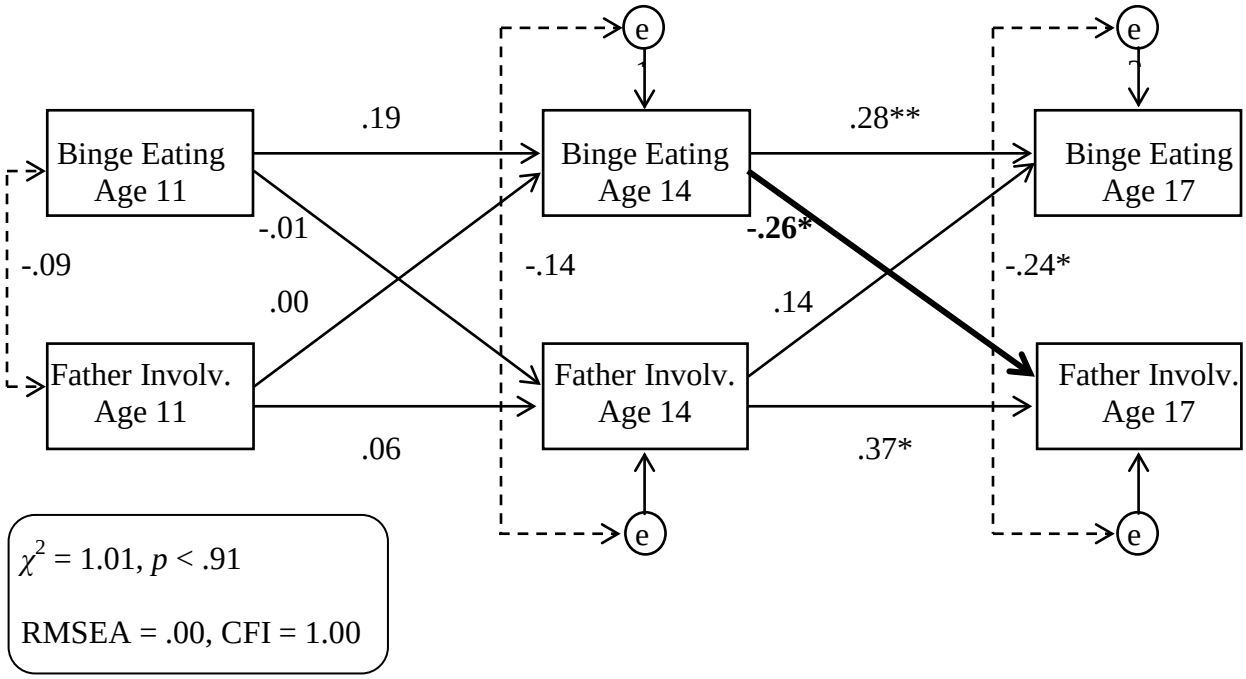
Summary. BMI did not account for a significant amount of the longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship. That is, observed associations between eating pathology and the parent-child relationship in Part 1 appeared specific to disordered eating thoughts, behaviors, and attitudes that comprise eating pathology, rather than any associated weight status.

Figure 5. Cross-lagged model of Total eating pathology and twin ratings of Father Involvement within the discordant subsample.



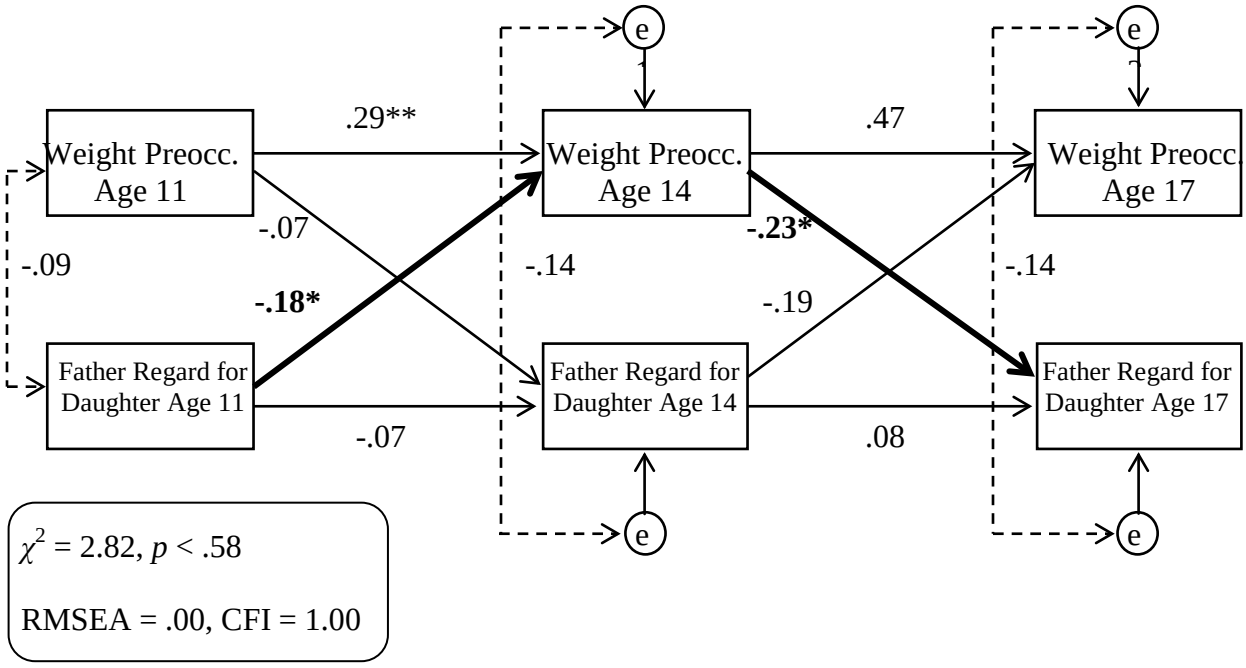
Note. The above model indicates that twin differences in MEBS Total at age 14 predicted twin differences in twin ratings of Father Involvement at age 17 within the discordant subsample. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. MEBS = Minnesota Eating Behaviors Survey, Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 6. Cross-lagged model of Binge Eating and twin ratings of Father Involvement within the discordant subsample.



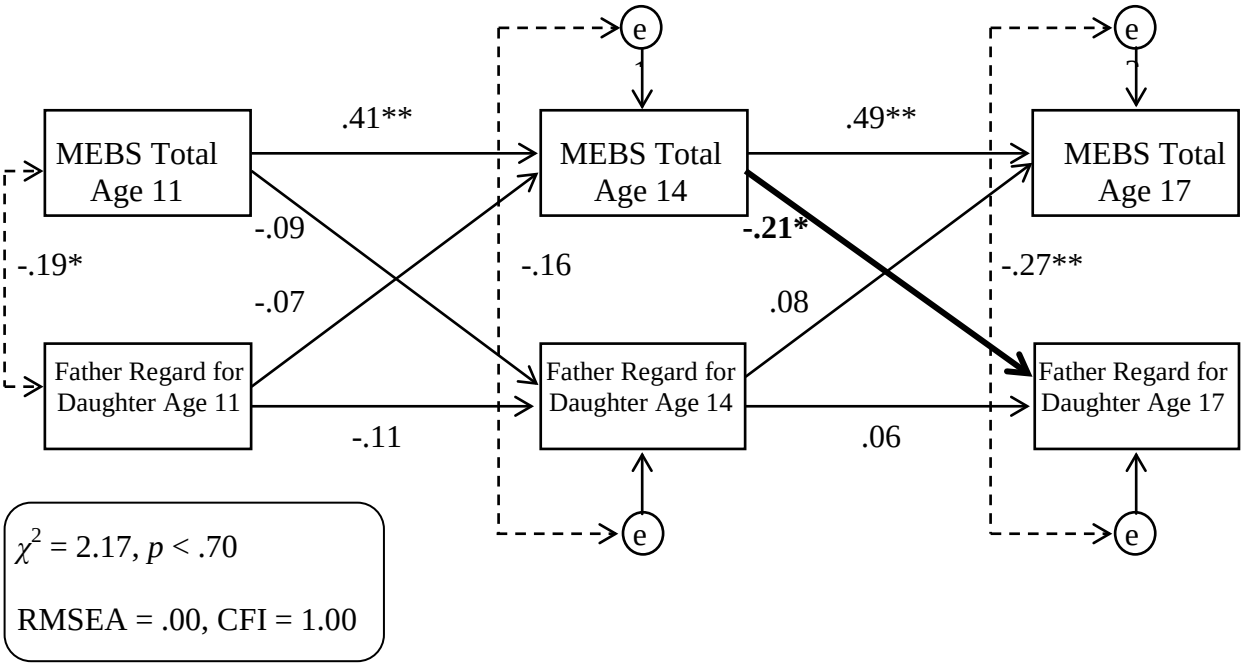
Note. The above model indicates that twin differences in Binge Eating at age 14 predicted twin differences in twin ratings of Father Involvement within the discordant subsample. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 7. Cross-lagged model of Weight Preoccupation and twin ratings of Father Regard for Daughter within the discordant subsample.



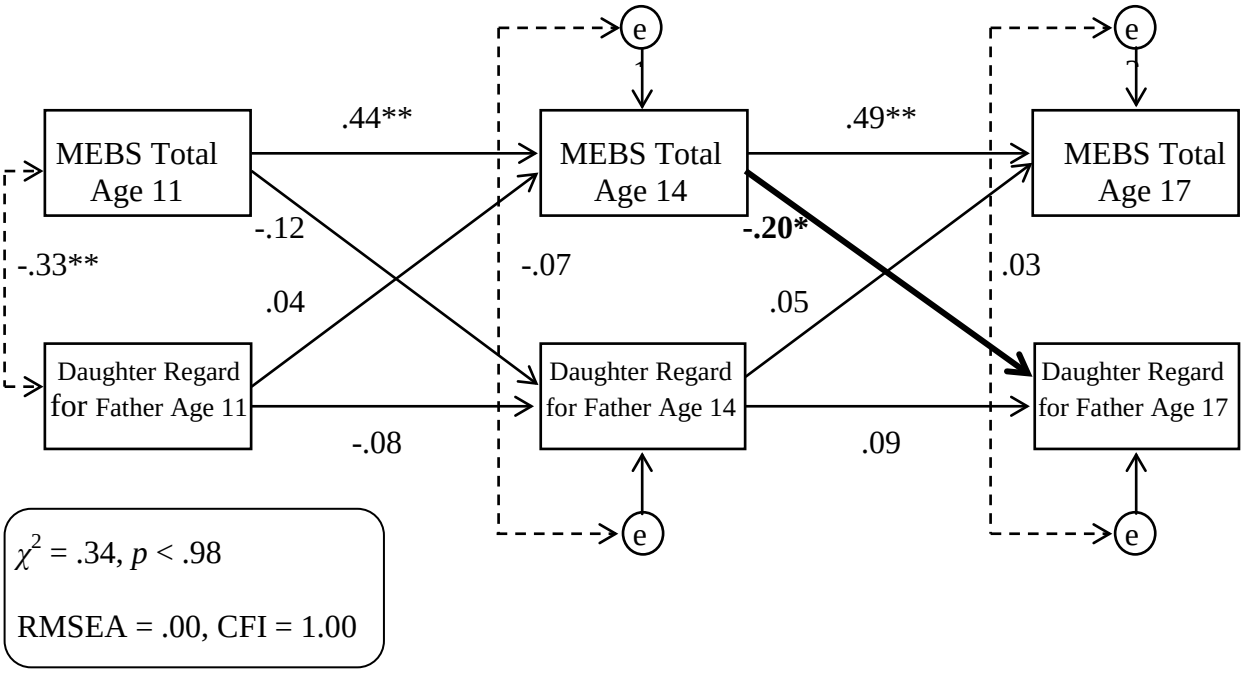
Note. The above model indicates that twin differences in twin ratings of Father Regard for Daughter at age 11 predicted twin differences in Weight Preoccupation at age 14 within the discordant subsample. In addition, twin differences in Weight Preoccupation at age 14 predicted twin differences in twin ratings of Father Regard for Daughter at age 17 within the discordant subsample. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Preocc. = Preoccupation, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 8. Cross-lagged model of Total eating pathology and twin ratings of Father Regard for Daughter within the discordant subsample.



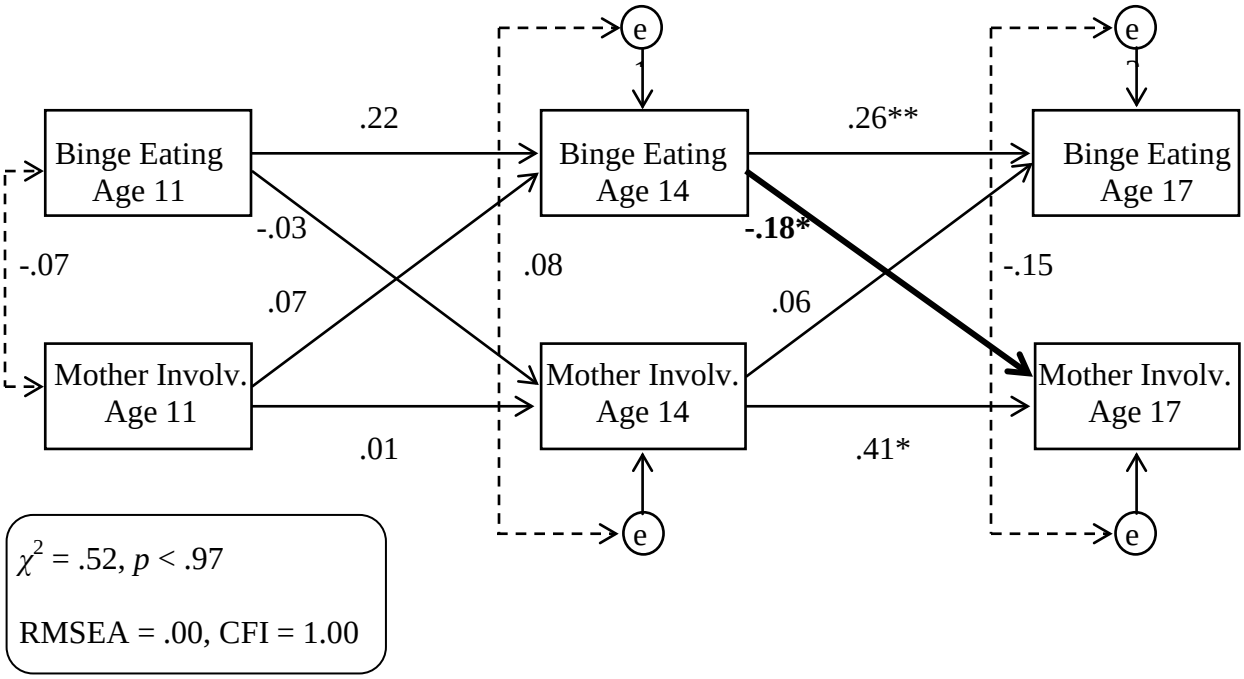
Note. The above model indicates that twin differences in MEBS Total at age 14 predicted twin differences in twin ratings of Father Regard for Daughter at age 17 within the discordant subsample. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. MEBS = Minnesota Eating Behaviors Survey, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 9. Cross-lagged model of Total eating pathology and twin ratings of Daughter Regard for Father within the discordant subsample.



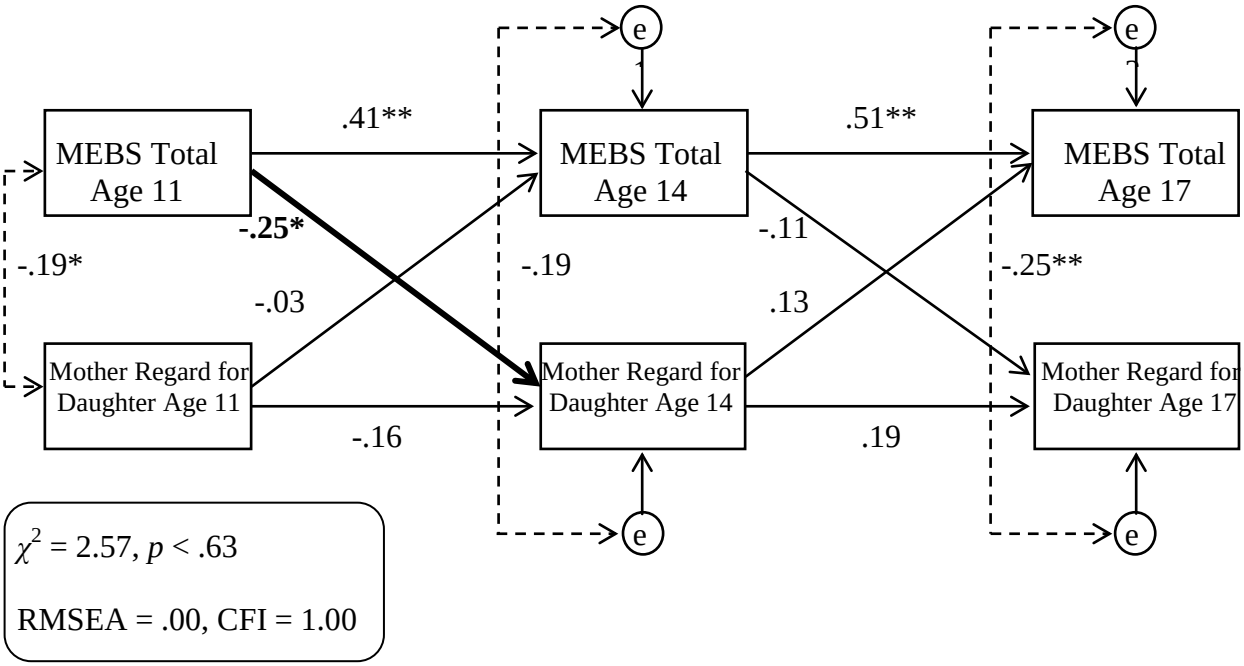
Note. The above model indicates that twin differences in MEBS Total at age 14 predicted twin differences in twin ratings of Daughter Regard for Father at age 17 within the discordant subsample. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. MEBS = Minnesota Eating Behaviors Survey, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 10. Cross-lagged model of Binge Eating and Mother Involvement within the discordant subsample.



Note. The above model indicates that twin differences in Binge Eating at age 14 predicted twin differences in twin ratings of Mother Involvement at age 17 within the discordant subsample. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 11. Cross-lagged model of Total eating pathology and twin ratings of Mother Regard for Daughter within the discordant subsample.



Note. The above model indicates that twin differences in MEBS Total at age 11 predicted twin differences in twin ratings of Mother Regard for Daughter at age 14 within the discordant subsample. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. MEBS = Minnesota Eating Behaviors Survey, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Despite determining that BMI did not account for associations between variables, the specificity of these associations to eating pathology – rather than other psychopathology – remained uncertain. Thus, Part 4 expanded on findings to consider the extent to which observed associations between twin differences in eating pathology and twin differences in the parent-child relationship were unique to eating pathology versus generalizable to other psychopathology.

PART 4:

Are longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship unique to eating pathology or generalizable to other psychopathology?

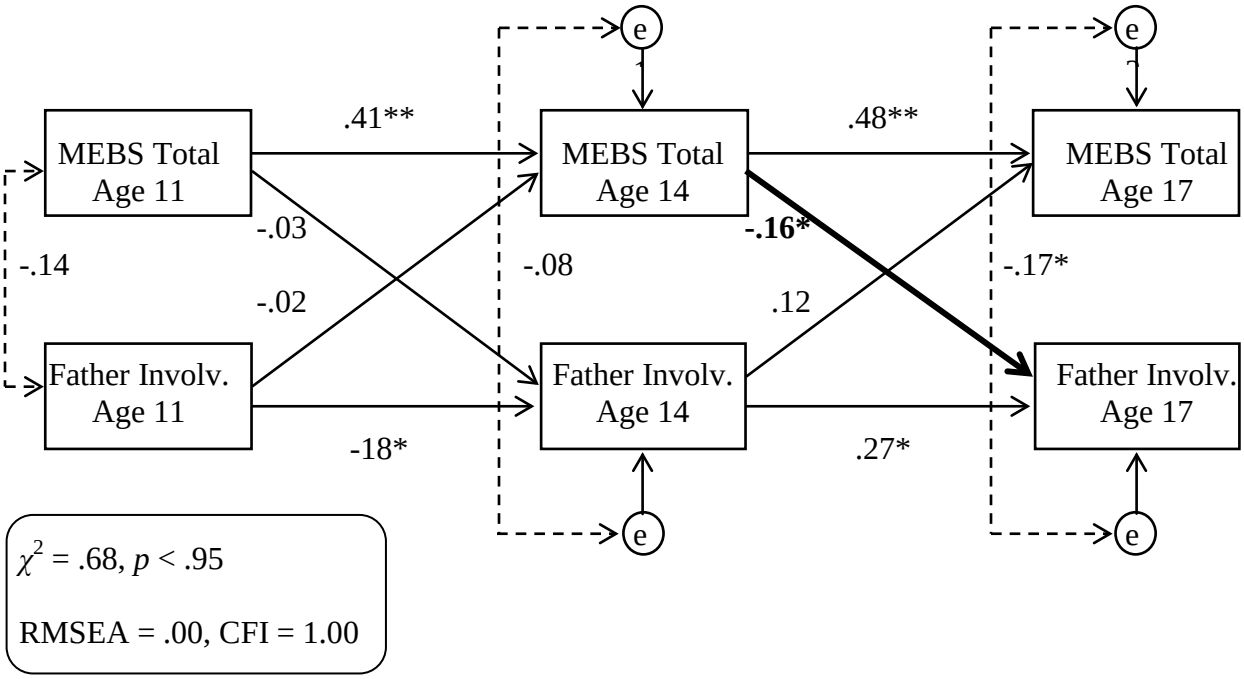
Part 4 Data Analyses

Analyses in Part 4 involved re-running analyses from Part 1 with anxiety scores replacing eating pathology scores. Analyses included (1) descriptive statistics, (2) cross-sectional and longitudinal Pearson's correlations, and (3) cross-lagged models.

Descriptive statistics. Descriptive statistics for the STAI State scale were computed. Both individual twin scores and twin difference scores were examined.

