

This is to certify that the dissertation entitled

SEPARATION-INDIVIDUATION AND DISORDERED EATING IN ADOLESCENCE

presented by

JANET WENDY EGGERT

has been accepted towards fulfillment of the requirements for the

Doctoral	degree in	Psychology	
Major Professor's Signature			
	4-1-0	7	
	D	ate	

MSU is an affirmative-action, equal-opportunity employer

LIBRARY Michigan State University PLACE IN RETURN BOX to remove this checkout from your record.

TO AVOID FINES return on or before date due.

MAY BE RECALLED with earlier due date if requested.

DATE DUE	DATE DUE	DATE DUE

6/07 p:/CIRC/DateDue.indd-p.1

SEPARATION-INDIVIDUATION AND DISORDERED EATING IN ADOLESCENCE

By

Janet Wendy Eggert

A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

Department of Psychology

2007

ABSTRACT

SEPARATION-INDIVIDUATION AND DISORDERED EATING IN ADOLESCENCE

By

Janet Wendy Eggert

Objective: Eating disorders most commonly begin in adolescence, and theory has speculated that difficulties with the separation-individuation process may contribute to the development of eating pathology during this developmental period. However, studies that have examined this link have used adult rather than adolescent participants, thus there is no empirical support for these theories. In addition, previous studies have not examined whether parent-child relationship problems lead to the separation-individuation difficulties that then lead to disordered eating, nor have studies considered the role of other related factors in the process (i.e., pubertal development, temperament). This study aimed to be the first to examine the extent to which separation-individuation difficulties mediate associations between mother-adolescent relationship difficulties and disordered eating, as well as the potential moderating effects of puberty and temperament on these relationships. Method: Participants included 202 adolescent twins and 101 of their mothers who took part in the Adolescent Twin Study of Behavioral Adjustment and Development from the Michigan State University Twin Registry. Adolescent reports of AN and BN-like disordered eating were collected. Adolescent and maternal reports of the maternal-adolescent relationship, adolescent separation-individuation difficulties, pubertal development, and adolescent temperament, were also obtained. Structural equation modeling was used to examine whether: 1) under-separation mediates relationships between an enmeshed mother-child relationship and AN symptoms; 2) over-

separation mediates relationships between an uninvolved mother-child relationship and BN symptoms; 3) puberty and temperament moderate relationships between variables in the models. Results: Confirming hypotheses, separation-individuation difficulties were found to mediate relationships between difficulties in the mother-adolescent relationship and AN and BN-like disordered eating. In general, difficulties with under-separation explained the relationship between overprotective maternal behavior and AN-like disordered eating, while difficulties with over-separation explained the relationship between characteristics of the mother-adolescent relationship and BN-like disordered eating. Hypotheses regarding the moderating effects of puberty and temperament were partially supported. While puberty moderated relationships between the motheradolescent relationship and separation-individuation difficulties, temperament did not. Discussion: Findings from this study suggest that maternal overprotection and underseparation are associated with increased risk for developing AN, while maternal overprotection, less maternal care, and over-separation are associated with BN. Further, pubertal development appears to influence the development of disordered eating indirectly, by affecting relationships between parenting and separation-individuation. These findings are significant in confirming that characteristics of the mother-adolescent relationship and separation-difficulties are associated with disordered eating during adolescence. Findings from this study highlight the potential benefits of addressing difficulties within the mother-daughter relationship and separation-individuation difficulties in existing prevention and family treatment programs for eating pathology.

ACKNOWLEDGMENTS

This project would not have been possible without the support of many people.

Many thanks to my committee chair, Dr. Kelly Klump, for her guidance and dedication to this project. Her enthusiasm and interest in the work were invaluable and helped make this dissertation possible.

I received equally important assistance from my husband, David, my parents, Hugh and Anne Solomon, and my brother, Jonathan, who provided on-going love and support throughout the dissertation process. Thanks also to my friends for their support and encouragement. Finally, I would like to thank Leo and Lily, who helped make the writing process more fun.

TABLE OF CONTENTS

LIST OF FIGURES	
LIST OF TABLES	viii
KEY FOR THE FIGURES AND TABLES	x
INTRODUCTION	1
Etiology of Eating Disorders	2
Parent-Child Relationships, Separation-Individuation, and Disordered Eating	8
Theories of Separation-Individuation	
Relationships to Eating Disorders	18
Effects of Pubertal Development	29
Puberty and Parent-Child Relationships	30
Puberty and Separation-Individuation	
Effects of Temperament	34
Temperament and Eating Pathology	
Temperament and Separation-Individuation	
Conclusions	37
Study Overview	38
Aims and Hypotheses	41
METHODS	44
Participants	44
Measures	
Statistical Analyses	56
Preliminary Data Analyses	56
Structural Equation Models	
Power Analyses	60
RESULTS	62
Correlations	
SEM Analyses	
Aim 1	
Confirmatory Factor Analyses	
Structural Equation Model	
Aim 2	
Confirmatory Factor Analyses	
Structural Model	
Aim 3	69
AN Model	
BN Model	
Exploratory Analyses: Age as Moderator	
Aim 4	
AN Model	

BN Model	75
Aim 5	75
DISCUSSION	76
Relationships among the Mother-Child Relationship, Separation-Individuatio	
and Disordered Eating	-
AN-Like Disordered Eating	
BN-Like Disordered Eating	
Moderators	
Effects of Puberty, Age, and Temperament	
Limitations and Future Directions	
Conclusions and Treatment Implications	
FIGURES	. 100
TABLES	. 116
APPENDICES	. 135
Appendix A: Examining age as a moderator in the AN and BN models	
REFERENCES	. 140
TABLES IN APPENDICES	
Table A1. Goodness of fit indices for multiple-group analyses of AN models examinage as a moderator	
Table A2. Goodness of fit indices for multiple-group analyses of BN models examining age as a moderator	ing . 138
Table A3. Unstandardized and standardized coefficients for BN – Reduced Model an	_

LIST OF FIGURES

Figure 1. Conceptual and structural model of relationships between the mother-child relationship, separation-individuation, and AN-like disordered eating
Figure 2. Latent Variable Structural Equation Model of AN – Original Model (includes all paths and variables)
Figure 3. Latent Variable Structural Equation Model of AN – Model with Correlated Errors (i.e., e1 and e2)
Figure 4. Latent Variable Structural Equation Model of AN- Reduced Model (without aenmesh (Enmeshment Seeking) and nonsignificant paths between mother-child relationship and separation-individuation variables)
Figure 5. AN-Reduced Model with direct effects between adolescent reported maternal protection and AN-like disordered eating
Figure 6. AN-Reduced Model with direct effects between maternal reported maternal protection and AN-like disordered eating
Figure 7. Conceptual and structural model of relationships between the mother-child, relationship, separation-individuation, and BN-like disordered eating
Figure 8. Structural model of BN – Original Model (includes all paths and variables). 108
Figure 9. Structural model of BN – Model with Correlated Errors (i.e., e1 and e2) 109
Figure 10. Structural model of BN – Reduced Model (without nonsignificant paths between mother-child relationship and separation-individuation variables)
Figure 11. BN – Reduced Model with direct effects between the mother-child relationship variables and binge eating
Figure 12. AN-Reduced Model with puberty moderating relationships between the mother-child relationship and separation-individuation variables
Figure 13. BN – Reduced Model with puberty moderating relationships between the mother-child relationship and separation-individuation variables (correlation values for exogenous variables not shown)
Figure 14. AN – Reduced Model with AN temperament moderating relationships among factor loadings of AN-like disordered eating
Figure 15. Latent Variable Structural Equation Model of AN- Reduced Model with Negative Affect added as a direct effect

LIST OF TABLES

Table 1. Internal consistency reliability estimates for subscales used in analyses 117
Table 2. Pearson product correlations between PBI, SITA, PASAS, MEBS, and EDEQ scores
Table 3. Goodness of fit indices for single-group analyses of models with AN-like disordered eating
Table 4. Unstandardized coefficients for AN - Reduced Model
Table 5. Goodness of fit indices for single-group analyses of BN-like disordered eating
Table 6. Unstandardized coefficients for BN – Reduced Model 122
Table 7. Goodness of fit indices for multiple-group analyses of AN models examining puberty as a moderator
Table 8. Unstandardized coefficients for AN – Model with Correlated Errors among puberty groups
Table 9. Goodness of fit indices for multiple-group analyses of BN models examining puberty as a moderator
Table 10. Unstandardized coefficients for BN – Model with Correlated Errors among puberty groups
Table 11. Cross-tabulation table examining composition of age and puberty groups 127
Table 12. Goodness of fit indices for multiple-group analyses of AN – Reduced Model examining temperament as a moderator
Table 13. Unstandardized coefficients for AN - Reduced Model among summed AN Temperament groups
Table 14. Goodness of fit indices for single-group analyses of AN – Reduced Model examining direct effects of temperament
Table 15. Unstandardized and standardized coefficients for AN - Reduced Model with direct effects of temperament variables
Table 16. Goodness of fit indices for multiple-group analyses of BN – Reduced Model examining temperament as a moderator

Table 17. Goodness of fit indices for multiple-group analyses of BN models of	examining
direct effects of temperament	133
Table 18. Unstandardized and standardized coefficients for BN – Reduced M	odel with
Inhibitory Control and Shyness as direct effects	134
initially control and sury mess as an enter officers in the sury message in the sury m	,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,,