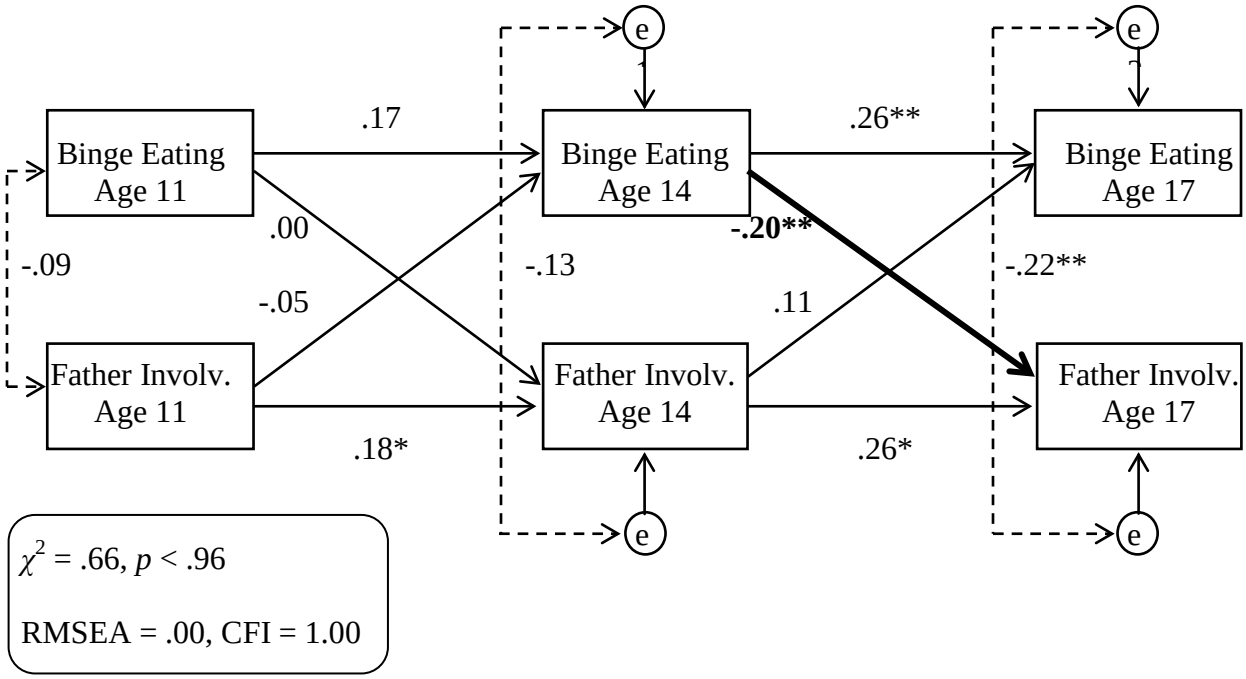
Cross-sectional and longitudinal correlations. Exploratory Pearson's correlations using individual twin scores examined within-individual cross-sectional and longitudinal associations between STAI State scores and twin ratings of PEQ scores, to examine whether a nonzero association existed between anxiety and the twins' perception of the parent-child relationship. Prior to analyses, scatterplots were examined to ensure observed associations among variables analysed in Pearson's correlations followed a roughly linear, rather than curvilinear, relationship.

Figure 12. Cross-lagged model of Total eating pathology and twin ratings of Father Involvement, wherein the effect of body mass index on Total eating pathology is controlled.



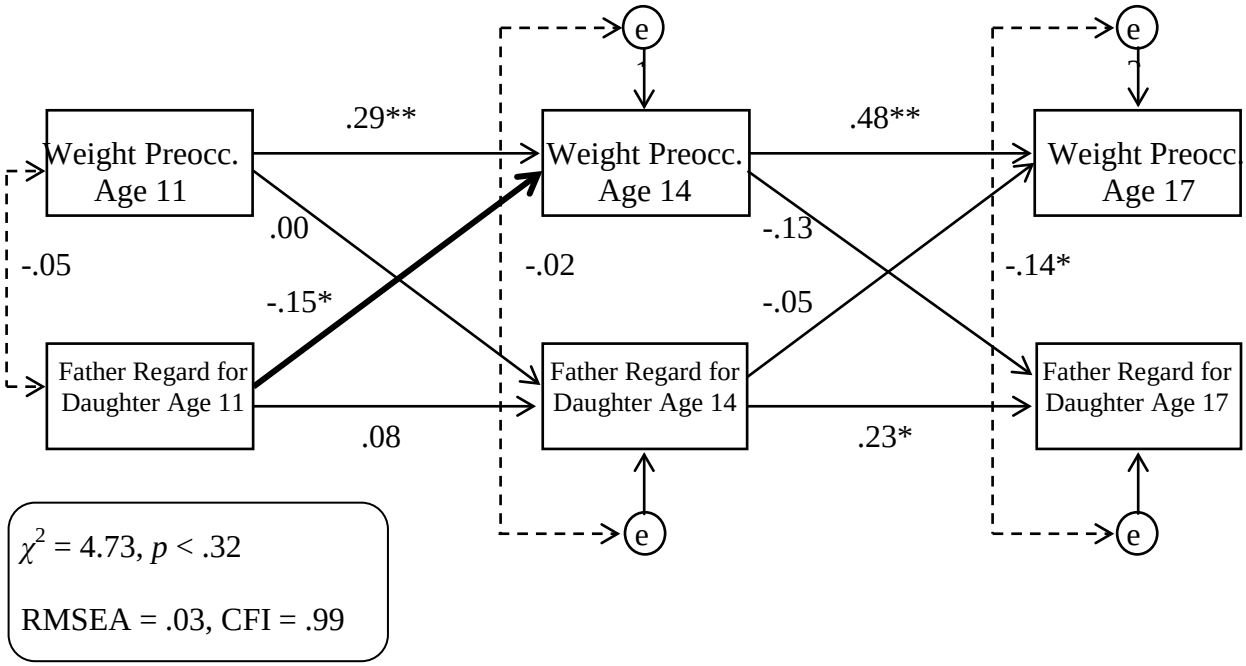
Note. The above model indicates that twin differences in MEBS Total at age 14 predicted twin differences in twin ratings of Father Involvement at age 17, when the effect of body mass index on Total eating pathology was controlled. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. MEBS = Minnesota Eating Behaviors Survey, Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 13. Cross-lagged model of Binge Eating and twin ratings of Father Involvement, wherein the effect of body mass index on Binge Eating is controlled.



Note. The above model indicates that twin differences in Binge Eating at age 14 predicted twin differences in twin ratings of Father Involvement at age 17, when the effect of body mass index on Binge Eating was controlled. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 14. Cross-lagged model of Weight Preoccupation and twin ratings of Father Regard for Daughter, wherein the effect of body mass index on Weight Preoccupation is controlled.



Note. The above model indicates that twin differences in twin ratings of Father Regard for Daughter at age 11 predicted twin differences in Weight Preoccupation at age 14, when the effect of body mass index on Weight Preoccupation was controlled. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Preocc. = Preoccupation, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Only one twin from each pair was randomly selected to be included in each analysis, so as to satisfy the assumption of independent cases (described above), and variables that violated the assumption of normality were transformed to approximate a normal distribution (using the same criteria described above).

Pearson's correlations were repeated with twin difference scores replacing individual twin scores, so as to verify whether any associations between STAI State and PEQ individual twin scores remained when the parent-child relationship was isolated as a nonshared environmental factor (i.e., with both genetics and shared environment controlled). As previously discussed (see Part 1), if associations were observed between variables only when individual twin scores were analysed, findings would suggest that associations were largely accounted for by genetic and/or shared environmental factors. In contrast, if associations were also observed between variables when twin difference scores were analysed, findings would suggest that associations were, at least in part, accounted for by nonshared environmental factors. As difference score variables included positive, negative, and zero values and the sign (i.e., positive or negative) of difference scores was essential to retain to facilitate meaningful, interpretable correlations, any difference score variables that violated the assumption of normality were not transformed to approximate a normal distribution. As a result, standard errors may be underestimated and statistical significance may be overestimated (i.e., increased likelihood of type I error).

Cross-lagged models. Cross-lagged analyses were run to examine associations between anxiety and twin ratings of the parent-child relationship. If similar associations emerged as were observed in Part 1 between eating pathology and twin ratings of the parent-child relationship, were not specific to eating pathology and may be general to other forms of psychopathology.

Separate cross-lagged models were performed to analyse associations between the STAI State scale and each PEQ scale (i.e., Mother Involvement, Father Involvement, Daughter Regard for Mother, Daughter Regard for Father, Mother Regard for Daughter, and Father Regard for Daughter). Analyses followed procedures outlined in Part 1, with one exception: STAI State scores replaced MEBS scores at each time point (see Figure 15 [p. 86] for the model's structure).

In Figure 15, associations between anxiety and parent-child relationship scores across time are indicated by the cross-lagged paths that connect STAI State at age 11 to PEQ at age 14 (b12), PEQ at age 11 to STAI State at age 14 (b21), STAI State at age 14 to PEQ at age 17 (b34), and PEQ at age 14 to STAI State at age 17 (b43). Temporal stability of anxiety and parent-child relationship scores across time is indicated by the regression paths that connect STAI State at age 11 to STAI State at age 14 (b11), PEQ at age 11 to PEQ at age 14 (b22), STAI State at age 14 to STAI State at age 17 (b33), and PEQ at age 14 to PEQ at age 17 (b44). The correlation between STAI State at age 11 and PEQ scores at age 11 was included in the model (r1) as well as the correlation between residuals associated with STAI State and PEQ scores at age 14 (r2) and 17 (r3). The MLR estimator was used to provide maximum likelihood parameter estimates with standard errors and a chi-square test statistic that are robust to non-normality; FIML was used to accommodate missing data; and adequate model fit was determined by a non-significant chi-square value, RMSEA value of .05 or less, and CFI values of .90 or greater.

Part 4 Results

Descriptive statistics. Descriptive statistics for STAI State scale individual twin scores and twin difference scores across adolescence are presented in Table 4 (p. 87).

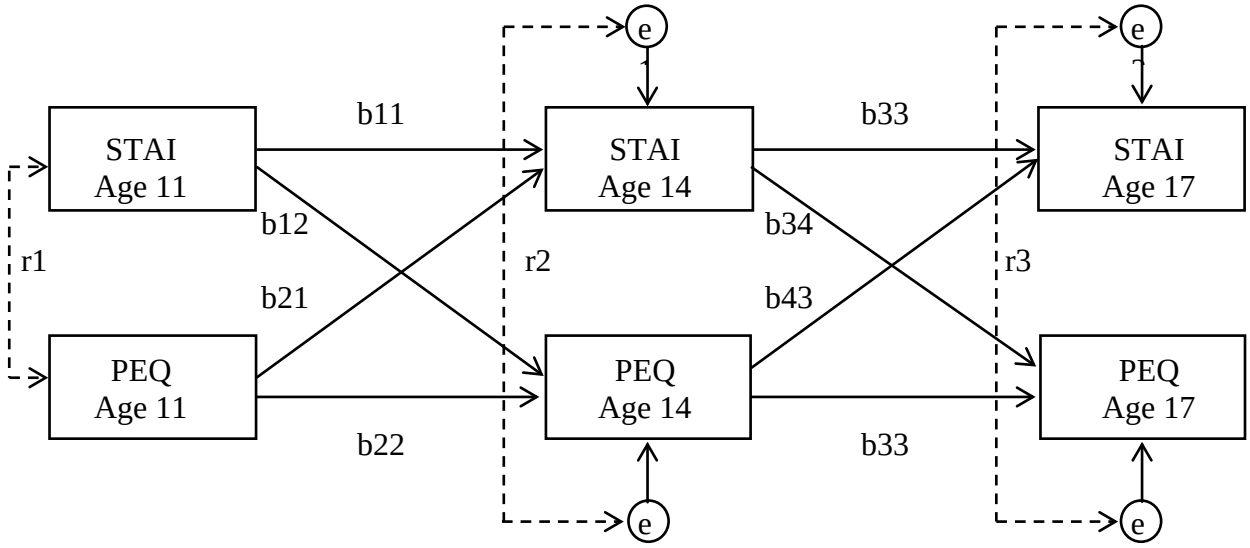
Cross-sectional and longitudinal correlations. Within- and across-age correlations between individual twin scores on STAI State and PEQ scales are presented in Table 5 (p. 89).

As the overall pattern of results largely remained consistent regardless of whether or not data that violated the assumption of normality was transformed to approximate normality, results from analyses with untransformed data are presented to assist with interpretation. However, results from analyses using transformed data are presented in Appendix J. Within- and across-age correlations between twin difference scores on STAI State and PEQ scales are presented in Table 6 (p. 91).

Significant individual twin correlations were observed both within- and across-age, with small to medium effect sizes. Where results were significant, they indicated that greater anxiety was associated with lower twin ratings of the mother-child and father-child relationship. There were fewer significant correlations observed when twin difference scores were examined, emphasizing that genetics and the shared environment contribute to associations between anxiety and the parent-child relationship. When significant associations between twin difference scores were observed, the associations tended to occur between both anxiety and the parent-child relationship measured at age 17 years and not at earlier ages. This finding suggests associations between anxiety and the parent-child relationship increase across adolescence, particularly when the parent-child relationship is measured as a nonshared environmental factor. The direction of significant associations was negative, indicating that the twin who reported lower parental regard for self, regard for parent, and involvement with parent was also more likely to report greater severity of anxiety than her co-twin.

Cross-lagged models. Overall results from analyses of cross-lagged models for STAI State and twin ratings of PEQ difference scores are described below. Detailed results for these cross-lagged models can be reviewed in Appendix K. For cross-lagged models that included one

Figure 15. Structure of cross-lagged models examining longitudinal associations between anxiety and the parent-child relationship.



Note. Partial regression coefficients are on the cross-age paths (b). Within-age correlations are illustrated by dotted lines (r). Due to the inclusion of stability and cross-lagged coefficients in the model, these within-age correlations function as residuals at ages 14 and 17 (e). Residuals reflect associations between STAI and PEQ difference scores that are specific to the age at which they were measured and independent of any preexisting associations. STAI = State-Trait Anxiety Inventory, PEQ = Parental Environment Questionnaire.

Table 4. Descriptive statistics for anxiety.

	Age 11					Age 14					Age 17				
	M	SD	Min.	Max.	*N*	M	SD	Min.	Max.	*N*	M	SD	Min.	Max.	*N*
Individual Twin Scores															
State-Trait Anxiety Inventory State scale	29.98	4.78	20	53	436	29.68	3.76	20	44	371	31.77	7.94	20	60	307
Twin Difference Scores															
State-Trait Anxiety Inventory State scale	3.59	3.56	0	23	214	2.97	2.93	0	13	185	6.38	5.65	0	27	153

or more significant cross-lagged path coefficients, the results are additionally displayed in Figure 16 (p. 94) below. All paths referenced below are labelled in Figure 15 (p. 86).

Model fit. With one exception (i.e., the cross-lagged model that examined associations between the STAI State scale and Father Regard for Daughter), all of the cross-lagged models provided adequate fit to the data, as evidenced by non-significant chi-square values, RMSEA values equal or close to zero, and CFI values of .90 or higher.

Stability. STAI State difference scores were more stable during later adolescence (path b33) than early adolescence (path b11). Specifically, STAI State difference scores at age 11 accounted for up to 3% of variance in the same construct three years later, and STAI State difference scores at age 14 accounted for up to 21% of variance in the same construct three years later. In regards to PEQ scores, stability followed the same pattern described in Part 1: Mother Regard for Daughter difference scores at age 11 and 14 accounted for up to 0% and 6%, respectively, of variance in the same construct three years later; Father Regard for Daughter difference scores at age 11 and 14 accounted for up to 1% and 6%, respectively, of variance in the same construct three years later; Daughter Regard for Mother difference scores at age 11 and 14 accounted for up to 1% and 7%, respectively, of variance in the same construct three years later; Daughter Regard for Father difference scores at age 11 and 14 accounted for up to 0% and 13%, respectively, of variance in the same construct three years later; Mother Involvement difference scores at age 11 and 14 accounted for up to 3% and 13%, respectively, of variance in the same construct three years later; and Father Involvement difference scores at age 11 and 14 accounted for up to 3% and 8%, respectively, of variance in the same construct three years later.

Concurrent associations. Overall, significant within-age correlations were strongest between STAI State and PEQ scores at age 17 years (r3) than at age 11 (r1) or 14 years (r3).

Table 5. Within-twin Pearson correlations for anxiety and twin ratings of the parent-child relationship (N = 223).

	STAI State Subscale Age 11	STAI State Subscale Age 14	STAI State Subscale Age 17
PEQ Mother Regard for Daughter			
Age 11	-.20**	-.17*	-.16
Age 14	-.14	-22**	-.12
Age 17	-.24**	-.19*	-.40**
PEQ Father Regard for Daughter			
Age 11	-.15*	-.14	-.15
Age 14	-.23**	-.25**	-.15
Age 17	-.22**	-.18*	-.38**
PEQ Daughter Regard for Mother			
Age 11	-.17*	-.26**	-.18*
Age 14	-.14	-.16	-.11
Age 17	-.18*	-.10	-.29**
PEQ Daughter Regard for Father			
Age 11	-.12	-.20**	-.17*
Age 14	-.22**	-.18*	-.13
Age 17	-.13	-.02	-.18*
PEQ Mother Involvement			
Age 11	-.12	-.16*	-.15
Age 14	-.16*	-.17*	-.13

Age 17	-.19**	-.10	-.38**
PEQ Father Involvement			
Age 11	-.08	-.10	-.11
Age 14	-.22**	-.26**	-.20*
Age 17	-.20**	-.10	-.25**

Note. STAI = State Trait Anxiety Inventory. *$p < .05$, **$p < .01$.

Table 6. Within-pair twin difference score Pearson correlations for anxiety and twin ratings of the parent-child relationship (N = 223).

	STAI State Subscale Age 11	STAI State Subscale Age 14	STAI State Subscale Age 17
PEQ Mother Regard for Daughter			
Age 11	-.13	-.05	-.01
Age 14	-.07	.02	-.02
Age 17	-.10	-.08	-.27**
PEQ Father Regard for Daughter			
Age 11	-.13	.00	-.05
Age 14	-.07	.09	.06
Age 17	-.12	-.06	-.17*
PEQ Daughter Regard for Mother			
Age 11	-.14	-.10	-.09
Age 14	-.02	.10	.01
Age 17	-.05	.01	-.32**
PEQ Daughter Regard for Father			
Age 11	-.16*	-.08	-.11
Age 14	-.16	.07	.05
Age 17	-.05	-.12	-.19*
PEQ Mother Involvement			
Age 11	-.04	-.03	-.03
Age 14	.01	.02	-.09

Age 17	-.14	.07	-.28**
PEQ Father Involvement			
Age 11	-.06	.04	.02
Age 14	-.17*	-.08	-.19*
Age 17	-.21**	.04	-.19*

Note. STAI = State Trait Anxiety Inventory. *$p < .05$, **$p < .01$.

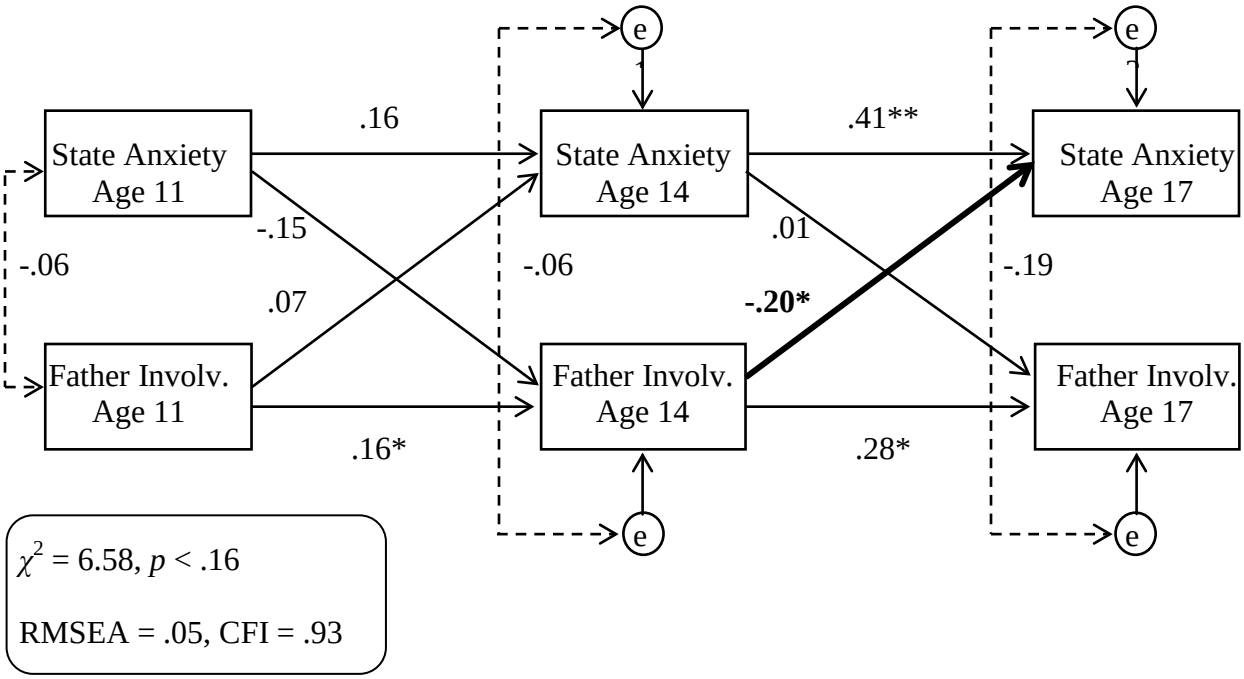
Observed associations indicated that the twin who reported greater eating pathology also reported lower concurrent parent-child relationship quality. Thus, when pre-existing associations and effects of genetics and shared environment were controlled, eating pathology and the parent-child relationship became more strongly associated as adolescents grew older.

Longitudinal associations. Only one cross-lagged model demonstrated a significant cross-lagged path: twin differences in Father Involvement at age 14 predicted twin differences in STAI State at age 17 (see Figure 16, p. 94). Specifically, the direction of association indicated that the twin who reported lower father involvement at age 14 was more likely to report more anxiety at age 17. Notably, this path was not significant when examined in cross-lagged models of eating pathology and twin ratings of father involvement (see Part 1). Thus, there was no overlap between cross-lagged associations of anxiety and the parent-child relationship and cross-lagged associations of eating pathology and the parent-child relationship.

Summary. There was a lack of overlap in findings observed in cross-lagged models of eating pathology and the parent-child relationship and cross-lagged models of anxiety and the parent-child relationship. These findings suggest that the patterns of longitudinal associations between eating pathology and the parent-child relationship identified in Part 1 are not generalizable to anxiety and the parent-child relationship. That is, associations between parent-child relationship variables and levels of eating pathology and anxiety are distinct.

As a final means of expanding on findings documented in the current study, in Part 5 we examined whether twin differences in eating pathology were differentially associated with twin differences in the parent-child relationship dependent on the informant rating the parent-child relationship. That is, as Part 1 only considered twin perceptions of the parent-child relationship, Part 5 expanded on findings to consider parent perceptions of the parent-child relationship.

Figure 16. Cross-lagged model of State anxiety and twin ratings of Father Involvement.



Note. The above model indicates that twin differences in twin ratings of Father Involvement at age 14 predicted twin differences in State anxiety at age 17. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

PART 5:

Do longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship differ based on the informant (i.e., twin versus parent) who rates the parent-child relationship?

Part 5 Data Analyses.

Analyses in Part 5 involved re- running analyses from Part 1 with parent ratings of the parent-child relationship replacing twin ratings of the parent-child relationship. Analyses included (1) descriptive statistics, (2) cross-sectional and longitudinal Pearson's correlations, and (3) cross-lagged models.

Descriptive statistics. Descriptive statistics for parent ratings of PEQ scores were computed, using both individual twin scores and twin difference scores.

Paired-samples *t*-tests compared maternal and paternal ratings of PEQ scores at age 11 and 17 years so as to indicate whether there was a significant difference in mothers' and fathers' ratings of their relationships with their children. For all paired samples *t*-tests, ratings for only one twin from each pair were randomly selected to be included in each analysis to satisfy the assumption of independent cases (described above), and variables that violated the assumption of normality were transformed (using the same criteria described above). In addition, a maximum likelihood estimator, the EM algorithm, was used to generate predicted values for missing data. The EM algorithm attempts to predict the value that is the most likely to have been reported if the participant had responded, based on other data available. Further details regarding use of the EM algorithm are included in Appendix D.

Cross-sectional and longitudinal correlations. Pearson's correlations were run using individual twin scores to examine the strength of associations between MEBS scores and

parental ratings of PEQ scores. Prior to analyses, scatterplots were examined to ensure observed associations among variables analysed in Pearson's correlations followed a roughly linear, rather than curvilinear, relationship. Parental ratings for only one twin from each pair were randomly selected to be included in each analysis, so as to satisfy the assumption of independent cases (described above), and variables that violated the assumption of normality were transformed (using the same criteria described above).

Pearson's correlations were repeated with twin difference scores replacing individual twin scores. These latter analyses were included to verify whether any associations between MEBS and PEQ individual twin scores remained when the parent-child relationship was isolated as a nonshared environmental factor (i.e., with both genetics and shared environment controlled). As previously discussed (see Part 1), if associations were observed between variables only when individual twin scores were analysed, findings would suggest that associations were largely accounted for by genetic and/or shared environmental factors. In contrast, if associations were also observed between variables when twin difference scores were analysed, findings would suggest that associations were, at least in part, accounted for by nonshared environmental factors. As previously discussed, difference score variables included positive, negative, and zero values and the sign (i.e., positive or negative) of difference scores was essential to retain to facilitate meaningful, interpretable correlations. Thus, difference score variables that violated the assumption of normality were not transformed to approximate normality, and standard errors therefore may be underestimated and statistical significance may be overestimated (i.e., increased likelihood of type I error).

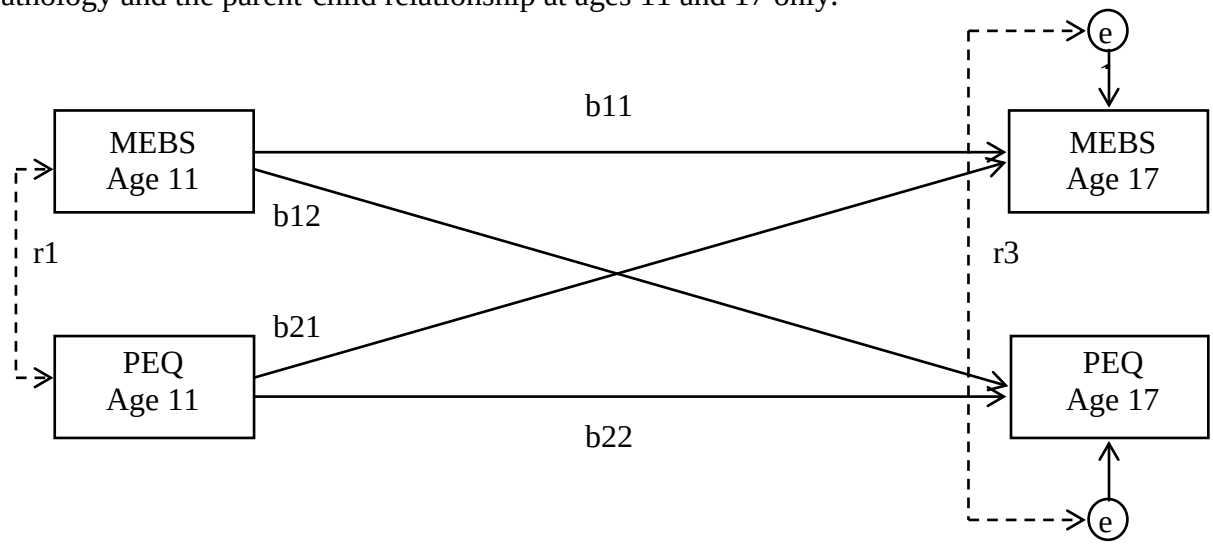
Cross-lagged models. Cross-lagged models were fitted to the twin difference scores to explore prospective, reciprocal associations between eating pathology and parental ratings of the

parent-child relationship. As in Part 1, separate cross-lagged models were performed to analyse associations between each PEQ scale (i.e., Mother Involvement, Father Involvement, Daughter Regard for Mother, Daughter Regard for Father, Mother Regard for Daughter, and Father Regard for Daughter) and each MEBS scale and subscale (i.e., Total, Body Dissatisfaction, Weight Preoccupation, and Binge Eating).

Maternal ratings. When examining maternal ratings of PEQ scores in cross-lagged models, analyses followed procedures outlined in Part 1, with one exception: maternal ratings of PEQ scores replaced twin ratings of PEQ scores at each time point (see Figure 1 [p. 44] for the model's structure). The MLR estimator was used to provide maximum likelihood parameter estimates with standard errors and a chi-square test statistic that are robust to non-normality; FIML was used to accommodate missing data; and adequate model fit was determined by a non-significant chi-square value, RMSEA value of .05 or less, and CFI values of .90 or greater.

Paternal ratings. When examining paternal ratings of PEQ scores in cross-lagged models, analyses were modified to account for the fact that the PEQ was not administered to fathers at age 14 years. Specifically, cross-lagged models included each variable (i.e., PEQ and MEBS) at age 11 and 17 years only; cross-lagged paths indicated associations between one variable at age 11 years and the other variable at age 17 years; and regression paths indicated the relative temporal stability of each construct across time from age 11 to 17 years (see Figure 17 [p. 98] for the model's structure). In Figure 17, associations between eating pathology and paternal ratings of father-child relationship scores across time are indicated by the cross-lagged paths that connect MEBS at age 11 to PEQ at age 17 (b12) and PEQ at age 11 to MEBS at age 17 (b21), and temporal stability of eating pathology and father-child relationship scores across time is indicated by the regression paths that connect MEBS at age 11 to MEBS at age 17 (b11)

Figure 17. Structure of cross-lagged models examining longitudinal associations between eating pathology and the parent-child relationship at ages 11 and 17 only.



Note. Partial regression coefficients are on the cross-age paths (b). Within-age correlations are illustrated by dotted lines (r). Due to the inclusion of stability and cross-lagged coefficients in the model, these within-age correlations function as residuals at age 17 (e). Residuals reflect associations between MEBS and PEQ difference scores that are specific to the age at which they were measured and independent of any preexisting associations. MEBS = Minnesota Eating Behaviors Survey, PEQ = Parental Environment Questionnaire.

and PEQ at age 11 to PEQ at age 17 (b22). The correlation between MEBS at age 11 and paternal ratings of PEQ scores at age 11 was included in the model (r1) as well as the correlation between residuals associated with MEBS at age 17 and paternal ratings of PEQ scores at age 17 (r2). As such, cross-lagged models using paternal ratings of PEQ scores were just-identified. In just-identified models there is a direct path from each variable to each other variable. As any just-identified model will reproduce the correlation matrix perfectly, fit indices are not relevant to cross-lagged models using paternal ratings of PEQ scores and so were not examined.

The MLR estimator was used to provide maximum likelihood parameter estimates with standard errors that are robust to non-normality. Notably, there was a large amount of missing data in paternal ratings of the father-child relationship at age 17 years (i.e., 52% missing). As such, only cases with paternal ratings of the father-child relationship available at age 17 years were included in cross-lagged models (n = 106), rather than including all cases and using FIML to account for missing data. Although restricting analyses to this subsample of data may have created a more biased sample than used in other cross-lagged models, the decision was made to proceed with analyses recognizing the importance of considering paternal perceptions, an often overlooked variable in developmental psychopathology research. However, results from these analyses should be interpreted with caution.

Part 5 Results

Descriptive statistics. Descriptive statistics for mother and father PEQ ratings are presented in Table 7 (p. 100), including individual twin scores and twin difference scores. Absolute values of twin difference scores are presented to indicate the magnitude of differences. Notably, very little variability was observed within twin pairs on both maternal ratings of Mother Regard for Daughter and paternal ratings of Father Regard for Daughter. That is, parents

Table 7. Descriptive statistics for parent ratings of the parent-child relationship.

	Age 11					Age 14					Age 17				
	M	SD	Min.	Max.	*N*	M	SD	Min.	Max.	*N*	M	SD	Min.	Max.	*N*
Individual Twin Scores															
PEQ Mother Regard for Daughter	19.77	.64	16	20	403	19.70	.79	15	20	367	19.67	.79	15	20	330
PEQ Father Regard for Daughter	19.54	1.04	14	20	325	-	-	-	-	-	19.33	1.30	13	20	214
PEQ Daughter Regard for Mother	28.91	2.5	21	32	403	27.66	2.84	18	32	362	27.59	2.80	15	32	329
PEQ Daughter Regard for Father	28.52	2.62	19	32	322	-	-	-	-	-	27.46	2.90	21	32	213
PEQ Mother Involvement	44.72	3.39	28	48	403	43.00	3.91	30	48	363	42.34	4.44	28	48	327
PEQ Father Involvement	41.12	4.71	23	48	320	-	-	-	-	-	39.11	5.01	26	48	213
Twin Difference Scores															
PEQ Mother Regard for	.17	.48	0	3	200	.16	.51	0	4	183	.21	.49	0	3	164

Daughter															
PEQ Father Regard for Daughter	.33	.84	0	5	160	-	-	-	-	-	.36	.94	0	7	107
PEQ Daughter Regard for Mother	.67	.93	0	4	200	.91	1.06	0	6	180	.97	.98	0	4	164
PEQ Daughter Regard for Father	.85	1.18	0	5	159	-	-	-	-	-	.79	1.09	0	7	106
PEQ Mother Involvement	1.03	1.28	0	6	200	1.10	1.64	0	9	181	1.64	1.71	0	8	163
PEQ Father Involvement	1.64	1.98	0	11	157	-	-	-	-	-	1.71	1.93	0	10	106

Note. The absolute values of twin difference scores are presented. PEQ = Parental Environment Questionnaire.

tended to report the same level of regard for their twin regardless of which twin he/she rated. Such lack of variability suggests parent rated Regard for Daughter may reflect the parent's personal characteristics and/or the shared environment more so than reflect a nonshared environmental factor. Lack of variability may also reflect a reporting bias – e.g., a tendency to avoid rating one's regard for each child differentially. Considering this low variability as well as the aforementioned low reliability of parent ratings of Regard for Daughter, difference scores for maternal ratings of Mother Regard for Daughter and paternal ratings of Father Regard for Daughter were not included in correlational or cross-lagged analyses. As a result of excluding these variables from further analyses, analyses cannot examine longitudinal associations between eating pathology and parental regard for the child from the perspective of multiple informants.

Paired-samples *t*-tests that compared maternal and paternal ratings of the parent-child relationship indicated that mothers tended to rate their relationship with their twin more positively than fathers rated their relationship with their twin. Specifically, maternal ratings of Mother Involvement were higher than paternal ratings of Father Involvement at age 11 ($t(222) = -11.21$, $p < .001$) and 17 ($t(222) = -7.61$, $p < .001$) years and maternal ratings of Mother Regard for Daughter were higher than paternal ratings of Father Regard for Daughter at age 11 ($t(222) = -4.40$, $p < .001$) and 17 ($t(222) = -3.34$, $p < .01$) years. Further, maternal ratings of Daughter Regard for Mother were rated higher than paternal ratings of Daughter Regard for Father at age 11 years ($t(222) = 3.00$, $p < .01$), although there was no significant difference in these ratings at age 17 years. As aforementioned, recognizing the large amounts of missing data for paternal ratings of the father-child relationship at age 17, results should be interpreted with caution. See Table 7 (p. 100) for mean scores.

Cross-sectional and longitudinal associations. Within- and across-age correlations between individual twin scores on MEBS and parent rating PEQ scales are presented in Table 8 (p. 104). As the overall pattern of results largely remained consistent regardless of whether or not data that violated the assumption of normality was transformed to approximate normality, results from analyses with untransformed data are presented to assist with interpretation. Results from analyses with transformed data are presented in Appendix L. Within- and across-age correlations between twin difference scores on MEBS and parent rating PEQ scales are presented in Table 9 (p. 106).

Fewer statistically significant correlations were observed between MEBS and parent rating PEQ scores than observed in Part 1 between MEBS and twin rating PEQ scores, emphasizing that the twin's perspective of the parent-child relationship is more strongly associated with eating pathology. Where statistically significant correlations were observed, effect sizes tended to be small to medium. Notably, a minority of correlations between MEBS scores and specific parent rating PEQ scores (e.g., Mother Involvement) were stronger when twin difference scores were examined rather than individual twin scores, suggesting associations between these variables may at times be masked by genetic and shared environmental effects.

Cross-lagged models. Overall results from analyses of cross-lagged models for MEBS and parent ratings of PEQ difference scores are described below. Detailed results for cross-lagged models that used mother PEQ ratings can be reviewed in Appendix M and details results for cross-lagged models that used father PEQ ratings can be reviewed in Appendix N. For cross-lagged models that included one or more significant cross-lagged path coefficient, the results are

Table 8. Within-twin Pearson correlations for eating pathology and parent ratings of the parent-child relationship (*N* = 223).

	Age 11				Age 14				Age 17			
	Total	BD	WP	Binge	Total	BD	WP	Binge	Total	BD	WP	Binge
PEQ Mother Regard for Daughter												
Age 11	.04	.05	.04	.03	.09	.08	.10	.09	-.05	-.05	-.06	-.02
Age 14	.01	.06	.04	-.03	.02	-.02	.06	.02	.12	.07	.12	.11
Age 17	.05	.05	.06	.02	.10	.11	.05	.11	-.03	-.09	-.03	.07
PEQ Father Regard for Daughter												
Age 11	-.02	.01	-.02	.03	-.13	-.02	-.18*	-.02	-.01	.05	-.07	.09
Age 14	-	-	-	-	-	-	-	-	-	-	-	-
Age 17	-.17	-.17	-.13	-.13	-.11	-.10	-.06	-.10	-.03	.00	-.03	-.10
PEQ Daughter Regard for Mother												
Age 11	.04	.04	.02	.04	.03	.03	.09	-.12	-.07	-.04	-.07	-.10
Age 14	.04	.07	.05	-.01	-.01	.01	.02	-.05	.00	.00	-.03	.04
Age 17	.05	.07	.06	.02	.01	.08	.00	-.02	.01	.06	-.03	.02
PEQ Daughter Regard for Father												

Age 11	.08	.05	.06	.06	.00	.02	.00	.01	-.01	.03	-.06	-.01
Age 14	-	-	-	-	-	-	-	-	-	-	-	-
Age 17	-.21*	-.21*	-.26**	.03	-.10	-.13	-.05	-.06	-.06	-.07	-.04	-.05
PEQ Mother Involvement												
Age 11	.02	.02	.01	.02	.05	.04	.08	-.02	-.12	-.19*	-.06	-.07
Age 14	-.10	-.05	-.06	-.05	-.10	-.10	-.02	-.14	-.13	-.13	-.07	-.13
Age 17	-.03	-.03	-.05	.03	.01	.00	.02	.05	-.10	-.08	-.10	-.10
PEQ Father Involvement												
Age 11	-.01	-.05	.03	-.04	-.08	-.08	-.08	.01	-.12	-.10	-.13	-.04
Age 14	-	-	-	-	-	-	-	-	-	-	-	-
Age 17	-.16	-.17	-.18	-.03	-.10	-.07	-.12	-.02	-.03	-.01	-.04	-.05

Note. Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, PEQ = Parental Environment Questionnaire. *$p < .05$, **$p < .01$.

Table 9. Within-pair twin difference score Pearson correlations for eating pathology and parent ratings of the parent-child relationship (N = 223).

	Age 11				Age 14				Age 17			
	Total	BD	WP	Binge	Total	BD	WP	Binge	Total	BD	WP	Binge
PEQ Daughter Regard for Mother												
Age 11	.02	.10	-.02	-.06	-.04	-.13	.06	-.07	.07	.03	.05	.10
Age 14	-.10	-.11	-.10	-.01	-.03	.02	-.05	.10	-.02	-.06	.01	.06
Age 17	-.03	.03	-.12	-.11	-.05	-.06	-.04	.11	.03	.09	-.01	-.05
PEQ Daughter Regard for Father												
Age 11	.01	.09	-.06	.04	-.09	-.04	-.11	-.02	-.06	-.04	-.01	-.04
Age 14	-	-	-	-	-	-	-	-	-	-	-	-
Age 17	-.18	-.26*	-.03	.00	-.09	-.04	.00	-.30**	.04	-.03	.00	.15
PEQ Mother Involvement												
Age 11	.00	.02	-.07	.06	.05	.07	.07	-.06	-.02	-.07	.02	-.05
Age 14	-.09	-.06	-.04	-.03	.07	.05	.07	.06	.17*	.14	.26**	-.07

Age 17	-.21*	-.15	-.20*	-.15	-.18*	.09	-.22**	.03	-.05	.04	-.07	-.04
PEQ Father Involvement												
Age 11	-.05	-.10	-.10	.14	-.14	-.13	-.10	-.13	-.12	-.11	-.09	-.05
Age 14	-	-	-	-	-	-	-	-	-	-	-	-
Age 17	-.18	-.21*	-.08	.01	-.16	-.18	-.09	-.13	-.12	-.11	-.13	-.01

Note. Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, PEQ = Parental Environment Questionnaire. *$p < .05$, **$p < .01$.

additionally displayed in Figures 18-21 (p. 112-115) below. Paths listed below that relate to cross-lagged models that include mother PEQ ratings are labelled in Figure 1 (p. 44); paths listed below that relate to cross-lagged models that include father PEQ ratings are labelled in Figure 17 (p. 98), unless otherwise noted.

Model fit. Model fit was examined for cross-lagged models that included mother rating PEQ scores only, as cross-lagged models that included father rating PEQ scores were just-identified models (i.e., fit statistics were not relevant). Almost all of the cross-lagged models that included mother rating PEQ scores provided adequate fit to the data, as evidenced by non-significant chi-square values, RMSEA values equal or close to zero, and CFI values of .90 or greater. However, models that included the Daughter Regard for Mother scale tended to demonstrate poor fit. As the Daughter Regard for Mother scale also demonstrated little variability within twin pairs when rated by mothers (see Table 7, p. 100), lack of model fit may have been affected by minimal variability between scores. This interpretation is supported by the fact that model fit was poorest when this scale was analysed in models that included MEBS scales with lesser variability within twin pairs.

In addition, models that included the Body Dissatisfaction subscale tended to demonstrate poor fit. Thus, as described in Part 1, analyses of cross-lagged models that included the Body Dissatisfaction subscale and mother rating PEQ scores were re-run with an additional longitudinal path included between Body Dissatisfaction at age 11 years and Body Dissatisfaction at age 17 years. The additional longitudinal path was significant in all models (*p*s < .05) and improved model fit across all models, but did not change the statistical significance of any cross-lagged path in any model.

Stability. Stability of mother rating PEQ twin difference scores was low during both early (path b22) and later (b44) adolescence. Specifically, Daughter Regard for Mother difference scores at age 11 and 14 accounted for 0% and up to 4%, respectively, of the variance in the same construct three years later, and Mother Involvement difference scores at age 11 and 14 accounted for approximately 0% of variance in the same construct three years later. Thus, mothers' perceptions of her relationship with each twin varied over time. In contrast, stability of MEBS scores was stronger, following the same pattern described in Part 1: MEBS Total difference scores at age 11 and 14 accounted for up to 18% and 21%, respectively, of the variance in the same construct three years later; Body Dissatisfaction difference scores at age 11 and 14 accounted for up to 6% and 12%, respectively, of the variance in the same construct three years later; Weight Preoccupation difference scores at age 11 and 14 accounted for up to 10% and 22%, respectively, of the variance in the same construct three years later; and Binge Eating difference scores at age 11 and 14 accounted for up to 4% and 6%, respectively, of the variance in the same construct three years later.

In regards to stability of father rating PEQ twin difference scores, Daughter Regard for Father difference scores at age 11 accounted for approximately 0% of the variance in the same construct at age 17 years, and Father Involvement difference scores at age 11 accounted for up to 3% of variance in the same construct at age 17 years. Thus, the father's perception of differences in the quality of his relationship with each twin at age 11 years generally was not predictive of his perception of differences in the quality of his relationship with each twin at age 17 years. When stability of MEBS scores from age 11 to 17 years was examined in these models, MEBS Total difference scores at age 11 accounted for up to 6% of the variance in the same construct six years later; Body Dissatisfaction difference scores at age 11 accounted for up to 7% of the

variance in the same construct six years later; Weight Preoccupation difference scores at age 11 accounted for up to 1%, respectively, of the variance in the same construct six years later; and Binge Eating difference scores at age 11 accounted for 0% of the variance in the same construct six years later.

Concurrent associations. Overall, within-age correlations between maternal or paternal PEQ ratings and MEBS scores generally did not reach statistical significance at any age (r1-r3). Where significant concurrent associations did emerge, associations were typically between paternal reports of the father-child relationship and eating pathology at age 17 years. Thus, when pre-existing associations and effects of genetics and shared environment were controlled, concurrent associations between eating pathology and the parent-child relationship were generally stronger when the parent-child relationship was rated by the twin (see Part 1), rather than by parents.