KEY FOR THE FIGURES AND TABLES

Disordered Eating

MEBS

Weight Preoccupation – Adolescent Report wp
Body Dissatisfaction – Adolescent Report bd
Binge Eating – Adolescent Report be

EDEQ

Dietary Restraint – Adolescent Report dr

Separation-Individuation

SITA

Enmeshment-Seeking – Adolescent Report aenmesh
Engulfment-Seeking – Adolescent Report aengulf
Separation Anxiety – Adolescent Report asepanx
Dependency Denial – Adolescent Report adepden
Healthy Separation – Adolescent Report ahealthsep

PASAS

Anxiety about Adolescent Distancing -

Mother Report maaad

Mother-Child Relationship

PBI Care

Care – Adolescent Report acare
Care – Mother Report mcare

Protection

Protection – Adolescent Report aprotect
Protection – Mother Report mprotect

Temperament

EATO-R

Negative Affect

Negative Affect – Adolescent Report anegaffect
Negative Affect – Maternal Report mnegaffect

Shyness

Shyness – Adolescent Report ashy Shyness – Maternal Report mshy

Inhibitory Control

Inhibitory Control – Adolescent Report ainhib
Inhibitory Control – Mother Report minhib

INTRODUCTION

Eating disorders, such as anorexia nervosa (AN) and bulimia nervosa (BN), are significant public health problems that affect over 5 million women and girls every year (NIMH, 1994). Symptoms of AN include an intense fear of gaining weight, a refusal to maintain a minimally normal body weight (i.e., 85% of expected weight for height and age), a disturbance of body image, and the absence of at least three consecutive menstrual cycles (APA, 1994). There are two sub-types of AN: the restricting type, in which the individual does not regularly engage in binge-eating or purging behavior, and the bingeeating/purging type, in which the individual regularly engages in binge-eating and purging behavior. BN is characterized by a disturbance of body image and recurrent episodes of binge eating and compensatory behaviors, such as self-induced vomiting or the abuse of laxatives (APA, 1994). Similarities and differences between AN bingeeating/purging type and BN in terms of their diagnostic profiles remain unclear, but studies suggest that individuals with AN binge-eating/purging type may show characteristics that are more similar to individuals with BN than individuals with AN restricting type in areas such as personality (Vervaet, Heeringen, & Audenaert, 2004).

Eating disorders can have serious medical and psychiatric consequences due to the severity and physical symptoms of the disorders and the high relapse rate (Herzog et al., 1999). Furthermore, eating disorders often have a protracted course (Keel et al., 1999; Strober, Freeman, & Morrell, 1997) and AN has the highest mortality rate of any psychiatric diagnosis (Sullivan, 1995). AN and BN afflict approximately 8-12/100,000 individuals per year and .5-3% of females in the United States (APA, 1994) while as many as 55% of young adolescent females feel that they look fat and 43% of young

adolescent females engage in dieting behaviors (Childress, Brewerton, Hodges, & Jarrell, 1993). Understanding the etiology of eating pathology can help inform treatment and prevention efforts that may reduce the severe consequences of the disorders.

Etiology of Eating Disorders

A substantial amount of research has investigated the etiology of eating disorders, yet the etiology remains unknown. However, there are three categories of risk factors that have emerged from this research: biological, psychological, and social. Some significant biological factors include genes, neurotransmitters, and pubertal development, while psychological factors include temperament and restrained eating, and social factors include societal influences and family relationships. Among the factors described within these categories, several key factors have been identified. Specifically, temperament, pubertal development, and family relationships have been found to be important etiological factors for the development of eating pathology.

Biological Factors

Recent behavioral genetic research suggests that eating disorders are significantly heritable (i.e., greater than 50%), while nonshared (rather than shared) environment accounts for the remaining variance (Bulik, Sullivan, & Kendler, 1998; Bulik, Sullivan, Wade, & Kendler, 2000; Klump, Miller, Keel, McGue, & Iacono, 2001; Kortegaard, Jorgensen, Gillberg, & Kyvik, 2001; Wade, Bulik, Neale, & Kendler, 2000). Research also provides evidence for familial transmission of full and subclinical eating disorders in addition to shared transmission between eating and anxiety disorders, suggesting a common genetic liability (Strober et al., 2000; Walters & Kendler, 1995).

Certain neurotransmitters have been associated with eating pathology and are considered to be another biological influence on eating pathology (Kaye, 1997).

Research has focused on the role of serotonin dysfunction in increasing the risk for developing disordered eating due to its association with appetite, however results from studies have been mixed. There is evidence for decreased serotonin function among individuals with eating disorders (Jimerson, Lesem, Kaye, & Brewerton, 1992; Kaye, Gwirtsman, George, Jimerson, & Ebert, 1988). However, serotonin functioning appears to normalize after nutritional rehabilitation, which suggests that the decrease in serotonin may be a result rather than a cause of eating pathology (Kaye et al., 1988). Research has not examined premorbid serotonin function however, research has found increased cerebral spine fluid concentrations of serotonin after recovery from AN (Kaye, 1997; Kaye, Gwirtsman, George, & Ebert, 1991), and thus, there is speculation that increased serotonin may be a trait disturbance that contributes to the development of eating pathology.