Longitudinal associations. Some significant coefficients were observed on cross-lagged paths in models that examined various MEBS scores and mother rating PEQ scores. Specifically, twin differences in maternal ratings of Daughter Regard for Mother at age 11 predicted twin differences in Body Dissatisfaction at age 14 (see Figure 18, p. 112), with the direction of association indicating that the twin who was perceived by her mother to have lower regard for her mother later reported greater body dissatisfaction. In addition, twin differences in MEBS Total (see Figure 19, p. 113) and MEBS Weight Preoccupation (see Figure 20, p. 114) at age 14 both predicted twin differences in maternal ratings of Mother Involvement at age 17. The direction of association indicated that the twin who reported greater total eating pathology or greater weight preoccupation was later described by her mother as having less involvement in the mother-child relationship. In contrast, twin differences in maternal ratings of Mother

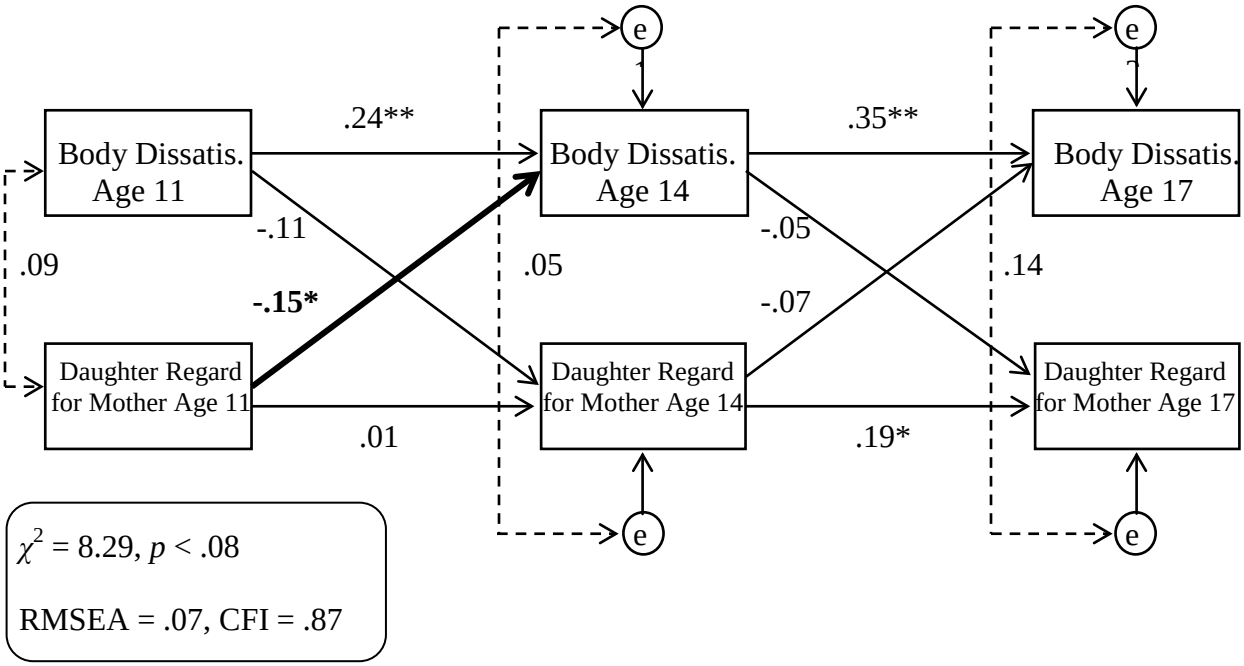
Involvement at age 14 predicted twin differences in Weight Preoccupation at age 17 (see Figure 20, p. 114). Unexpectedly, the direction of association indicated that the twin who the mother described having greater involvement with later reported greater weight preoccupation. No significant cross-lagged paths were observed in any of the models that examined associations between any mother rating PEQ scale and MEBS Binge Eating.

Only one significant coefficient was observed on cross-lagged paths in models that examined various MEBS scores and father rating PEQ scores. Specifically, twin differences in Body Dissatisfaction at age 11 predicted twin differences in paternal ratings of Daughter Regard for Father at age 17 (see Figure 21, p. 115). The direction of association indicated that the twin who reported greater body dissatisfaction later was described by her father as having lower regard for her father. However, due to large amounts of missing data for paternal PEQ ratings at age 17 and exclusion of cases with missing paternal PEQ ratings at age 17 in these analyses, results should be interpreted with caution. [12]

Recognizing within-pair twin difference score correlations for eating pathology and paternal ratings of the father-child relationship indicated a moderate negative correlation between Binge Eating at age 14 and Daughter Regard for Father at age 17 (see Table 9, p.106), an additional cross-lagged model was run to examine whether this association would be observed. See Figure 22 (p. 117) for the model's structure. Specifically, the cross-lagged model included Binge Eating twin difference scores at age 11, 14, and 17 years and paternal ratings of Daughter Regard for Father twin difference scores at age 11 and 17. Cross- lagged paths

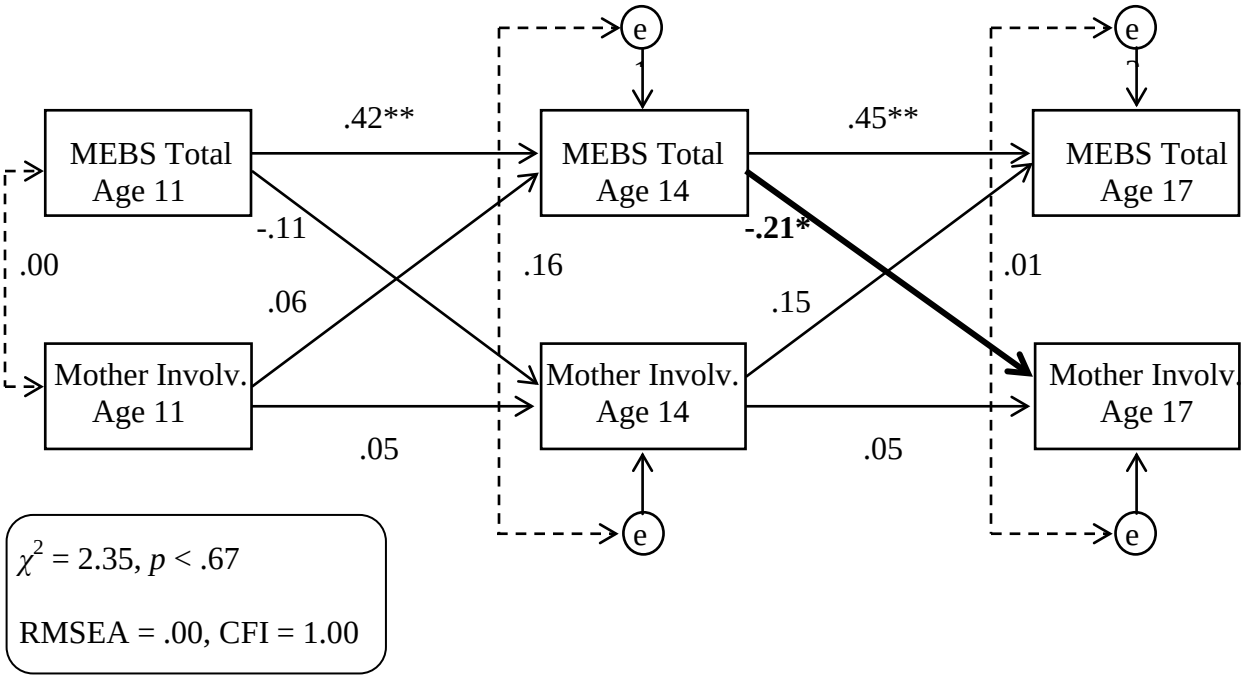
[12] To consider the extent to which exclusion of cases with missing data on paternal reports at age 17 may have generated biased results, analyses were subsequently re-run using FIML to accommodate missing data. As aforementioned, one simulation study indicated FIML produced regression coefficients that were biased by approximately 12% when 50% of data was missing (Schlomer et al., 2010).The overall pattern of findings remained the same. That is, cross-lagged path coefficients that were significant in analyses that used only cases with complete paternal data at age 17 continued to be significant in analyses that used FIML to accommodate missing data, and no additional cross-lagged path coefficients were significant. However, values of path coefficients changed. Results of analyses that used FIML to accommodate missing data are included in Appendix O.

Figure 18. Cross-lagged model of Body Dissatisfaction and maternal ratings of Daughter Regard for Mother.



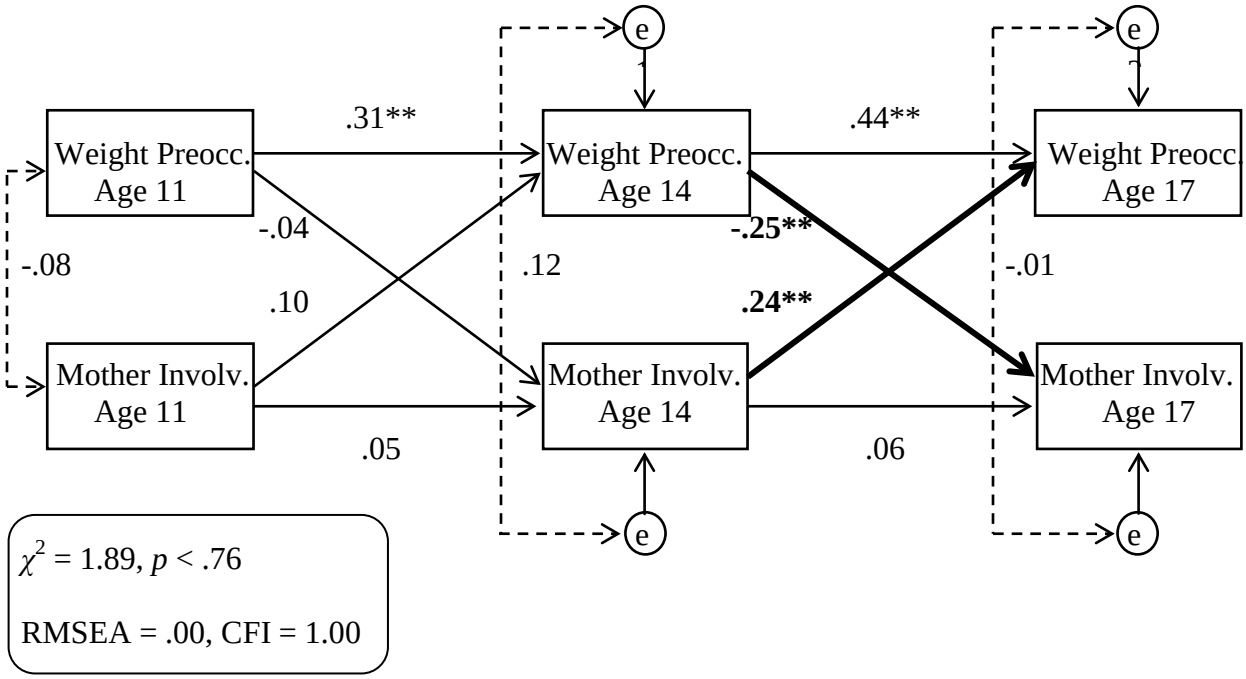
Note. The above model indicates that twin differences in maternal ratings of Daughter Regard for Mother at age 11 predicted twin differences in Body Dissatisfaction at age 14. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Dissatis. = Dissatisfaction, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 19. Cross-lagged model of Total eating pathology and maternal ratings of Mother Involvement.



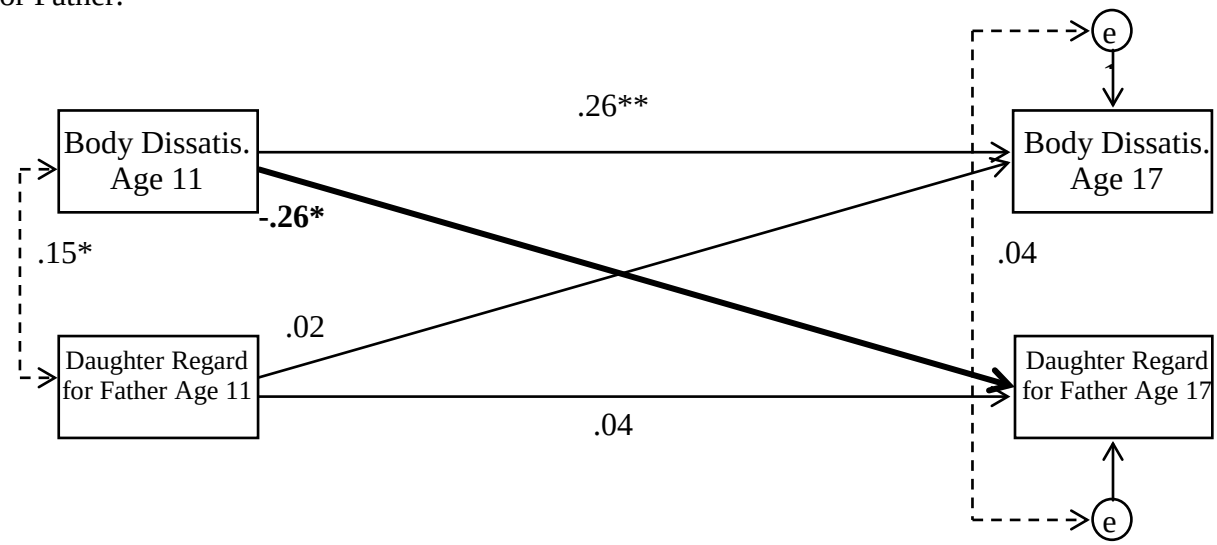
Note. The above model indicates that twin differences in Total eating pathology at age 14 predicted twin differences in maternal ratings of Mother Involvement at age 17. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. MEBS = Minnesota Eating Behaviors Survey, Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 20. Cross-lagged model of Weight Preoccupation and maternal ratings of Mother Involvement.



Note. The above model indicates that twin differences in Weight Preoccupation at age 14 predicted twin differences in maternal ratings of Mother Involvement at age 17, and that twin difference in maternal ratings of Mother Involvement at age 14 predicted twin differences in Weight Preoccupation at age 17. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Preocc. = Preoccupation, Involv. = Involvement, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Figure 21. Cross-lagged model of Body Dissatisfaction and paternal ratings of Daughter Regard for Father.



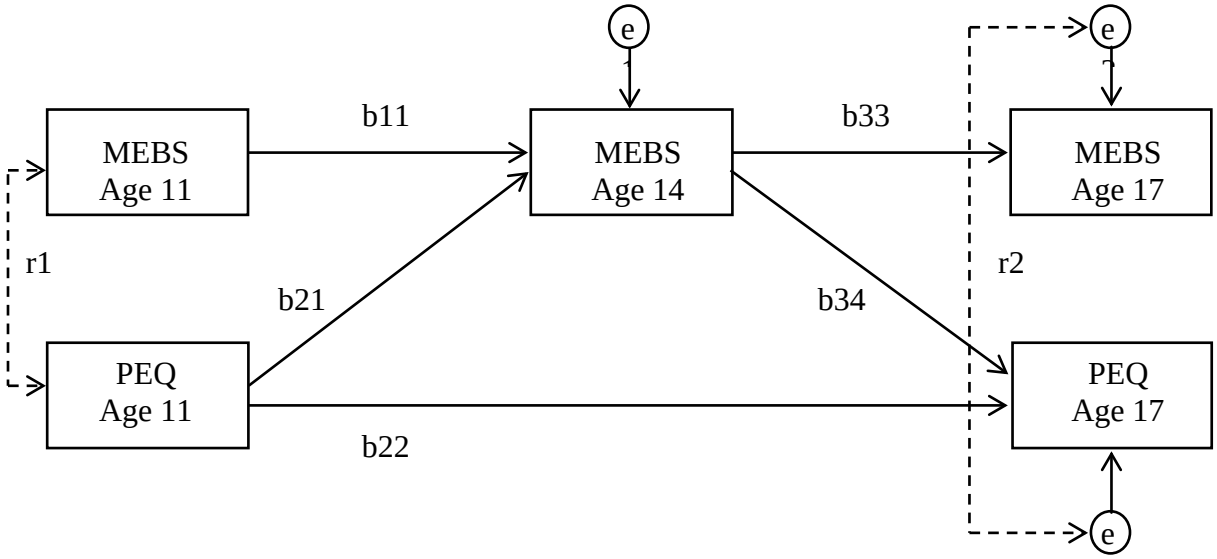
Note. The above model indicates that twin differences in Body Dissatisfaction at age 11 predicted twin differences in paternal ratings of Daughter Regard for Father at age 17. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. Dissatis. = Dissatisfaction, e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *$p < .05$, **$p < .01$.

connected Daughter Regard for Father at age 11 to Binge Eating at age 14 (b21) and Binge Eating at age 14 to Daughter Regard for Father at age 17 (b34). The model demonstrated adequate fit to the data and one significant cross-lagged path: twin differences in Binge Eating at age 14 predicted twin differences in paternal ratings of Daughter Regard for Father at age 17 (see Figure 23, p. 118). The direction of association indicated the twin who reported greater binge eating was later described by her father as having lower regard for her father.[13]

Summary. Overall, correlations between twin differences in eating pathology and twin differences in the parent-child relationship were less robust when parent ratings, as compared to twin ratings, of the parent-child relationship were examined. Among cross-lagged models of maternal ratings of the mother-child relationship, where significant cross-lagged associations emerged findings generally followed the pattern that emerged with twin ratings of the mother-child relationship: i.e., parent-child relationship variables were more likely to predict later eating pathology across early adolescence whereas eating pathology was more likely to predict later parent-child relationship variables across later adolescence. Findings related to paternal ratings of the father-child relationship were more difficult to compare to the pattern observed with twin ratings of the father-child relationship due to missing paternal ratings at age 14 and the need to change the structure of cross-lagged models. Nonetheless, where significant cross-lagged associations emerged, some similarities were observed to associations that emerged with twin ratings of the father-child relationship: i.e., development of eating pathology predicted that specific aspects of the father-child relationship would be poorer by late adolescence.

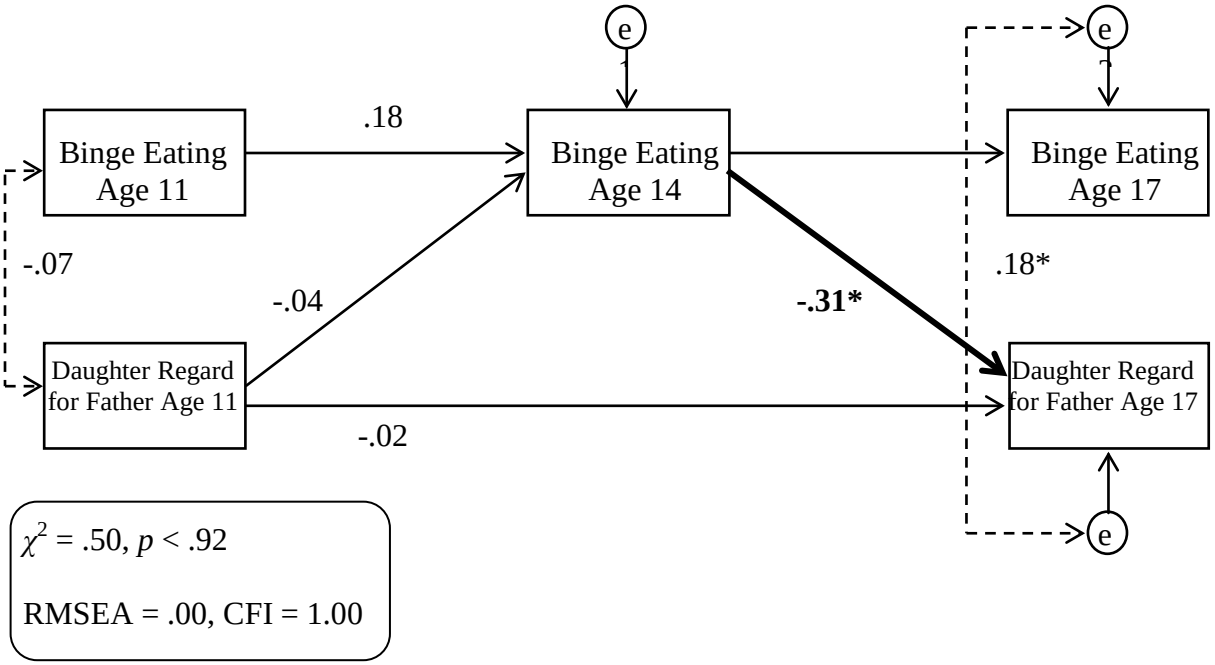
[13] Both cross-lagged models that included paternal PEQ ratings and indicated a significant cross-lagged path (see Figures 21 and 23) were rerun including only cases wherein the biological father provided PEQ ratings at both age 11 and 17 years (i.e., 12 cases that involved non-biological father PEQ ratings were removed). The pattern of significant findings on cross-lagged paths remained the same, suggesting that results were not affected by inclusion of ratings from both biological fathers and non-biological fathers.

Figure 22. Structure of follow-up cross-lagged model examining longitudinal associations between eating pathology at age 11, 14, and 17 and the parent-child relationship at age 11 and 17.



Note. Partial regression coefficients are on the cross-age paths (b). Within-age correlations are illustrated by dotted lines (r). Due to the inclusion of stability and cross-lagged coefficients in the model, these within-age correlations function as residuals at age 17 (e). Residuals reflect associations between MEBS and PEQ difference scores that are specific to the age at which they were measured and independent of any preexisting associations. MEBS = Minnesota Eating Behaviors Survey, PEQ = Parental Environment Questionnaire.

Figure 23. Cross-lagged model of Binge Eating and paternal ratings of Daughter Regard for Father.



Note. The above model indicates that twin differences in Binge Eating at age 14 predicted twin differences in paternal ratings of Daughter Regard for Father at age 17. Standardized across-age path coefficients are illustrated by single-headed arrows; within-age correlations are illustrated by dotted lines. Significant cross-lagged associations are denoted in bold. e = residual variance at ages 14 and 17 years; RMSEA = root mean square error of approximation, CFI = comparative fit index. *p < .05, **p < .01.

Discussion

The current study explored associations between girls' eating pathology and both the mother-child and father-child relationship across adolescence. A large, population-based sample of MZ twins was assessed at three time points: age 11, 14, and 17 years. Mothers and fathers were also included in assessment, facilitating ratings of the parent-child relationship from the unique perspectives of the daughter, mother, and father. Importantly, this study sought to better understand associations between eating pathology and the parent-child relationship via cross-lagged analyses that examined longitudinal, bidirectional associations between these variables and differentiated whether problems in the parent-child relationship emerged as a risk factor for, or sequelae of, eating pathology. In addition, this study used an MZ twin differences design that enabled examination of the parent-child relationship as a nonshared environmental factor by controlling for influences of genetic and shared environmental factors. Studying nonshared environmental factors can identify why siblings living within the same household differ from each other. By examining the parent-child relationship as a nonshared environmental factor, this design enables identification of specific factors (e.g., increasing communication between parent and child) that are often more amenable to change than more contextual, shared environmental factors (e.g., socioeconomic status, family structure). These nonshared environmental factors can then be targeted for change by prevention and/or treatment research.

Notably, study methodology was modelled after methodology used by Spanos et al. (2010) in a previous study of associations across adolescence between eating pathology and parent-child conflict – another nonshared environmental factor. The current study extended analyses beyond those used by Spanos et al. (2010) by examining different parent-child relationship variables (i.e., parent-child regard and involvement) and addressing additional

limitations of past research. For example, the current study considered differential associations between eating pathology and the mother-child versus father-child relationship; examined if findings generalized to other psychopathology; and considered whether findings differed based on the informant (i.e., twin versus parent) who rated the parent-child relationship.

Overall, longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship were generally neither strong nor robust, highlighting the fallacy of the notion, historically promulgated, that poor parenting is a primary mechanism underlying risk for eating pathology. However, some longitudinal associations between twin differences in eating pathology and twin differences in the parent-child relationship did emerge, with overall findings indicating reciprocal associations between certain symptoms of eating pathology (e.g., binge eating, weight preoccupation) and specific facets of the parent-child relationship (e.g., parent-child involvement). Notably, these associations shifted across developmental periods. Specifically, twin differences in the parent-child relationship tended to predict subsequent twin differences in eating pathology across early adolescence, whereas twin differences in eating pathology tended to predict subsequent twin differences in the parent-child relationship across later adolescence. These findings suggest that the process of separation and individuation from parents during later adolescence may be “accelerated” among adolescents with eating pathology, presenting further risk for negative outcomes. In particular, some of the strongest and most consistent associations indicated that twins who reported greater eating pathology relative to their co-twin were more likely to subsequently endorse more negative perceptions of their relationship with their father. These findings raise questions regarding paternal responses to adolescent eating pathology and suggest the potential importance of efforts to support the father-child relationship within the context of adolescent eating pathology. In

addition, these findings expand upon existing literature that has focused on associations between eating pathology and the mother-child relationship, and often overlooked associations with the father-child relationship.

Eating Pathology

Review of descriptive statistics suggested that total levels of eating pathology, the portion of the sample that met clinical cutoffs for eating pathology, and variability in levels of eating pathology tended to increase across adolescence. Notably, comparison of the current sample to a sample of adopted and biological sibling pairs recruited from the same region as the current study (i.e., Minnesota) indicated similar eating pathology levels and variability across studies (see Klump, Suisman, et al., 2009). Thus, the range of and changes in eating pathology across adolescence that were observed in the current sample reflected relatively normative population trends.

Parent-Child Relationship

Importantly, the current study assessed the parent-child relationship from the perspective of multiple informants – i.e., the daughter, mother, and father – and differentiated between perceptions of the mother-child relationship and perceptions of the father-child relationship. In doing so, results highlighted differences in perceived quality of the mother-child and father-child relationship that varied depending on whether the parent or daughter rated the relationship. Such differences were expected and aligned with the notion of informant variability that is commonly observed across psychological research.

Specifically, both parents and daughters tended to underestimate the level of regard the other held for them. In addition, parents – including both mothers and fathers – tended to perceive themselves as more involved in the parent-child relationship than reported by daughters.

The nature of informant variability observed in the current study generally aligned with previous research that suggests adolescents perceive family relationships to involve less cohesion and communication than perceived by parents (Ohannessian et al., 2000). Such differences in ratings of the parent-child relationship between informants highlight that relationship quality is often best considered as a subjective perception, rather than an objective state. In addition, such differences in ratings of the parent-child relationship support the importance of obtaining multiple informants' perspectives of family relationships in order to best understand relationship functioning and dynamics (Carlson, Cooper, & Spradling, 1991; Dekovic & Buist, 2005).

Regardless of these differences in ratings, both daughters and mothers rated the mother-child relationship as stronger than daughters and fathers rated the father-child relationship. Stronger relationships with mothers than fathers have oft been reported throughout the literature, including during adolescence (e.g., Ma & Huebner, 2008; Markiewicz et al., 2006; Williams & Kelly, 2005). However, both parent and adolescent reports have infrequently been included in such research to corroborate this perception across raters. In addition, both parents and twins perceived mothers' and fathers' involvement in the parent-child relationship and daughters' regard for their parents to somewhat decline across adolescence, particularly during early adolescence. These findings suggest mutual recognition of the normative developmental process of adolescent separation and individuation from parents (De Goede et al., 2008; Markiewicz et al., 2006; Smetana, et al., 2006).

Associations between Eating Pathology and the Parent-Child Relationship

When individual twin scores were examined, small to medium correlations were observed at specific time points across adolescence between certain eating pathology symptoms and twin ratings of regard for parent, perceived parent regard for daughter, and parental

involvement. In contrast, correlations between parental ratings of the parent-child relationship and eating pathology were generally weaker and often nonsignificant, emphasizing that different informants' perceptions of the parent-child relationship were differentially associated with eating pathology. These findings emphasize that perceptions of the parent-child relationship are unique to each individual within the relationship. Stronger correlations between eating pathology and daughter ratings of the parent-child relationship compared to parental ratings of the parent-child relationship also likely reflect that reports within-informants tend to correlate more strongly than reports between informants. Possibly, common method variance and/or reporting biases may have inflated associations between variables rated by the same rater (see Strengths and Limitations subsection for discussion of this potential limitation).

In comparison, when twin difference scores were examined, isolating parent-child relationship variables as nonshared environmental factors, some correlations between eating pathology and daughter ratings of the parent-child relationship were no longer significant. Thus, associations between eating pathology and the parent-child relationship appear to be, in part, accounted for by genetics and shared environmental factors. However, when parental ratings of the parent-child relationship were examined with twin difference scores, the strength of some correlations with eating pathology increased. The latter findings suggest that associations between eating pathology and parent ratings of the parent-child relationship may at times be masked by genetic and shared environmental effects,[14] thus most evident when the parent-child relationship is isolated as a nonshared environmental factor.

[14] For example, greater parent-child involvement may be associated with lower eating pathology. However, greater parent-child involvement may also be associated with a genetic predisposition for obsessionality and with living in a shared family environment of demanding expectations for achievement. Such genetic and shared environmental factors may be associated with increased eating pathology, thereby confounding the association between greater parent-child involvement and lower eating pathology and masking the specific association. However, once these genetic and environmental factors are controlled via the difference scores used in the MZ differences design, the specific association between parent-child involvement and eating pathology may be examined and revealed.

Notably, correlations between eating pathology and daughter ratings of the parent-child relationship were less robust than correlations reported in similar research by Spanos et al. (2010) that examined associations between eating pathology and parent-child conflict. The present findings suggest that parent-child regard and involvement are less strongly correlated with eating pathology than is parent-child conflict.

Cross-lagged models. Overall, cross-lagged models highlighted differential associations between eating pathology and the parent-child relationship that shifted across developmental periods and between the mother-child and father-child relationship.

Developmental changes. Differences in the direction of longitudinal associations between eating pathology and the parent-child relationship were observed across early and later adolescence. Specifically, although there were few associations observed across early adolescence, almost all observed associations during this developmental period indicated that parent-child relationship variables predicted later eating pathology. In contrast, almost all observed associations during later adolescence indicated that eating pathology predicted later parent-child relationship variables. Overall, these findings align with accumulating findings across other studies (e.g., Archibald et al., 1999; Archibald et al., 2002) that together emphasize that parent-child relationships are most predictive of the development of eating pathology during early adolescence. Notably, early adolescence is a developmental period wherein adolescents continue to spend a large portion of time with their parents and rely on them for attachment, security, and emotional needs (De Goede et al., 2008; Markiewicz et al., 2006; Smetana, et al., 2006).

In comparison, parent-child relationships generally are not predictive of the development of eating pathology during later adolescence, a developmental period wherein adolescents begin

to spend increasing time with peers and increasingly seek out friends instead of parents for support. Rather, findings suggest eating pathology that develops in middle adolescence (i.e., controlling for pre-existing eating pathology) may negatively affect the parent-child relationship across later adolescence, leading to a decline in the parent-child relationship as a possible outcome of eating pathology.

Notably, these findings generally aligned with those identified by Spanos et al. (2010) using similar methodology (i.e., cross-lagged models of MZ twin difference scores) but different parent-child relationship variables. In Spanos et al.'s study, the only observed longitudinal association between eating pathology and parent-child conflict across adolescence indicated that greater eating pathology – in particular, weight preoccupation – at middle adolescence predicted greater parent-child conflict at late adolescence.

In interpreting these results, it is important to recognize that a general decline in the parent-child relationship over the course of adolescence was observed in descriptive statistics for the entire sample, regardless of presence or absence of eating pathology. This finding emphasizes that perceptions of a more negative parent-child relationship is a normative developmental process related to separation and individuation from parents. However, findings from cross-lagged models suggest that this process of separation and individuation may be "accelerated" among adolescents with eating pathology, which may impede their ability to access parental coping supports and resources that are likely beneficial for managing symptoms and distress associated with eating pathology. Notably, research indicates that adolescents who perceive their relationships with their parents as less secure and supportive will turn to peers to seek the support they need (Nickerson & Nagle, 2005), yet continued positive parent-child relationships throughout later adolescence (e.g., time spent together, supportiveness, and

monitoring and awareness) have been demonstrated to be critical for positive mental well-being (Hair et al., 2008). Thus, weakened parent-child relationships subsequent to development of eating pathology may place the adolescent at risk for developing additional mental health issues. Possibly, disrupted parent-child relationships following the development of eating pathology may serve as a maintenance factor that could, in part, account for the persistence of eating pathology and development of comorbid mental health problems across time. Further research is needed to investigate these interpretations of findings.

Eating pathology and the father-child relationship. Despite the majority of past research related to eating pathology and the parent-child relationship having focused on examining the mother-child, rather than father-child, relationship, the current study highlights that longitudinal associations between eating pathology and the parent-child relationship were more consistently observed with the father-child, rather than mother-child, relationship. In particular, the majority of significant cross-lagged associations indicated that greater eating pathology predicted development of a more negative father-child relationship, particularly during later adolescence. For example, twin differences in both greater total levels of eating pathology and specific symptoms of binge eating predicted later twin differences in their perceptions of involvement in the father-child relationship. Further, analyses of the discordant subsamples highlighted that twin differences in total levels of eating pathology and specific symptoms of weight preoccupation additionally predicted twin differences in their perceptions of regard within the father-child relationship (i.e., daughter's regard for her father and her perceptions of her father's regard for herself). In all of these associations, the twin who reported greater eating pathology subsequently rated the father-child relationship more poorly, as compared to her co-twin.

Similarly, twin differences in body dissatisfaction at age 11 and binge eating at age 14 predicted later twin differences in the father's perceptions of each twin's regard for him, with fathers perceiving that the twin who reported greater body dissatisfaction or binge eating subsequently regarded him less highly. However, twin differences in eating pathology were not significantly associated with twin differences in the father's ratings of his involvement with each twin, suggesting associations between eating pathology and some father-child relationship variables were specific to the daughter's, not the father's, perceptions of the father-child relationship. Findings related to paternal ratings of the father-child relationship should be interpreted with caution due to the large amount of missing data and exclusion of cases without paternal ratings at age 17 years.

It is important to remember that these findings do not necessarily suggest a causal relationship. As detailed by Kraemer and colleagues (1997), to identify a causal risk factor the variable must be shown to be manipulable and, when manipulated, must be shown to change the risk of the outcome. Thus, an experimental design is required, which was not included in the current study. However, establishing the temporal precedence of a proposed risk factor to a proposed outcome is a crucial criterion for distinguishing risk factors from correlates (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001), which is achieved via the current study. Specifically, these findings suggest that development of eating pathology by middle adolescence is a risk factor for father-child relationship problems across later adolescence. While findings suggest eating pathology may present greatest risk for the adolescent's perceptions of the father-child relationship, specific aspects of the father's perception of the relationship (e.g., perceptions of his daughter's regard for himself) may also be at risk.

These findings raise questions regarding the way in which fathers respond to their daughters' eating pathology. A better understanding of how fathers recognize, understand, and respond to their daughter's symptoms of eating pathology may help better understand associations observed in this study. With such understanding, intervention strategies could be developed to support the father-child relationship in the context of eating pathology. Unfortunately, minimal research has explored paternal perceptions of and responses to daughters' eating pathology. Of available research, findings suggest that fathers may be more likely to use avoidant coping strategies in managing their daughters' eating pathology (Whitney et al., 2005). Research also suggests that effective treatment for eating pathology is associated with a reduction in critical comments between fathers and daughters (Eisler et al., 2000) and paternal perceptions of improved parent-child problem-solving (Ciao et al., 2015). Thus, although more research is needed in this area, findings suggest that fathers may have difficulties coping with and managing problems that develop in the context of eating pathology. Prevention and/or intervention strategies may be helpful for preventing and/or responding to these difficulties.

On the other hand, the fact that the majority of associations with eating pathology involved the daughter's perception of the father-child relationship, rather than the father's perception, emphasizes the importance of considering other factors that may affect daughters to perceive their relationship with their father in this way. For example, future research could consider the role of stigma and shame related to eating pathology that may cause daughters to perceive that their fathers regard them less highly.

Notably, only one significant association was observed between eating pathology and twin perceptions of the father-child relationship during early adolescence. Specifically, twin

differences in ratings of perceived father's regard for her at pre-adolescence predicted twin differences in weight preoccupation at middle adolescence. That is, the twin who perceived her father regarded her more poorly later reported greater preoccupation with her weight, as compared to her co-twin. This finding highlights an association between variables that emerged with opposite temporal precedence to associations observed during later adolescence. This association may reflect a general negative self-perception wherein young girls who perceive they are not viewed favourably by their fathers may become self-conscious of and concerned about their weight after transitioning into puberty. Alternatively, it is possible that perceptions of low regard are a result of weight-related comments and criticism from fathers, which has been demonstrated to predict concerns about weight and weight-control behaviours among adolescents (Annus et al., 2007; Neumark-Sztainer et al., 2010). Unfortunately, such variables were not measured in this study. This association is particularly interesting as a parallel association was not observed when twin ratings of the mother-child relationship were examined. Thus, this finding adds to the emerging literature that suggests there may be a unique association between the opposite-sex parent's parenting and the child/adolescent's weight and weight-related behaviors (e.g., Berge, Wall, Bauer, et al., 2010; Berge, Wall, Loth, et al., 2010; Temple, Wrotniak, Paluch, Roemmich, & Epstein, 2006). Finally, it is also important to note that a parallel association was not observed when the father rated his regard for his daughters, suggesting that this association was unique to the daughter's perception of her father's regard. Thus, it may be important to encourage fathers to more openly express their positive regard for their daughters.

Finally, in understanding associations between eating pathology and the father-child relationship it may be important to consider differences between associations with biological

fathers versus non-biological fathers. Analyses of twin ratings of the father-child relationship that excluded ratings of non-biological fathers indicated some significant associations between variables no longer reached statistical significance. Results trended in the same direction and were likely affected by reduced sample size and resultant reduced power. This interpretation is supported by the finding that statistical significance of results did not change when non-biological fathers' ratings were excluded from analyses of father ratings of the father-child relationship, wherein models were smaller and included fewer parameters. Regardless, further research with larger samples should consider the extent to which daughter perceptions of her relationship with a non-biological father be differentially associated with eating pathology as compared to daughter's perceptions of her relationship with a biological father. Factors that account for any differential associations should also be explored.

Eating pathology and the mother-child relationship. No associations were observed between twin differences in eating pathology and twin differences in their perception of the mother-child relationship when examined within the full sample, although a few associations emerged within the discordant sample. Specifically, across early adolescence, twin differences in total levels of eating pathology later predicted twin differences in their perceptions of their mother's regard for her; across later adolescence, twin differences in binge eating later predicted twin differences in their perceptions of involvement in the mother-child relationship. The direction of both associations indicated the twin who reported greater eating pathology subsequently rated the mother-child relationship more poorly (i.e., less perceived regard, less involvement), as compared to her co-twin.

When maternal ratings of the parent-child relationship were examined, across later adolescence twin differences in total levels of eating pathology and twin differences in weight

preoccupation both later predicted twin differences in maternal perceptions of their involvement in the mother-child relationship. The direction of associations indicated that mothers perceived themselves as less involved with the twin who previously reported greater symptoms of eating pathology, as compared to her co-twin. Thus, while associations between eating pathology and ratings of the mother-child relationship were less consistently observed compared to when associations between eating pathology and ratings of the father-child relationship were examined, the overall trend paralleled that observed among ratings of the father-child relationship. That is, development of eating pathology by middle adolescence may present risk to the mother-child relationship across later adolescence. In particular, findings suggest eating pathology may be followed by reduced involvement in the mother-child relationship. Further research is needed to corroborate these findings and determine whether reduced parent-child involvement following the development of eating pathology is more attributable to parents versus adolescents withdrawing from the relationship, or whether it reflects mutual disengagement from the relationship.

In addition, one association emerged that suggested aspects of the mother-child relationship predicted eating pathology, rather than the reverse. Specifically, twin differences in maternal perceptions of daughter regard for her mother at pre-adolescence later predicted twin differences in body dissatisfaction. The twin who was rated by her mother as having lower regard for her mother at pre-adolescence later reported greater body dissatisfaction, as compared to her co-twin. Notably, this association was not observed when daughters directly rated their regard for their mother, indicating the association was specific to maternal perceptions of the twin's regard. Possibly, a mother who believes one of her daughters does not respect her as highly may respond less warmly and supportively toward that daughter. The daughter may then

internalize this relative disapproval by becoming more critical of her own body, leading to greater body dissatisfaction. Relatedly, mothers who use more authoritarian parenting strategies – which have been linked to greater body dissatisfaction and other eating pathology (Enten & Golan, 2009; Zubatsky, Berge, & Neumark-Sztainer, 2014) – may be more likely to perceive the adolescent as regarding her less highly due to unreasonable expectations for regard. Alternatively, this association may be accounted for by a third variable not measured in this study. For example, it is possible that early-onset childhood depressive symptoms and/or general negative affectivity increases risk for mothers to perceive their daughter as regarding their mother less highly, while early-onset childhood depressive symptoms and/or general negative affectivity may also increase risk for the development of body dissatisfaction during the transition through puberty (Bearman, Martinez, & Stice, 2006). Further research to better understand why maternal perceptions of the twin's regard for her mother emerged as a significant predictor of later body dissatisfaction is necessary.

Notably, an unexpected association also emerged. Specifically, twin differences in maternal perceptions of involvement in the mother-child relationship at middle adolescence later predicted twin differences in weight preoccupation during late adolescence. Unexpectedly, the twin who was rated by her mother as having greater involvement in the mother-child relationship later reported greater weight preoccupation, as compared to her co-twin. This unexpected association is discussed further below.

Maternal involvement and weight preoccupation. Post hoc analyses that explored reasons to account for this unexpected association between greater maternal involvement and greater weight preoccupation are included in Appendix P, although no clear explanation was identified. These post hoc analyses emphasized that this unexpected association was unique to maternal

ratings of Mother Involvement at age 14 years (i.e., not generalizable to maternal ratings of Mother Involvement at age 11 years) and was not accounted for by the fact that higher maternal ratings of Mother Involvement may reflect greater discrepancies between twin and mother ratings of mother-child involvement.

This unexpected association may reflect numerous factors. For example, recognizing that descriptive statistics of the Mother Involvement scale suggested that it was normative for maternal involvement to slightly decrease from age 11 to 14 years, it is possible that greater maternal ratings of their involvement in the mother-child relationship at age 14 years reflects a lack of normative parent-child separation and individuation. Lack of normative separation-individuation during adolescence has been associated with difficulties in socioemotional adjustment and symptoms of psychopathology, including eating pathology (Marsden, Meyer, Fuller, & Waller, 2002; Rhodes & Kroger, 1992). Further, as middle-aged mothers often report high dissatisfaction with their bodies (e.g., von Ranson et al., 2005), it is also possible that daughters who remain particularly close with their mothers during adolescence develop weight preoccupation in response to observing their mothers model body dissatisfaction.

In addition, recognizing that this unexpected association was the only significant cross-lagged association with the Mother Involvement scale at age 14 across the entire study, it is possible that Mother Involvement assessed a somewhat different construct at age 14 as opposed to the other ages. For example, although the PEQ Involvement scale was intended to measure parent-child communication, closeness, and support, it is possible that Mother Involvement at age 14 also reflected aspects of maternal psychological control. Maternal psychological control is a negative quality involving intrusive, manipulative, and/or coercive parenting behaviours and general parental control of the adolescent's psychological world that has been associated with a

range of adolescent psychopathology and socioemotional problems (Barber et al., 2005; Barber & Schluterman, 2008). For example, whereas parental connection and parental knowledge about an adolescent's whereabouts are associated with lower risk for eating pathology, parental psychological control is associated with greater risk for eating pathology (Berge et al., 2014). This possible shift in the meaning of Mother Involvement assessed at age 14 years reflects a challenge in longitudinal research, wherein researchers must decide between using the same measure across time points to enhance consistent measurement of a construct versus using different measures across time points to enhance developmentally appropriate measurement of a construct. In addition, this lack of conceptual clarity reflects a greater need for clarification of the construct of parent-child involvement, a need that has been recognized throughout the greater literature on child and adolescent health and development (Barber & Schluterman, 2008). To further explore the construct of Mother Involvement, post hoc analyses in Appendix P explored different facets of the Involvement scale to consider any differential cross-lagged associations with Weight Preoccupation. However, factor analysis failed to confirm meaningful facets of parent-child involvement within the greater scale. See Appendix P for further details.

Finally, in interpreting this unexpected association it is important to recognize that Pearson's correlations between maternal ratings of Mother Involvement and Weight Preoccupation were only significant when examined as twin difference scores (see Table 9, p. 106) and not when examined as individual twin scores (see Table 8, p. 104). Indeed, when individual twin scores were examined, the association between variables was nonsignificant (r = -.07). These findings emphasize that the unexpected cross-lagged association between greater maternal ratings of Mother Involvement at age 14 and greater Weight Preoccupation at age 17 may be specific to consideration of mother-child involvement as a nonshared environmental

factor. That is, this association may be specific to differential maternal involvement with each twin rather than the absolute level of involvement in a specific mother-child relationship.

Discordant subsamples. Associations between twin differences in eating pathology and twin differences in ratings of the parent-child relationship were more robust within the discordant subsamples than the full sample. These findings suggest that associations between these variables and effects of nonshared environmental processes may be attenuated in unselected, population-based samples. Possibly, small differences in eating pathology are not associated with differences in the parent-child relationship, although larger, more clinically significant differences in eating pathology are associated with differences in the parent-child relationship. That is, associations between eating pathology and the parent-child relationship may be a function of the severity of twin differences in eating pathology. Notably, post hoc analyses compared overall levels of eating pathology between twins who were included versus excluded within the discordant sample to provide a better understanding of these differences in findings across the full and discordant samples. Results indicated that the twin who reported higher eating pathology within each twin pair included in the discordant sample tended to demonstrate greater eating pathology across time points (i.e., $M = 9.56$, $SD = 4.81$ at age 11 years; $M = 10.67$, $SD = 5.70$ at age 14 years; $M = 10.82$, $SD = 5.59$ at age 17 years) than the twin who reported greater eating pathology within each twin pair excluded from the discordant sample (i.e., $M = 4.52$, $SD = 3.67$ at age 11 years; $M = 4.14$, $SD = 4.28$ at age 14 years; $M = 5.09$, $SD = 4.73$). Thus, it is also possible that more robust associations between eating pathology and the parent-child relationship were observed within the discordant sample due to a higher absolute level of eating pathology in the most affected twin. Possibly, associations between eating

pathology and the parent-child relationship are more likely to emerge when eating pathology passes a certain threshold of severity.

Specificity of findings. In interpreting these associations between eating pathology and the parent-child relationship, it is important to recognize that associations were not accounted for by BMI. Thus, findings suggest attitudes and behaviors that comprise eating pathology, rather than body weight, are associated with the parent-child relationship. Further, similar associations were not observed between anxiety and the parent-child relationship, suggesting that the extent to which eating pathology may predict difficulties in the parent-child relationship and/or may be an outcome of difficulties in the parent-child relationship is not simply an indicator of the extent to which general psychopathology is associated with parent-child relationship difficulties. It is possible that there is something unique about eating pathology that challenges the parent-child relationship, although further research is needed to confirm and understand the specificity of associations demonstrated in the current study. Further research should also follow up the observed association between daughters' perceptions of lower involvement in the father-child relationship at age 14 years and greater anxiety at age 17 years. Previous longitudinal research has indicated similar longitudinal associations between father-child involvement and closeness and later adolescent anxiety (Bogels & Phares, 2008). Together, these findings suggest the importance of prevention and treatment strategies to strengthen the father-child relationship and provide anxiety management strategies to adolescents who report lower involvement in the father-child relationship.

Implications

Recognizing the exploratory nature of the current study, the implications of these findings should be considered with caution prior to further research that corroborates results. On

the other hand, recognizing the methodologically rigorous nature of the current design that overcame several methodological challenges that limited conclusions of past research, the potential implications of these findings should also not be dismissed. In particular, findings may have implications for understanding, assessing, and treating eating pathology, as well as for promoting healthy parent-child relationships and adolescent development.

In regard to implications for understanding eating pathology, it is important to recognize that very few significant associations suggested that a negative parent-child relationship was a risk factor for subsequent eating pathology; rather, significant associations tended to highlight that eating pathology was a risk factor for subsequent negative parent-child relationships. Such recognition may help to reduce stigma and blame that has historically been placed on parents of children with eating disorders, which may at times interfere with recognition and acceptance of their child's eating pathology and appropriate treatment-seeking for their child. For example, research indicates that the proportion of adolescents with eating disorders who seek treatment specifically for their eating pathology is low (approximately one in five; Swanson et al., 2011), and is lower than treatment-seeking observed for other adolescent psychopathology (Merikangas et al., 2011). Recognizing that adolescent treatment-seeking is often initiated by parents, reducing parental stigmatization and blame for the development of their child's eating pathology may be important for increasing treatment-seeking. If future research corroborates these findings regarding the direction of longitudinal associations between variables, greater effort should be placed on disseminating such findings at the community-level.

On the other hand, significant associations that suggested eating pathology was a risk factor for subsequent negative parent-child relationships – in particular, twin's negative perception of the father-child relationship – are important to consider. Regardless of whether the

twin or parent was rating the relationship, specific eating pathology scores at middle adolescence were negatively associated with specific parent-child relationship scores at later adolescence, although the specific scores involved differed based on the rater and the specific parent-child relationship (i.e., mother-child or father-child) being rated. These findings suggest that development of eating pathology may subsequently challenge parent-child relationships, particularly across later adolescence, suggesting the importance of supporting parent-child relationships among families of adolescents with eating pathology. As discussed by Spanos et al. (2010), given that negative family relationships impact the family's ability to care for family members with eating disorders, knowing when these effects are most likely to occur is important for treatment and prevention research. Notably, later adolescence is a period wherein risk for numerous issues (e.g., substance use, risky sexual behavior, etc.) emerges. Thus, recognizing and addressing negative parent-child relationship factors that may develop secondary to eating pathology may help promote positive adolescent development in general, beyond associations with eating pathology. Development and dissemination of community programs to help parents support their adolescents through these difficult years without reducing involvement and regard for one another may be beneficial. Providing such community programs in a format that is easily accessible for fathers may be particularly important.

In regard to assessment of eating pathology in clinical settings, the current findings suggest the importance of including family members in the assessment process and gaining both the adolescent and parents' perspectives of parent-child relationships. Both the mother-child and father-child relationship should likely be assessed to identify any parent-child relationship problems that may have developed subsequent to the eating pathology. Such assessment may provide important information regarding the adolescent's current familial environment and

support system and identify targets for potential intervention. Notably, as Eisler (2005) has emphasized, understanding how families reorganize themselves around a problem such as eating pathology is typically more important for treatment than knowing how the problem initially developed.

For adolescents receiving treatment for eating pathology, current findings suggest that intervention may benefit from considering any need to strengthen parent-child relationships. In particular, findings from the current study suggest that younger adolescents presenting to treatment may have parent-child relationship problems that are longer-standing as compared to older adolescents presenting to treatment who may have parent-child relationship problems that developed more recently, subsequent to the expression of eating pathology. It may be important to provide parents with psychoeducation related to eating pathology in order to help them understand and best respond to eating pathology symptoms and remain involved and connected in their child's life and recovery. Notably, a primary tenet of family-based therapy for eating disorders, an evidence-based treatment (Couturier et al., 2013), is to mobilize parents as a resource in their adolescent's treatment and reinvigorate parental roles in the family system while showing respect and regard for the adolescent's point of view and experience. Thus, it is possible that one of the mechanisms through which family-based therapy promotes positive change is through preventing and/or addressing the negative parent-child relationship factors that may develop subsequent to eating pathology. Findings from the current study suggest that including fathers in treatment may be particularly important.

Finally, two specific cross-lagged associations indicated parent-child relationship problems predict later eating pathology (i.e., that twin's perception of lower father regard predicts greater weight preoccupation and mother's perception of lower twin regard predicts

greater body dissatisfaction). Based on these findings, it may be important to monitor lower parent and child perceptions of parent-child regard during pre-adolescence and consider efforts to align perceptions of regard and promote more positive parent-child regard. Further, it may be important to consider other risk factors that may be present, as it is possible that such factors may serve as "third variables" that account for these associations with later eating pathology. Finally, further research could consider whether improving regard may be helpful in protecting against eating pathology. For example, it is possible that improving the father-child relationship and open communication may reduce the likelihood of becoming preoccupied with one's weight.

Strengths and Limitations

In interpreting results from the current study, limitations must be recognized. As aforementioned, it is essential to recognize that these findings do not suggest a causal relationship between eating pathology and parent-child relationship variables; however, these results identify directions of associations between variables across time.

In addition, there are limitations related to the sample included in the study. For example, the sample is predominantly Caucasian. Although comparisons to census data indicated the demographic characteristics of the sample were representative of the Minnesota population (Holdcraft & Iacono, 2004), lack of ethnic diversity within the sample may limit generalizability of findings to other ethnic groups. Further, the sample was a nonclinical, population-based sample – i.e., not a clinical sample. Although use of population-based samples is generally considered to be a research strength as it increases generalizability of findings to the greater population, it is possible that findings would not generalize to clinical samples and clinical levels of eating pathology. On the other hand, as aspects of eating pathology have been suggested to exist upon a dimension that extends from normal to abnormal (Keel, Brown, Holland, & Bodell,

2012; Wildes & Marcus, 2013), it appears more likely that associations within the current study would continue to be observed within clinical samples. Possibly, such associations may even strengthen and be more easily observed within clinical samples.

Relatedly, inclusion of twins, although essential to facilitate an MZ twin differences design, raises questions regarding generalizability of findings to non-twin samples. Opponents of twin-based methodology (e.g., Evans & Martin, 2000; Kamin & Goldberger, 2002) have argued that findings with twin samples do not generalize to singleton samples. For example, skeptics have argued that twins exhibit higher rates of prematurity, low birth weights, and perinatal complications which, in turn, can increase risk for psychopathology. Recently, the twin representativeness assumption (i.e., that twins are representative of the general population) for eating pathology and internalizing symptoms was investigated among a large sample of MZ twins, dizygotic twins, and singletons (Munn-Chernoff et al., 2013). Results indicated statistically significant differences between levels of eating pathology and internalizing symptoms reported by twins and singletons, with twins reporting lower levels of symptoms. However, generally small effect sizes led the authors to question whether differences in eating pathology and internalizing symptoms between twins and singletons reflected clinical significance and ultimately suggested results from twin samples should be considered to generalize to singletons. Similar rates of psychopathology across twin and singleton samples have also been reported in other (e.g., Kendler, 1993; Pulkkinen, Vaalamo, Hietala, Kaprio, & Rose, 2003), but not all (e.g., Klump, Keel, Leon, & Fulkerson, 1999), studies. Regardless, further research that seeks to better understand the nature in which the experience of having a twin with eating pathology may differ from the experience of having a non-twin sibling with eating pathology would be beneficial.

Further, the MZ differences design, although important to facilitate isolation of nonshared environmental processes, also has limitations. For example, this design utilizes difference scores, a practice that has frequently been criticized (e.g., Cronbach & Furby, 1970; Edwards, 2001). However, as difference scores used in the current study were calculated using scores from twin pairs assessed on the same measure administered at the same time point, some criticisms of difference scores are less relevant to this study than to other research (e.g., the difference scores do not represent change scores, are not calculated using different measures, etc.). However, a potential problem with the use of difference scores most relevant to the current study is attenuated reliability. Difference scores are usually less reliable than their component scores, with the reliability of a difference score impacted by the reliability of the component scores and the correlation between component scores. Lower reliability of difference scores attenuates their observed correlations with other variables. Thus, this limitation should be considered when interpreting results. However, recognizing the importance of isolating and examining nonshared environmental effects, and the MZ difference design is touted as a "gold standard" method for doing so, the limitations of using difference scores do not outweigh the advantages of using an MZ difference design.

Another limitation of the MZ differences design relates to the evolving, important field of epigenetics. Epigenetics is the the "reversible regulation of various genomic functions mediated principally through changes in DNA methylation and chromatin structure" (Wong et al., 2010, p. 516). Although the classical DNA sequence is not changed by epigenetic mechanisms, the *expression* of the DNA sequence is affected. Thus, despite the fact that MZ twins share 100% of their genetic code, they do not share 100% of their epigenome – i.e., the biological system that regulates gene functioning. Indeed, research suggests epigenetic differences within MZ twin

pairs may be an important contributing factor to phenotypic discordance (Hogenson, 2013). As research suggests that the epigenome can be affected by environmental factors, the distinction between genetic and environmental factors that affect phenotype becomes somewhat blurred. Thus, whereas the foundation of the MZ twin differences design lies in the assumption that differential outcomes within pairs can only be the result of nonshared environmental factors, epigenetics suggests that these differences within pairs could also be mediated, in part, by the epigenome. That is, differential exposure to nonshared environmental factors (e.g., unique stressors, diet, substances, etc.) may lead to unique changes to each twin's epigenome, subsequent differential expression of the same genetic code within pairs, and resultant phenotypic differences within pairs. Within the MZ twin differences design, epigenetic differences within pairs that resulted from unique exposure to nonshared environmental factors would not be controlled. As discussed by Vitaro et al. (2009), ignoring this issue may lead to a false conclusion of direct environmental causes to explain phenotypic differences within MZ twin pairs. On the other hand, research suggests there are "epigenetically sensitive developmental periods." In particular, the prenatal period and early postpartum period are suggested to be such periods, during which time factors such as maternal stress, nutrition, and exposure to neurotoxins can lead to epigenetic changes. Notably, exposure to such factors is likely to be very similar for MZ twins, especially those reared in the same household, making epigenetic differences resulting from such exposures less likely among twin pairs in the current study. Indeed, epigenetic research has suggested that the majority of epigenetic differences within MZ twin pairs are often not expressed and recognized until developmental periods (e.g., adulthood) that occur beyond that examined in the current study (e.g., Fraga et al., 2015). Thus, although epigenetic processes pose a limitation to the MZ twin differences design that is

important to consider, this limitation may be less relevant to the current study. In addition, to the extent that the MZ twin difference design's efforts to control for genetic factors is somewhat confounded by epigenetic processes that may alter expression of genes, most other methodological efforts to control for genetic factors would also be confounded by epigenetic processes.

Other limitations of the current study regard assessment methods. For example, assessment of eating pathology was based solely on a self-report questionnaire, and some evidence suggests interview-based assessment is superior to questionnaire-based assessment for some – but not all– symptoms of eating pathology (e.g., Black & Wilson, 1996; Fairburn & Beglin, 1994; Keel, Crow, Davis, & Mitchell, 2002). In particular, interview-based assessment may be important for assessing more complex features of binge eating and dietary restraint and for determining a clinical diagnosis. Further, assessment of eating pathology did not indicate diagnosable eating disorders, thus it remains uncertain to what degree the current findings would relate to the development of clinical eating disorders. In addition, assessment of the parent-child relationship was not based on direct observation but rather parent and daughter self-reports, raising questions as to the extent parent-child relationship scores reflect personal perceptions versus objective relationship functioning. For example, it is possible that daughters with greater eating pathology were more likely to rate their relationships with their parents as weaker due to a negative cognitive set that affected their reports. However, recognizing that some associations were also observed between eating pathology and parent reports of the parent-child relationship, it is unlikely that such a reporting bias would have entirely accounted for observed associations between eating pathology and daughter reports of the parent-child relationship. Further, even if eating pathology contributed to negatively biased perceptions of

the parent-child relationship, these biased perceptions remain important to recognize as they likely affect daughters' interactions with their parents and interpretation of parenting behaviors.

Relatedly, as both eating pathology and parent-child relationship variables were assessed via questionnaire, it is possible that associations between variables were inflated due to common method variance. Common method variance is variance that is attributable to the measurement method rather than to the constructs the measures represent, which can create an apparent association among variables generated by their common source (Podsakoff, Mackenzie, Lee, & Podsakoff, 2003). Indeed, as eating pathology was rated by the daughter, more robust associations between daughter ratings of the parent-child relationship and eating pathology compared to parent ratings of the parent-child relationship and eating pathology may be attributable, in part, to common method variance. However, recognizing that some associations were also observed between parent ratings of the parent-child relationship and eating pathology increases confidence that associations between variables were not solely attributable to common method variance.

Furthermore, the PEQ was not administered to fathers at the twins' age 14 assessment, thus cross-lagged models of father ratings of the parent-child relationship could not parallel the structure used in cross-lagged models of daughter and mother ratings of the parent-child relationship. Relatedly, large amounts of data were missing from paternal ratings of the father-child relationship at age 17. In addition, some specific PEQ scales (e.g., Mother/Father Regard for Daughter) demonstrated low reliability, likely due to low endorsement of negative parental perceptions of their children. Thus, findings pertaining to analyses of paternal reports of the father-child relationship and scales with low reliability should be interpreted with greater caution and further examined in future research.

Assessment was also complicated by lack of conceptual clarity regarding some PEQ scales. For example, it remains unclear the extent to which the PEQ Involvement scale may have reflected parental psychological control, a negative parenting dimension that has been demonstrated to have opposite associations with eating pathology in comparison to other facets of parent-child involvement such as connection and knowledge about the adolescent's whereabouts (Berge et al., 2014). Unfortunately, adequate validity research specific to the PEQ Involvement scale is lacking. A better understanding of longitudinal associations between parental psychological control and eating pathology may be helpful in understanding how to increase involvement in the parent-child relationship without increasing parental psychological control. This limitation relates to a larger limitation of the use of archival data: avenues that can be explored are limited by the variables chosen to be included within the study. Although the MTFS data set provided an excellent opportunity to consider longitudinal associations among eating pathology and the parent-child relationship among a large, population-based sample of MZ twins, several variables that may be important to consider in a truly comprehensive examination of this area were not assessed (e.g., parental psychological control, parental ratings and understanding of daughter's eating pathology, etc.).

Another limitation associated with assessment in the current study was the time at which assessment began. Specifically, data collection did not begin until twins were approximately 11 years old. Thus, mechanisms involved in associations between eating pathology and the parent-child relationship that may have onset during early or middle childhood cannot be examined. On the other hand, by beginning data collection at age 11, assessment began prior to the median age of onset of most eating disorders (i.e., approximately age 12.5 years in adolescent samples

examined [Swanson et al., 2011] and approximately age 18-21 years in adult samples examined [Hudson, Hiripi, Pope Jr., & Kessler, 2007]).

In addition, limitations concerning the strength of associations and interpretive meaning should also be recognized. Notably, the magnitude of significant cross-lagged associations was relatively small, with the earlier variable accounting for only 7% or less of the variance in the later variable. However, recognizing that approximately three years passed between each assessment, this variance is neither trivial nor unexpected. Indeed, several methodologically rigorous longitudinal studies of parenting variables and psychopathology have reported similarly small amounts of variance explained (Burt et al., 2005; Loukas, Fitzgerald, Zucker, & von Eye, 2001; Wasserman, Miller, Pinner, & Jaramillo, 1996). Further, it is pertinent to recognize that path coefficients, including both cross-lagged and stability coefficients, take into account pre-existing relationships between variables, and that associations are only indicative of nonshared environmental effects (i.e., shared environmental and genetic effects that may inflate associations are not considered). In addition, small percentages of explained variance are to be expected in MZ twin difference designs – particularly when examining intrafamilial environmental processes, due to the strict control of potential confounders (i.e., genetic and shared environmental factors; Vitaro et al., 2009). All of these issues can lead the magnitude of associations to appear smaller than expected.

Further, as aforementioned, numerous statistical analyses were included in this study without adjusting the alpha from $p < .05$, increasing the possibility of type I error. Thus, it is possible that some statistically significant results reported in the current study were uncovered by chance and do not reflect a true association between variables. However, as the current study reflects exploratory research and primary analyses (i.e., cross-lagged models) were already

statistically rigorous (e.g., used twin difference scores, controlled for both stability and within-age associations), the decision was made to retain the alpha level at $p < .05$ to avoid overlooking any associations between variables, which is consistent with previous research (e.g., Burt et al., 2006; Spanos et al., 2010).

Finally, it is important to recognize that, through the use of a MZ twin differences design to examine the parent-child relationship as a nonshared environmental process, interactions with other factors, including genetic and shared environment factors, were ignored. This is another limitation that must be considered in interpreting results. For example, recent research has reported that associations between parenting factors and eating pathology may be dependent in part on genetic makeup (e.g., van Strien, Snoek, van der Zwaluw, & Engels, 2010). It is likewise possible that the strength of association between eating pathology and the parent-child relationship depends on the shared environmental context that these variables exist within, such as socioeconomic status, parental health literacy, parental psychopathology, etc. Considering interactions with other factors is an important next research step to build upon the current findings.

Despite the aforementioned limitations of the current study, numerous strengths can also be recognized, many of which have already been discussed. For example, this study utilized a longitudinal design that considered reciprocal associations between eating pathology and the parent-child relationship, including the temporal order of variables associated across time. Twins were all approximately the same age as the rest of the sample at each assessment point, facilitating interpretation of developmental processes associated with narrowly defined age intervals that spanned pre-adolescence to late adolescence. In addition, the analyses were genetically informed, facilitating isolation of the parent-child relationship as a nonshared

environmental factor via controlling for genetics as well as shared environmental factors. Further, daughter, mother, and father ratings of the parent-child relationship were all included, facilitating consideration of multiple informants' perceptions. Moreover, daughter ratings of both the mother-child and father-child relationship were analysed, facilitating consideration of differential associations with each parent. Multiple aspects of the parent-child relationship were examined, as well as different symptoms of eating pathology, facilitating examination of different aspects of the parent-child relationship that are differentially associated with specific eating attitudes and behaviors. Eating pathology scores were considered both controlling for and not controlling for BMI, and results were compared to other symptoms of psychopathology (i.e., anxiety). In addition, a community-based sample was recruited, thereby avoiding biases associated with recruiting treatment-seeking samples.

Conclusion

In summary, the current study highlights reciprocal associations between symptoms of eating pathology and facets of the parent-child relationship that varied dependent on the rater of the relationship (i.e., daughter, mother, or father), the specific relationship being rated (i.e., mother-child or father-child relationship), and the developmental period examined (i.e., early or later adolescence). Such associations between eating pathology and parent-child relationship variables were observed when the parent-child relationship was isolated as a nonshared environmental factor and influences of genetic and shared environmental factors were controlled. Findings most consistently suggested that eating pathology by middle adolescence may present risk to the strength of the parent-child relationship – in particular, the father-child relationship – across later adolescence. However, longitudinal associations between eating pathology and the parent-child relationship were generally neither strong nor robust and no

evidence was identified to support the notion that parents are a primary mechanism underlying risk for eating pathology. Future research and clinical efforts to better understand and improve both the parent-child relationship and adolescent mental and physical well-being across adolescence should be encouraged. Recognizing the pervasive effects of eating pathology on mental and physical health (Klump, Bulik, et al., 2009) and the difficulties of effectively treating eating disorders (Hay, 2013), maintaining strong parent-child relationships within the context of emerging symptoms of eating pathology is likely important to managing symptoms and promoting positive adolescent development.

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Appendix A

Missing Data

Independent sample *t*-tests compared values on variables of interest in the current study (i.e., eating pathology, parent-child relationship, and anxiety scale and subscale scores) at each time point between participants who had available data on the variable of interest at all three time points versus only one or two time points. All test assumptions were examined and addressed. For example, only one twin from each pair was randomly selected to be included in each *t*-test to satisfy the assumption of independent cases, and data that were judged to be in violation of the assumption of normality (i.e., skewness greater than +/- 2.0 and/or kurtosis greater than +/- 3) were transformed to approximate normality. Almost no significant differences between groups were observed on any variable measured at any time point, with a few exceptions. Specifically, twin ratings of maternal regard for the twin at age 11 years were significantly lower ($p < .05$) in the group with complete data ($M = 19.14$, $SD = 1.41$) than the group with incomplete data ($M = 19.59$, $SD = .80$); twin ratings of their regard for their father at age 14 years were significantly higher ($p < .05$) in the group with complete data ($M = 28.47$, $SD = 3.37$) than the group with incomplete data ($M = 26.62$, $SD = 4.31$); and binge eating at age 14 years was significantly higher ($p < .001$) in the group with complete data ($M = .88$, $SD = 1.39$) than the group with incomplete data ($M = .28$, $SD = .46$). Comparison of means suggested minimal clinical significance associated with these differences, particularly differences in binge eating and perceptions of maternal regard for daughter. Differences in daughter regard for father may reflect the fact that daughters who regarded their father less highly may have been more likely to have a father who stopped being involved in the child's life and thus was not rated at at least one of the three time points, resulting in missing data. Overall, analyses of missing data supported the conclusion that missing data likely did not significantly bias findings in the current study.

Appendix B

Assessment Measures

Minnesota Eating Behaviors Survey (Age 11)

Items 3, 8, 12, 16, 18, and 22 comprised the Body Dissatisfaction subscale; items 2, 7, 10, 11, 14, 24, 25, and 29 comprised the Weight Preoccupation subscale; items 4, 6, 15, 19, 23, 27, and 28 comprised the Bing Eating subscale; and items 5, 9, 13, 17, 26, and 30 comprised the Compensatory Behaviors subscale.

MEBS

CIRCLE ONE

1. I can eat sweets and starches (like potatoes, pasta and bread) without feeling upset or nervous ……..	T	F
2. I often diet to control my weight…….	T	F
3. My stomach is too big…….…….	T	F
4. I eat when I'm upset about things...…….	T	F
5. I have thought about throwing up (vomiting) to lose weight…….	T	F
6. Sometimes I stuff myself with food…….	T	F
7. I think a lot about dieting (or losing weight)..…….	T	F
8. My thighs are about the right size…..…….	T	F
9. Sometimes I completely stop eating for more than a day to control my weight…….…….	T	F
10. I feel terribly guilty if I overeat..…….	T	F
11. I am really afraid of gaining weight..…..…….	T	F
12. The shape of my body is fine….…….	T	F

13. Sometimes I use laxatives (like Ex-Lax, Correctol, Phenamint, or Nature's Remedy) to control my weight…….	T	F
14. My weight is very important to me…….	T	F
15. Sometimes I eat lots and lots of food and feel like I can't stop.…….	T	F
16. My butt (behind) is too big…….…….	T	F
17. I sometimes use diet pills (like Dexatrim, Dietac, or Acutrim) to control my weight…….	T	F
18. I'm always wishing that I were thinner...…….	T	F
19. I think a lot about overeating (eating a really large amount of food)…...	T	F
20. Sometimes I have a hard time telling if I'm hungry or not…….	T	F
21. I exercise to control my weight more than other women my age...…….	T	F
22. My hips are just the right size…..…….	T	F
23. Sometimes, when I'm with other people I won't eat much, but later, when I'm alone, I'll eat a lot…...…….	T	F
24. I feel fat or stuffed even after eating a normal meal...…….	T	F
25. If I gain a pound, I worry that I will keep gaining more and more weight……...	T	F
26. Sometimes I make myself throw up (vomit) to control my weight……..…….	T	F
27. Sometimes I eat by myself so that others won't know what I'm eating….	T	F
28. When I get upset, I'm afraid that I will start eating…….	T	F
29. I often weigh myself to see if I am gaining weight…..…….	T	F
30. I sometimes use medicine that makes me lose water (diuretics like Sunril, Aqua-Ban, Pamprin, or Midol PMS) to control my weight…….	T	F

Minnesota Eating Behaviors Survey (Age 14 and 17)

MEBS

Instructions: This questionnaire contains a series of statements that you can use to describe your perceptions about eating and your body. After each item is a scale like this: T t f F. The meaning of the four possible answers is:

T = Definitely True
t = Probably True
f = Probably False
F = Definitely False

You should read each statement and decide how you feel about it. First decide if the statement is generally true or generally false of you. Then, indicate how certain you feel—is this definitely or only somewhat accurate—by circling the letter that corresponds with your answer. So, if the statement or item is definitely true for you, then you should circle the T like this: (T) t f F. If the statement or item is probably true for you (or more true than false) then you should circle the t like this: T (t) f F.

		CIRCLE ONE (1) (2) (3) (4)
1.	I can eat sweets and starches (like potatoes, pasta and bread) without feeling upset or nervous ……..	T t f F
2.	I often diet to control my weight…….	T t f F
3.	My stomach is too big…….…….	T t f F
4.	I eat when I'm upset about things...…….	T t f F
5.	I have thought about throwing up (vomiting) to lose weight…….	T t f F
6.	Sometimes I stuff myself with food…….	T t f F
7.	I think a lot about dieting (or losing weight)..…….	T t f F
8.	My thighs are about the right size…..…….	T t f F
9.	Sometimes I completely stop eating for more than a day to control my weight…….…….	T t f F

10. I feel terribly guilty if I overeat..……. T t f F

11. I am really afraid of gaining weight..…..……. T t f F

12. The shape of my body is fine….……. T t f F

13. Sometimes I use laxatives (like Ex-Lax, Correctol, Phenamint, or Nature's Remedy) to control my weight……. T t f F

14. My weight is very important to me……. T t f F

15. Sometimes I eat lots and lots of food and feel like I can't stop.……. T t f F

16. My butt (behind) is too big…….……. T t f F

17. I sometimes use diet pills (like Dexatrim, Dietac, or Acutrim) to control my weight……. T t f F

18. I'm always wishing that I were thinner...……. T t f F

19. I think a lot about overeating (eating a really large amount of food)….... T t f F

20. Sometimes I have a hard time telling if I'm hungry or not……. T t f F

21. I exercise to control my weight more than other women my age...……. T t f F

22. My hips are just the right size….……. T t f F

23. Sometimes, when I'm with other people I won't eat much, but later, when I'm alone, I'll eat a lot…...……. T t f F

24. I feel fat or stuffed even after eating a normal meal...……. T t f F

25. If I gain a pound, I worry that I will keep gaining more and more weight……... T t f F

26. Sometimes I make myself throw up (vomit) to control my weight…….……. T t f F

27. Sometimes I eat by myself so that others won't know what I'm eating…. T t f F

28. When I get upset, I'm afraid that I will start eating……. T t f F

29. I often weigh myself to see if I am gaining weight…..……. T t f F

30. I sometimes use medicine that makes me lose water (diuretics like Sunril, Aqua-Ban, Pamprin, or Midol PMS) to control my weight……. T t f F

Parental Environment Questionnaire (Age 11, 14, and 17)

There are numerous informant variants of the Parental Environment Questionnaire: e.g., each twin reports on her relationship with each parent separately and each parent reports on his/her relationship with each twin separately. Items were essentially the same across the versions used for each informant, except wording was changed to be appropriate for each particular rater and the parent-child relationship being rated. The PEQ parent version is presented below.

Items 2, 4, 6, 7, 18, 23, 25, 29, 40 41, 46, and 50 comprised the Involvement scale; items 1, 9, 11, 21, 24, 35, 39, and 42 comprised the Daughter Regard for Parent scale; and items 8, 22, 34, 37, and 43 comprised the Parent Regard for Daughter scale.

Parental Environment Questionnaire
Mother's and Father's Ratings
PEQ

These items are statements that you might use to describe your twin and their relationships with you. In front of each item is a scale like this:

T t f F

For each item you should circle one answer. The meaning of each letter is given below:

T = Definitely True
t = Probably True
f = Probably False
F = Definitely False

So if the statement is definitely true regarding your relationship with your child, you should circle the T in the column like this:

(T) t f F

If the statement or item is probably true regarding your relationship with your child (or more true than false), then you should circle the t in the column like this:

T (t) f F

PLEASE ANSWER EVERY QUESTION EVEN IF YOU ARE NOT SURE WHICH ANSWER IS RIGHT FOR YOU.

T = Definitely True
t = Probably True
f = Probably False
F = Definitely False

The first set of questions well be about your relationship with your twin named _____.

Circle one letter for each item as they apply to your relationship with _____ only. You will be asked about your relationship with your other twin later.

1	2	3	4	
T	t	f	F	1. This child can learn a lot of things from me.
T	t	f	F	2. This child talks about his/her concerns and experiences with me.
T	t	f	F	3. I often criticize this child.
T	t	f	F	4. I praise this child when he/she does something well.
T	t	f	F	5. I often interrupt this child before he/she can finish saying anything.
T	t	f	F	6. I don't know about this child's hobbies.
T	t	f	F	7. This child doesn't want his/her friends to meet me.
T	t	f	F	8. I love this child no matter what he/she does.
T	t	f	F	9. I often give advice to this child.
T	t	f	F	10. I often irritate this child.
T	t	f	F	11. I have taught this child useful things.
T	t	f	F	12. I am very much the same from one day to the next with this child.
T	t	f	F	13. Often there are misunderstandings between this child and me.
T	t	f	F	14. I only pay attention to this child when he/she has done something wrong.
T	t	f	F	15. I don't give the support this child needs.

T t f F 16. I always want this child to decide above all what is right and wrong, and do what is right.

T t f F 17. I seem to ignore this child's feelings.

T t f F 18. I comfort this child when he/she is discouraged or has disappointment.

T t f F 19. This child treats others with more respect than he/she treats me.

T t f F 20. I often hurt this child's feelings.

T t f F 21. I make a good impression on this child's friends.

T t f F 22. I don't think highly of this child.

T t f F 23. This child and I don't have much to talk about when we are together.

T t f F 24. This child wants to be like me in a number of ways.

T t f F 25. I try to keep up with how well this child does in school or on the job.

T t f F 26. I make it clear what I want this child to do or not do.

T t f F 27. I do not trust this child to make his/her own decisions.

T t f F 28. This child and I often get into arguments.

T t f F 29. This child prefers not to talk about his/her personal problems with me.

T t f F 30. This child often angers or annoys me.

T t f F 31. I often lose my temper with this child.

T t f F 32. I sometimes hit this child in ager.

T t f F 33. It is important to me that this child obeys the law.

T t f F 34. This child knows I love him/her.

T t f F 35. This child really likes me.

T t f F 36. Once in a while this child has been really scared of me.

T t f F 37. I am proud of this child.

T t f F 38. I want this child to go to bed at a certain time before school or work days.

T t f F 39. This child is proud of me.

T t f F 40. I don't seem to know much about how this child is doing in school.

T t f F 41. This child and I do not do a lot of things together.

T t f F 42. This child respects me.

T t f F 43. I like others in the family better than this child.

T t f F 44. My child gets along well with me.

T t f F 45. If this child asks for something he/she wants but doesn't really need, I will usually say 'no."

T t f F 46. This child doesn't seem to feel very close to me.

T t f F 47. I follow through on promises to this child.

T t f F 48. I am pretty strict with this child.

T t f F 49. When this child is given a job, I expect him/her to finish is by him/herself.

T t f F 50. I don't know much about how this child spends his/her spare time.

State-Trait Anxiety Inventory – State Anxiety (Age 11 and 14)

HOW-I-FEEL QUESTIONNAIRE

Developed by C. D. Spielberger, C. D. Edwards, J. Montouri and R. Lushene

STAIC FORM C-1

DIRECTIONS: A number of statements which boys and girls use to describe themselves are given below. Read each statement carefully and decide how you feel *right now*. Then put an X in the box right in front of the word or phrase which best describes how you feel. There are no right or wrong answers. Do not spend too much time on any one statement. Remember, find the word or phrase which best describes how you feel right now, *at this very moment.*

1.	I feel	□	very calm	□	calm	□	not calm
2.	I feel	□	very upset	□	upset	□	not upset
3.	I feel	□	very pleasant	□	pleasant	□	not pleasant
4.	I feel	□	very nervous	□	nervous	□	not nervous
5.	I feel	□	very jittery	□	jittery	□	not jittery
6.	I feel	□	very rested	□	rested	□	not rested
7.	I feel	□	very scared	□	scared	□	not scared
8.	I feel	□	very relaxed	□	relaxed	□	not relaxed
9.	I feel	□	very worried	□	worried	□	not worried
10.	I feel	□	very satisfied	□	satisfied	□	not satisfied
11.	I feel	□	very frightened	□	frightened	□	not frightened
12.	I feel	□	very happy	□	happy	□	not happy
13.	I feel	□	very sure	□	sure	□	not sure
14.	I feel	□	very good	□	good	□	not good
15.	I feel	□	very troubled	□	troubled	□	not troubled
16.	I feel	□	very bothered	□	bothered	□	not bothered

17.	I feel	□	very nice	□	nice	□ not nice
18.	I feel	□	very terrified	□	terrified	□ not terrified
19.	I feel	□	very mixed-up	□	mixed-up	□ not mixed-up
20.	I feel	□	very cheerful	□	cheerful	□ not cheerful

State-Trait Anxiety Inventory – State Anxiety (Age 17)

SLEF-EVALUATION QUESTIONNAIRE

Developed by Charles D. Spielberger

in collaboration with

R. L. Gorsuch, R. Lushene, P. R. Vagg, and G. A. Jacobs

STAIC FORM Y-1

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you feel *right* now, that is, *at this moment*. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

1 = Not at all

2 = Somewhat

3 = Moderately so

4 = Very much so

1.	I feel calm	1	2	3	4
2.	I feel secure	1	2	3	4
3.	I am tense	1	2	3	4
4.	I feel strained	1	2	3	4
5.	I feel at ease	1	2	3	4
6.	I feel upset	1	2	3	4
7.	I am presently worrying over possible misfortunes	1	2	3	4
8.	I feel satisfied	1	2	3	4
9.	I feel frightened	1	2	3	4
10.	I feel comfortable	1	2	3	4
11.	I feel self-confident	1	2	3	4
12.	I feel nervous	1	2	3	4
13.	I am jittery	1	2	3	4
14.	I feel indecisive	1	2	3	4
15.	I am relaxed	1	2	3	4

16.	I feel content	1	2	3	4
17.	I am worried	1	2	3	4
18.	I feel confused	1	2	3	4
19.	I feel steady	1	2	3	4
20.	I feel pleasant	1	2	3	4

Appendix C

Internal Consistency Reliability

As presented in Table C1 (see below), MEBS scales and subscales demonstrated adequate reliability in the current sample. Internal consistency reliability of the MEBS Total scale was particularly strong across time points (α = .86 - .94).

Twin ratings of PEQ scales also demonstrated adequate reliability in the current sample, with a few exceptions. For example, the reliability of twin ratings of their Regard for Mother and their perceptions of parental Regard for Daughter tended to be low when assessed at age 11 years, which is likely attributable, in part, to decreased variability among responses to these scales (see Table 1, p. 49). In regards to parent ratings of PEQ scales, reliability also tended to be adequate in the current sample, with the exception of the Regard for Daughter subscale. Specifically, parent ratings of Regard for Daughter demonstrated poor reliability (α = .40 - .69), particularly when rated by mothers at age 11 years. Poor reliability is likely attributable, in part, to low variability among responses to this scale that reflected infrequent negative responses (see Table 7, p. 100).

Finally, the STAI State scale demonstrated adequate internal reliability across time points (α = .83-.91) in the current sample.

Table C1. Internal consistency reliability coefficients for all measures administered across time points and raters.

	Age 11 (α)	Age 14 (α)	Age 17 (α)
Twin Ratings			
MEBS Total	.86	.94	.93

MEBS Body Dissatisfaction	.82	.90	.90
MEBS Weight Preoccupation	.79	.90	.89
MEBS Binge Eating	.61	.81	.81
PEQ Mother Regard for Daughter	.58	.66	.78
PEQ Father Regard for Daughter	.58	.79	.81
PEQ Daughter Regard for Mother	.58	.84	.84
PEQ Daughter Regard for Father	.79	.88	.90
PEQ Mother Involvement	.71	.80	.86
PEQ Father Involvement	.79	.85	.91
STAI State	.85	.83	.91
Parent Ratings			
PEQ Mother Regard for Daughter	.40	.62	.55
PEQ Father Regard for Daughter	.62	-	.69
PEQ Daughter Regard for Mother	.79	.80	.77
PEQ Daughter Regard for Father	.79	-	.80
PEQ Mother Involvement	.75	.79	.83
PEQ Father Involvement	.80	-	.79

Note. MEBS = Minnesota Eating Behaviors Survey; PEQ = Parental Environment Questionnaire; STAI = State Trait Anxiety Inventory.

Appendix D

Expectation Maximization Algorithm

The Expectation Maximization (EM) algorithm is a single imputation method for missing data. When used in analyses in the current study, the following variables at each time point were used to predict missing data: daughter, mother, and father ratings of the Parental Environment Questionnaire (PEQ) Parent Regard for Daughter, PEQ Daughter Regard for Parent, and PEQ Involvement scales; and daughter ratings of the Minnesota Eating Behaviors Survey (MEBS) Total score, Body Dissatisfaction, Weight Preoccupation, and Binge Eating subscales.

The EM algorithm assumes that data are normally distributed; thus, any variables with non-normal distributions were transformed to approximate normality prior to predicting scores. In addition, the EM algorithm assumes missing data is either Missing Completely At Random or Missing At Random as opposed to Missing Not At Random. Data that is Missing Not At Random may have resulted if participants failed to respond at any time-point because they were elevated on one or more of the outcome variables assessed (e.g., PEQ or MEBS scales/subscales). Of course, as the data missing from analyses is not available to be examined, it is not possible to determine this. However, independent sample *t*-tests that compared values on variables of interest in the current study at each time point between participants with versus without missing data revealed almost no significant differences on any variable (see Appendix A). Therefore, it was decided that using EM to impute missing values was appropriate.

Appendix E

Clinical Cutoff Scores for the Minnesota Eating Behaviors Survey

Table E1. Clinical cutoff scores for the Minnesota Eating Behaviors Survey used to identify clinical levels of eating pathology.

MEBS scale/subscale	Clinical cutoff score
Total	15.55
Body Dissatisfaction	3.95
Weight Preoccupation	6.11
Binge Eating	2.53

Note. Clinical cutoffs were identified by von Ranson and colleagues (2005) by calculating the mean MEBS scores for subsamples of female adolescents who met DSM-IV criteria for lifetime anorexia nervosa or bulimia nervosa. MEBS = Minnesota Eating Behaviors Survey.

Appendix F

Correlational Analyses of Eating Pathology and Twin Ratings of the Parent-Child Relationship Using Transformed Data for Variables with Non-Normal Distributions

Table F1. Within-twin correlations of eating pathology and twin ratings of the parent-child relationship using transformed data for variables with non-normal distributions (*N* = 223).

	Age 11				Age 14				Age 17			
	Total	BD	WP	Binge	Total	BD	WP	Binge	Total	BD	WP	Binge
PEQ Mother Regard for Daughter												
Age 11	.23**	.23**	.16*	.22**	.19**	.22**	.12	.14	.18*	.17*	.16*	.08
Age 14	-.09	-.11	-.06	.02	-.16*	-.18*	-.07	-.16*	-.05	-.07	-.04	.09
Age 17	.08	.05	.05	.02	.16*	.14	.10	.15	.18*	.14	.09	.21**
PEQ Father Regard for Daughter												
Age 11	.24**	.19**	.15*	.22**	.20**	.19*	.14	.14	.15	.13	.14	.07
Age 14	-.04	-.05	-.03	.01	-.15	-.12	-.06	-.16*	.02	.04	.00	.08
Age 17	.07	.06	.03	.09	.19*	.16*	.18*	.11	.27**	.17*	.22**	.25**
PEQ Daughter Regard for Mother												

Age 11	-.21**	-.14	-.17*	-.23**	-.20**	-.10	-.13	-.22**	-.24**	-.06	-.23**	-.27**
Age 14	-.02	-.03	.02	-.02	-.03	-.06	.05	-.03	-.04	-.14	-.04	.14
Age 17	.07	.01	.03	.07	.07	.06	-.02	.16*	-.01	-.02	-.05	.09
PEQ Daughter Regard for Father												
Age 11	-.26**	-.14*	-.19**	-.22**	-.19**	-.12	-.14	-.20**	-.10	-.03	-.11	-.17*
Age 14	-.01	-.02	-.03	.00	-.06	-.09	-.01	-.01	.08	.03	.06	.14
Age 17	-.01	.01	-.01	-.04	-.05	-.06	-.06	.00	-.05	-.01	-.07	-.03
PEQ Mother Involvement												
Age 11	-.10	-.09	-.06	-.15*	-.07	-.03	-.02	-.15*	-.10	.02	-.09	-.21**
Age 14	-.04	-.05	.00	.01	-.06	-.09	.06	-.15	-.04	-.08	.01	-.03
Age 17	-.05	-.01	-.02	-.05	-.14	-.12	-.04	-.19*	-.10	-.07	-.01	-.22**
PEQ Father Involvement												
Age 11	.17*	.12	.14*	.16*	.11	.08	.05	.11	.04	-.03	.03	.15
Age 14	-.08	-.06	-.08	-.01	-.19*	-.18*	-.08	-.22*	-.04	.03	-.03	.15
Age 17	-.06	-.01	-.06	-.07	-.19*	-.13	-.16*	-.13	-.15	-.04	-.13	-.21**

Note. Data transformations were selected based on the type of skew reflected in each variable's distribution. To account for a negative skew, PEQ variables were transformed using log10(k-x), wherein k = maximum x value + 1. To account for a positive skew, MEBS

variables were transformed using log10(x+1). Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, PEQ = Parental Environment Questionnaire. *$p < .05$, **$p < .01$.

Appendix G

Cross-Lagged Analyses of Eating Pathology and Twin Ratings of the Parent-Child Relationship

Table G1. Analyses of cross-lagged models of twin difference scores for eating pathology and twin ratings of the parent-child relationship (N = 223).

	Standardized path coefficients (early adolescence)		Standardized path coefficients (late adolescence)		Fit Indices		
	PEQ 11 → Total 14 (b21)	Total 11 → PEQ 14 (b12)	PEQ 14 → Total 17 (b43)	Total 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Mother Regard for Daughter	-.03	-.12	.05	-.10	2.16	.00	1.0
PEQ Father Regard for Daughter	-.07	.03	.07	-.14	5.06	.03	.99
PEQ Daughter Regard for Mother	-.02	-.05	.06	-.07	.54	.00	1.0
PEQ Daughter Regard for Father	-.02	.00	.05	-.07	1.37	.00	1.0
PEQ Mother Involvement	-.04	-.04	.07	-.10	1.64	.00	1.0
PEQ Father Involvement	-.02	-.03	.11	-.17*	.74	.00	1.0
	PEQ 11 → BD 14 (b21)	BD 11 → PEQ 14 (b12)	PEQ 14 → BD 17 (b43)	BD 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI

PEQ Mother Regard for Daughter	.01	-.11	.02	-.07	9.94*	.08	.85
PEQ Father Regard for Daughter	.01	-.02	.09	-.01	13.35**	.10	.75
PEQ Daughter Regard for Mother	-.03	.03	-.05	-.05	6.93	.06	.92
PEQ Daughter Regard for Father	-.01	-.06	-.03	-.01	6.73	.06	.92
PEQ Mother Involvement	-.04	-.03	-.01	-.04	8.29	.07	.90
PEQ Father Involvement	-.04	-.09	.09	.02	7.28	.06	.91
	PEQ 11 → WP 14 (b21)	WP 11 → PEQ 14 (b12)	PEQ 14 → WP 17 (b43)	WP 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Mother Regard for Daughter	-.08	-.10	.00	-.09	2.13	.00	1.0
PEQ Father Regard for Daughter	-.15*	.01	-.04	-.13	4.59	.03	.99
PEQ Daughter Regard for Mother	.03	-.08	-.02	-.02	.35	.00	1.0
PEQ Daughter Regard for Father	.03	-.04	-.04	-.11	3.50	.00	1.0
PEQ Mother Involvement	.01	-.05	.01	-.04	1.10	.00	1.0
PEQ Father Involvement	.04	-.03	-.01	-.11	1.38	.00	1.0
	PEQ 11 → Binge 14 (b21)	Binge 11 → PEQ 14 (b12)	PEQ 14 → Binge 17 (b43)	Binge 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI

PEQ Mother Regard for Daughter	-.04	-.04	.10	-.05	2.64	.00	1.0
PEQ Father Regard for Daughter	-.06	.04	.03	-.10	8.62	.07	.80
PEQ Daughter Regard for Mother	-.12	-.06	.16	-.12	.76	.00	1.0
PEQ Daughter Regard for Father	-.13	.01	.08	-.08	1.18	.00	1.0
PEQ Mother Involvement	.00	-.01	.09	-.13	.98	.00	1.0
PEQ Father Involvement	-.05	.00	.11	-.20**	.65	.00	1.0

Note. PEQ = Parental Environment Questionnaire, Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, RMSEA = Root Mean Square Error of Approximation; CFI = Comparative Fit Index. See Figure 1 (p. 44) for a figural representation of paths b21, b12, b43, and b34. *p < .05, **p < .01.

Appendix H

Cross-Lagged Analyses of Eating Pathology and Twin Ratings of the Parent-Child Relationship within Discordant Subsamples

Table H1. Analyses of cross-lagged models of twin difference scores for eating pathology and twin ratings of the parent-child relationship among twin pairs discordant for eating pathology (*N* = 106-114).

	Standardized path coefficients (early adolescence)		Standardized path coefficients (late adolescence)		Fit Indices		
	PEQ 11 → Total 14 (b21)	Total 11 → PEQ 14 (b12)	PEQ 14 → Total 17 (b43)	Total 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Mother Regard for Daughter	-.03	-.25*	.13	-.11	2.57	.00	1.0
PEQ Father Regard for Daughter	-.07	-.09	.08	-.21*	2.17	.00	1.0
PEQ Daughter Regard for Mother	.00	-.08	.16	-.06	.59	.00	1.0
PEQ Daughter Regard for Father	.04	-.12	.05	-.20*	.34	.00	1.0
PEQ Mother Involvement	-.05	-.02	.14	-.13	1.81	.00	1.0
PEQ Father Involvement	-.03	-.08	.13	-.27*	.89	.00	1.0
	PEQ 11 → BD 14 (b21)	BD 11 → PEQ 14 (b12)	PEQ 14 → BD 17 (b43)	BD 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI

PEQ Mother Regard for Daughter	.02	-.14	.03	-.11	5.21	.05	.96
PEQ Father Regard for Daughter	.03	-.03	.10	-.06	6.66	.08	.90
PEQ Daughter Regard for Mother	-.06	.03	-.07	-.08	6.09	.07	.93
PEQ Daughter Regard for Father	-.05	-.08	-.04	-.06	3.88	.00	1.0
PEQ Mother Involvement	-.02	-.03	-.01	-.06	5.52	.06	.95
PEQ Father Involvement	-.02	-.09	.11	-.03	5.23	.05	.96
	PEQ 11 → WP 14 (b21)	WP 11 → PEQ 14 (b12)	PEQ 14 → WP 17 (b43)	WP 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Mother Regard for Daughter	-.09	-.16	.01	-.12	3.82	.00	1.0
PEQ Father Regard for Daughter	-.18*	-.07	-.19	-.23*	2.82	.00	1.0
PEQ Daughter Regard for Mother	.04	-.10	.03	.05	1.07	.00	1.0
PEQ Daughter Regard for Father	.06	-.10	-.05	-.18	3.42	.00	1.0
PEQ Mother Involvement	.01	-.08	.04	-.03	1.25	.00	1.0
PEQ Father Involvement	.08	-.12	-.10	-.20	2.62	.00	1.0
	PEQ 11 → Binge 14 (b21)	Binge 11 → PEQ 14 (b12)	PEQ 14 → Binge 17 (b43)	Binge 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI

PEQ Mother Regard for Daughter	-.03	-.03	.10	-.06	6.23	.07	.60
PEQ Father Regard for Daughter	-.06	.07	.01	-.15	15.53**	.16	.50
PEQ Daughter Regard for Mother	-.12	-.08	.17	-.14	1.83	.00	1.0
PEQ Daughter Regard for Father	-.05	-.01	.03	.14	4.26	.03	1.0
PEQ Mother Involvement	.07	-.03	.06	-.18*	.52	.00	1.0
PEQ Father Involvement	.00	-.01	.14	-.26*	1.01	.00	1.0

Note. Discordant subsamples are based on discordance within-twin pairs on the MEBS total scale and subscales. PEQ = Parental Environment Questionnaire, Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, RMSEA = Root Mean Square Error of Approximation, CFI = Comparative Fit Index. See Figure 1 (p. 44) for a figural representation of paths b21, b12, b43, and b34. *$p < .05$, **$p < .01$.

Appendix I

Cross-Lagged Analyses of Eating Pathology and Twin Ratings of the Parent-Child Relationship Controlling for Body Mass Index

Table I1. Analyses of cross-lagged models of twin difference scores for eating pathology and twin ratings of the parent-child relationship, wherein the effect of body mass index on eating pathology is controlled (*N* = 223).

	Standardized path coefficients (early adolescence)		Standardized path coefficients (late adolescence)		Fit Indices		
	PEQ 11 → Total 14 (b21)	Total 11 → PEQ 14 (b12)	PEQ 14 → Total 17 (b43)	Total 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Mother Regard for Daughter	-.02	-.12	.05	-.11	2.49	.00	1.0
PEQ Father Regard for Daughter	-.07	.03	.06	-.14	5.16	.04	.98
PEQ Daughter Regard for Mother	-.02	-.04	.08	-.08	.57	.00	1.0
PEQ Daughter Regard for Father	-.02	.00	.06	-.10	1.39	.00	1.0
PEQ Mother Involvement	-.03	-.04	.07	-11	1.64	.00	1.0
PEQ Father Involvement	-.02	-.03	.12	-.16*	.68	.00	1.0
	PEQ 11 → BD 14 (b21)	BD 11 → PEQ 14 (b12)	PEQ 14 → BD 17 (b43)	BD 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI

PEQ Mother Regard for Daughter	.01	-.12	.02	-.08	10.62*	.09	.83
PEQ Father Regard for Daughter	.01	-.02	.09	-.01	14.06**	.11	.72
PEQ Daughter Regard for Mother	-.03	.03	-.04	-.07	7.15	.06	.91
PEQ Daughter Regard for Father	-.03	-.06	-.01	.00	7.13	.06	.90
PEQ Mother Involvement	-.03	-.03	.00	-.04	8.84	.07	.89
PEQ Father Involvement	-.04	-.09	.09	.03	7.80	.07	.90
	PEQ 11 → WP 14 (b21)	WP 11 → PEQ 14 (b12)	PEQ 14 → WP 17 (b43)	WP 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Mother Regard for Daughter	-.07	-.10	.01	-.10	2.56	.00	1.0
PEQ Father Regard for Daughter	-.15*	.00	-.05	-.13	4.73	.03	.99
PEQ Daughter Regard for Mother	.04	-.08	.00	-.03	.61	.00	1.0
PEQ Daughter Regard for Father	.02	-.04	-.01	-.10	3.29	.00	1.0
PEQ Mother Involvement	.02	-.05	.02	-.04	1.28	.00	1.0
PEQ Father Involvement	.04	-.03	-.01	-.11	1.50	.00	1.0
	PEQ 11 → Binge 14 (b21)	Binge 11 → PEQ 14 (b12)	PEQ 14 → Binge 17 (b43)	Binge 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI

PEQ Mother Regard for Daughter	-.04	-.03	.13	-.03	2.54	.00	1.0
PEQ Father Regard for Daughter	-.06	.04	.03	-.10	8.55	.07	.80
PEQ Daughter Regard for Mother	-.12	-.06	.16	-.13	.79	.00	1.0
PEQ Daughter Regard for Father	-.13	.01	.09	-.08	1.19	.00	1.0
PEQ Mother Involvement	.01	-.01	.10	-.13	.99	.00	1.0
PEQ Father Involvement	-.05	.00	.11	-.20**	.66	.00	1.0

Note. PEQ = Parental Environment Questionnaire, Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, RMSEA = Root Mean Square Error of Approximation, CFI = Comparative Fit Index. See Figure 1 (p. 44) for a figural representation of paths b21, b12, b43, and b34. *p < .05, **p < .01.

Appendix J

Correlational Analyses of Anxiety and Twin Ratings of the Parent-Child Relationship Using Transformed Data for Variables with Non-Normal Distributions

Table J1. Within-twin correlations of anxiety and twin ratings of the parent-child relationship using transformed data for variables with non-normal distributions (N = 223).

	STAI State Subscale Age 11	STAI State Subscale Age 14	STAI State Subscale Age 17
PEQ Mother Regard for Daughter			
Age 11	.21**	.20**	.17*
Age 14	-.14	-.22**	-.12
Age 17	.24**	.24**	.44**
PEQ Father Regard for Daughter			
Age 11	.21**	.21**	.19*
Age 14	-.23**	-.25**	-.15
Age 17	.25**	.22**	.37**
PEQ Daughter Regard for Mother			
Age 11	-.17*	-.26**	-.18*

Age 14	-.14	-.16	-.11
Age 17	.24**	.18*	.29**
PEQ Daughter Regard for Father			
Age 11	.13	.23**	.20*
Age 14	-.22**	-.18*	-.13
Age 17	-.13	-.02	-.18*
PEQ Mother Involvement			
Age 11	-.12	-.16*	-15
Age 14	-.16*	-.17*	-.13
Age 17	-.19**	-.10	-.38**
PEQ Father Involvement			
Age 11	.08	.06	.11
Age 14	-.22**	-.26**	-.20*
Age 17	-.20**	-.10	-.25**

Note. STAI = State Trait Anxiety Inventory. *$p < .05$, **$p < .01$.

Appendix K

Cross-Lagged Analyses of Anxiety and Twin Ratings of the Parent-Child Relationship

Table K1. Analyses of cross-lagged models of twin difference scores for anxiety and twin ratings of the parent-child relationship (*N* = 223).

	Standardized path coefficients (early adolescence)		Standardized path coefficients (late adolescence)		Fit Indices		
	PEQ 11 → STAI 14 (b21)	STAI 11 → PEQ 14 (b12)	PEQ 14 → STAI 17 (b43)	STAI 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Mother Regard for Daughter	-.03	-.08	-.04	-.09	3.00	.00	1.0
PEQ Father Regard for Daughter	.02	-.08	-.01	-.14	8.10	.07	.85
PEQ Daughter Regard for Mother	-.10	-.03	-.04	.00	.92	.00	1.0
PEQ Daughter Regard for Father	-.07	-.16	.01	.07	2.72	.00	1.0
PEQ Mother Involvement	-.02	.02	-.14	.06	3.77	.00	1.0
PEQ Father Involvement	.07	-.15	-.20*	.01	6.58	.05	.93

Note. STAI = State Trait Anxiety Inventory. RMSEA = Root Mean Square Error of Approximation, CFI = Comparative Fit Index. See Figure 15 (p. 86) for a figural representation of paths b21, b12, b43, and b34. *$p < .05$, **$p < .01$.

Appendix L

Correlational Analyses of Eating Pathology and Parent Ratings of the Parent-Child Relationship Using Transformed Data for Variables with Non-Normal Distributions

Table L1. Within-twin correlations of eating pathology and parent ratings of the parent-child relationship using transformed data for variables with non-normal distributions (N = 223).

	Age 11				Age 14				Age 17			
	Total	BD	WP	Binge	Total	BD	WP	Binge	Total	BD	WP	Binge
PEQ Mother Regard for Daughter												
Age 11	-.06	-.04	-.07	-.05	-.07	-.05	-.08	-.09	.05	.06	.04	.05
Age 14	.02	-.04	-.01	.04	.01	.05	-.02	.03	-.09	-.04	-.09	-.10
Age 17	-.05	-.05	-.05	-.02	-.09	-.10	-.06	-.10	.04	.09	.02	-.03
PEQ Father Regard for Daughter												
Age 11	.00	-.02	.01	-.07	.10	.01	.15	-.01	-.02	-.06	.03	-.09
Age 14	-	-	-	-	-	-	-	-	-	-	-	-
Age 17	.21*	.21*	.17	.13	.16	.16	.11	.13	.06	.02	.07	.09
PEQ Daughter Regard for Mother												

Age 11	.04	.04	.02	.04	.03	.03	.09	-.11	-.07	-.04	-.07	-.09
Age 14	.04	.07	.05	-.01	-.01	.01	.02	-.05	.00	.00	-.03	.06
Age 17	.05	.07	.06	.02	.01	.08	.00	.02	.01	.06	-.03	.02
PEQ Daughter Regard for Father												
Age 11	.08	.05	.06	.07	.00	.02	.00	.04	-.01	.03	-.06	.01
Age 14	-	-	-	-	-	-	-	-	-	-	-	-
Age 17	-.21*	-.21*	-.26**	.03	-.10	-.13	-.05	-.07	-.06	-.07	-.04	-.04
PEQ Mother Involvement												
Age 11	-.05	-.04	-.06	.00	-.07	-.05	-.11	-.03	.08	.16*	.02	.08
Age 14	-.10	-.05	-.06	-.05	-.10	-.10	-.02	-.14	-.13	-.13	-.07	-.13
Age 17	-.03	-.03	-.05	.03	.01	.00	.02	.07	-.10	-.08	-.10	-.11
PEQ Father Involvement												
Age 11	-.01	-.05	.03	-.03	-.08	-.08	-.08	.04	-.12	-.10	-.13	-.02
Age 14	-	-	-	-	-	-	-	-	-	-	-	-
Age 17	-.16	-.17	-.18	-.03	-.10	-.07	-.12	-.03	-.03	-.01	-.04	-.03

Note. Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, PEQ = Parental Environment Questionnaire. *$p < .05$, **$p < .01$.

Appendix M

Cross-Lagged Models for Eating Pathology and Maternal Ratings of the Parent-Child Relationship

Table M1. Analyses of cross-lagged models with twin difference scores for eating pathology and maternal ratings of the parent-child relationship (N = 223).

	Standardized path coefficients (early adolescence)		Standardized path coefficients (late adolescence)		Fit Indices		
	PEQ 11 → Total 14 (b21)	Total 11 → PEQ 14 (b12)	PEQ 14 → Total 17 (b43)	Total 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Daughter Regard for Mother	-.04	-.09	.00	-.02	3.32	.00	1.0
PEQ Mother Involvement	.06	-.11	.15	-.21*	2.35	.00	1.0
	PEQ 11 → BD 14 (b21)	BD 11 → PEQ 14 (b12)	PEQ 14 → BD 17 (b43)	BD 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Daughter Regard for Mother	-.15*	-.11	-.07	-.05	8.29	.07	.87
PEQ Mother Involvement	.06	-.06	.13	-.11	12.46*	.10	.60
	PEQ 11 → WP 14 (b21)	WP 11 → PEQ 14 (b12)	PEQ 14 → WP 17 (b43)	WP 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI

PEQ Daughter Regard for Mother	.06	-.09	.04	.00	2.77	.00	1.0
PEQ Mother Involvement	.10	-.04	.24**	-.25**	1.89	.00	1.0
	PEQ 11 → Binge 14 (b21)	Binge 11 → PEQ 14 (b12)	PEQ 14 → Binge 17 (b43)	Binge 14 → PEQ 17 (b34)	χ^2	RMSEA	CFI
PEQ Daughter Regard for Mother	-.06	-.01	.03	.09	5.75	.04	.88
PEQ Mother Involvement	-.06	-.04	-.09	.01	3.17	.00	1.0

Note. PEQ = Parental Environment Questionnaire, Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, RMSEA = Root Mean Square Error of Approximation, CFI = Comparative Fit Index. See Figure 1 (p. 44) for a figural representation of paths b21, b12, b43, and b34. *p < .05, **p < .01.

Appendix N

Cross-Lagged Analyses of Eating Pathology and Paternal Ratings of the Parent-Child Relationship

Table N1. Analyses of cross-lagged models with twin difference scores for eating pathology and paternal ratings of the parent-child relationship with cases missing paternal ratings at age 17 excluded (*N* = 106).

	Standardized path coefficients	
	PEQ 11 → Total 17 (b21)	Total 11 → PEQ 17 (b12)
PEQ Daughter Regard for Father	.06	-.17
PEQ Father Involvement	.12	-.18
	PEQ 11 → BD 17 (b21)	BD 11 → PEQ 17 (b12)
PEQ Daughter Regard for Father	.02	-.26*
PEQ Father Involvement	.03	-.20
	PEQ 11 → WP 17 (b21)	WP 11 → PEQ 17 (b12)
PEQ Daughter Regard for Father	.14	-.02
PEQ Father Involvement	.18	-.05
	PEQ 11 → Binge 17 (b21)	Binge 11 → PEQ 17 (b12)
PEQ Daughter Regard for Father	-.01	.00
PEQ Father Involvement	-.06	-.01

Note. PEQ = Parental Environment Questionnaire, Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, RMSEA = Root Mean Square Error of Approximation, CFI = Comparative Fit Index. See Figure 17 (p. 98) for a figural representation of paths b21, b12, b43, and b34. *$p < .05$, **$p < .01$.

Appendix O

Cross-Lagged Analyses of Eating Pathology and Paternal Ratings of the Parent-Child Relationship Using Full Information Maximum Likelihood to Account for Missing Data

Table O1. Analyses of cross-lagged models with twin difference scores for eating pathology and paternal ratings of the parent-child relationship using Full Information Maximum Likelihood to account for missing data (*N* = 223).

	Standardized path coefficients	
	PEQ 11 → Total 17 (b21)	Total 11 → PEQ 17 (b12)
PEQ Daughter Regard for Father	-.07	-.19
PEQ Father Involvement	-.12	-.20
	PEQ 11 → BD 17 (b21)	BD 11 → PEQ 17 (b12)
PEQ Daughter Regard for Father	-.08	-.27*
PEQ Father Involvement	-.09	-.21
	PEQ 11 → WP 17 (b21)	WP 11 → PEQ 17 (b12)
PEQ Daughter Regard for Father	-.01	-.02
PEQ Father Involvement	-.09	-.05
	PEQ 11 → Binge 17 (b21)	Binge 11 → PEQ 17 (b12)
PEQ Daughter Regard for Father	-.05	.01
PEQ Father Involvement	-.06	-.03

Note. PEQ = Parental Environment Questionnaire, Total = Minnesota Eating Behaviors Survey Total Score, BD = Body Dissatisfaction subscale, WP = Weight Preoccupation subscale, Binge = Binge Eating subscale, RMSEA = Root Mean Square Error of Approximation, CFI = Comparative Fit Index. See Figure 17 (p. 98) for a figural representation of paths b21, b12, b43, and b34. *$p < .05$, **$p < .01$.

Appendix P

Post Hoc Analyses of Associations between Maternal Ratings of Mother Involvement and Weight Preoccupation

Exploring Associations at Other Time Points

Additional cross-lagged models were run using maternal ratings of Mother Involvement and Weight Preoccupation scores. These models included data from assessments at age 11 and 17 years only. The structure of these additional models was parallel to that used to analyse cross-lagged associations between father ratings of PEQ scores and MEBS scores. Specifically, cross-lagged models included each variable (i.e., PEQ and MEBS) at age 11 and 17 years only; cross-lagged paths indicated associations between one variable at age 11 years and the other variable at age 17 years; and regression paths indicated the relative temporal stability of each construct across time from age 11 to 17 years (see Figure 17 [p. 98] for the model's structure). Like the other cross-lagged models in this study, the MLR estimator was used to provide maximum likelihood parameter estimates with standard errors that are robust to non-normality and FIML was used to accommodate missing data.

Results indicated that twin differences in Weight Preoccupation at age 11 years predicted twin differences in Mother Involvement at age 17 years (path coefficient = -.20, $p < .001$). However, the cross-lagged path between twin differences in Mother Involvement at age 11 years and twin differences in Weight Preoccupation at age 17 years was not significant. Thus, the unexpected positive cross-lagged association that indicated greater maternal involvement predicted later greater weight preoccupation was specific to assessment of maternal involvement at age 14 years (i.e., not generalizable to maternal involvement at age 11 years).

Controlling for Mother-Child Discrepancy Ratings

As aforementioned, research emphasizes discrepant perceptions across family members regarding the quality of the parent-child relationship (Holtom-Viesel & Allan, 2013), a finding that was corroborated in the current study. Research also suggests that the magnitude of such discrepancies may be associated with eating pathology (Holtom-Viesel & Allan, 2013; Paikoff, Carlton-Ford, & Brooks-Gunn, 1993), as well as other symptoms of psychopathology (e.g., anxiety, depression, and delinquent behaviors; De Los Reyes, Goodman, Kliewer, & Reid-Quinones, 2010; Ohannessian et al., 1995). Recognizing that mothers tended to rate Mother Involvement higher than daughters rated Mother Involvement, it is possible that higher maternal ratings of Mother Involvement reflect a greater discrepancy between mother and daughter perceptions of involvement in the mother-child relationship.

Thus, revised scores for maternal ratings of Mother Involvement were computed that controlled for the size of discrepancy between maternal and daughter ratings of Mother Involvement. Specifically, mother-child discrepancy ratings were created by subtracting maternal ratings of Mother Involvement from each twin's ratings of Mother Involvement at each time point. Subsequently, linear regression analyses were run to regress out the size of mother-child discrepancy ratings for Mother Involvement from maternal ratings of Mother Involvement at all ages. Standardized residuals were then retained as an indicator of maternal ratings of Mother Involvement that were not reflective of the size of discrepancy between maternal and daughter ratings. Revised maternal ratings of Mother Involvement twin difference scores were then calculated using these standardized residuals. These revised maternal ratings of Mother Involvement twin difference scores were then used in cross-lagged models.

Specifically, to ensure that cross-lagged associations between maternal ratings of Mother Involvement and Weight Preoccupation demonstrated in Part 5 were not accounted for by mother-child discrepancies in their perceptions of involvement in the mother-child relationship, cross-lagged models examined in Part 5 were run using these revised maternal ratings of Mother Involvement that controlled for associations with mother-child discrepancy ratings (see Figure 1 [p. 44] for the model's structure). Analyses followed the same procedure outlined in Part 5, with one exception: revised maternal ratings of Mother Involvement that controlled for associations with mother-child discrepancy ratings of Mother Involvement replaced original maternal ratings of Mother Involvement. The MLR estimator was used to provide maximum likelihood parameter estimates with standard errors and a chi-square test statistic that are robust to non-normality; FIML was used to accommodate missing data; and adequate model fit was determined by a non-significant chi-square value, RMSEA value of .05 or less, and CFI values of .90 or greater.

Results indicated adequate model fit ($\chi^2 = 1.31$, $p > .05$, RMSEA = .00, CFI = 1.00). However, twin difference scores in revised maternal ratings of Mother Involvement at age 14 years continued to demonstrate significant cross-lagged associations with twin differences in Weight Preoccupation at age 17 years ($p < .001$). Thus, the unexpected positive cross-lagged association in Part 5 that indicated higher maternal involvement predicted greater weight preoccupation was not accounted for by the magnitude of discrepancy between mother and twin perceptions of Mother Involvement.

Exploring Facets of Involvement

To further explore the unexpected association between higher maternal ratings of Mother Involvement and greater Weight Preoccupation, post hoc analyses explored whether the Involvement scale may include multiple factors related to parent-child involvement. It is possible

that different factors of parent-child involvement would demonstrate differential cross-lagged associations with weight preoccupation. Thus, the Involvement scale was examined to consider whether items could be grouped into theoretically meaningful subgroups. Visual inspection of Involvement scale items suggested two subgroups of items that aligned with aspects of parental involvement emphasized throughout the literature. Specifically, eight of the 12 items on this scale (2, 4, 7, 18, 23, 29, 41, and 46) reflected "connectedness" and four of the 12 items (6, 25, 40, and 50) reflected "awareness" (see Appendix B for all PEQ items). Connectedness was considered to reflect bonding, affective support, and communication within the parent-child relationship. In contrast, awareness was considered to reflect parental monitoring and knowledge of how the child spends her time, including what she does and the extent to which she does or does not succeed in various areas. Connectedness and awareness largely align with two of the three dimensions of parenting – i.e., parental support and behavioral control, respectively – that have been identified as demonstrating important associations with adolescent functioning across culture and time in a large-scale review and analysis of the parenting literature by Barber and colleagues (2005). Unfortunately, psychological control – the third parenting variable identified in Barber and colleagues' research, of which has been suggested to predict increased eating pathology (Berge et al., 2014) – did not appear to be measured by items in the Involvement scale.

Confirmatory factor analysis was run on the Mother Involvement scale using oblique rotation and maximum likelihood extraction that forced a two-factor solution. However, the solution did not support the aforementioned subgroups. Rather, the Involvement scale appeared to reflect one primary factor that was best represented as one unitary scale. When two factors were forced, items often did not clearly load on one factor versus the other, and subgroups of

items that were extracted did not appear to reflect theoretically meaningful factors. Thus, no further analyses were conducted.