Finally, the development of eating disorders has been associated with developmental transitions, such as adolescence (Keel, Leon, & Fulkerson, 2001). Puberty has been studied as a potential etiological factor and research has consistently shown that disordered eating symptoms increase significantly with the onset of puberty (Dorn, Crokett, & Petersen, 1988). More advanced pubertal development is associated with more disordered eating (Dorn, Crokett, & Petersen, 1988). Puberty could influence the development of disordered eating directly through associated physical changes (i.e., increased body fat in girls), increases in ovarian hormones (Klump et al., 2003; Klump et al., 2006) and also indirectly through its effect on other relevant variables (e.g., negative

body image; Koff & Reirdan, 1993). Despite interest in this area and the extent to which it is discussed in the field, there is not a great deal of research examining the role of puberty in the development of disordered eating, and factors associated with this developmental stage. This suggests that there is a need for further research in this area.

Psychological Factors

Temperament has been defined as consisting of broad, stable characteristics which influence the behavior of individuals, while personality describes these stable characteristics as they are shaped by the environment (Rothbart & Bates, 1998). Research shows a strong correlation between temperament and personality (Derryberry & Rothbart, 1988; Rothbart, Ahadi, & Evans, 2000). Much of the research in eating disorders has examined relationships between eating pathology and personality, rather than temperament, because it has been conducted with adult participants (Bulik, Sullivan, Fear, & Pickering, 2000; Casper, Hedeker, & McClough, 1992; Fassino et al., 2002; Kleifield, Sunday, Hurt, & Halmi, 1994; Kleifield, Sunday, Hurt, & Halmi, 1994b; Klump et al., 2000; Pryor & Wiederman, 1996; Tomotake & Ohmori, 2002). Therefore, findings pertaining to both personality and temperament and their association with eating pathology will be discussed. Specifically, negative affectivity in general, and high stress reactivity in particular, have been identified as predictors of the development of disordered eating (Bulik, Sullivan, Weltzin, & Kaye, 1995; Leon et al., 1999; Lillenfeld et al., 2000; Pryor & Wiederman, 1996). Further, perfectionism, neuroticism, and harm avoidance (i.e., the tendency to inhibit behavior to prevent punishment) have been associated with AN and BN (Brookings & Wilson, 1994; Ghaderi & Scott, 2000; Heaven et al., 2001; Lillenfeld et al., 2000; Podar, Hannus, & Allik, 1999; Pryor & Wiederman,

1996). Temperament and personality have been found to be robust predictors of eating pathology across various longitudinal studies, and have been found to be more predictive of eating pathology than other etiological variables studied simultaneously (e.g., other forms of psychopathology) (Bloks, Hoek, Callewaert, & van Furth, 2004; Keel, Fulkerson, & Leon, 1997; Leon, Fulkerson, Perry, & Early-Zald, 1995; Stice, 2002; Stice & Agras, 1998; Wonderlich, Connolly, & Stice, 2004).

Researchers have also identified other traits and behaviors that are associated with the development of eating pathology. Individuals who restrain their eating through chronic dieting may lose the ability to label hunger and satiation (Polivy & Herman, 1985) and subsequently develop disordered eating symptoms. Research shows that individuals with eating disorders are eight times more likely to have a history of dieting than individuals without eating disorders (Polivy & Herman, 1985). AN may develop following chronic dieting and BN may develop because individuals cannot sustain the restrained eating, which leads to binging (Polivy & Herman, 1985). In addition, eating pathology has been associated with depression and anxiety (Johnson, Cohen, Kasen, & Brook, 2003). Studies show that depressive and anxiety disorders during adolescence are associated with elevated risk for the development of disordered eating (Johnson, Cohen, Kasen, & Brook, 2003; Leon et al., 1999).

Social Factors

Because eating disorders are more prevalent among females and in industrialized nations (Hsu, 1990), the role of societal factors in the etiology of eating disorders has been examined. Studies show that the ideal body size has become thinner in our society over time (Garner, Garfinkel, Schwartz, & Thompson, 1980; Wiseman, Gray, Mosimann,

& Ahrens, 1992) and this may increase the discrepancy between ideal body size and actual body size, which has been linked to more body dissatisfaction (Tiggeman & Pickering, 1996). However, it does not appear that eating disorders are affected by culture in a uniform manner. Keel and Klump (2003) conducted a meta-analysis in which they found that BN is more influenced by culture than AN. They explain that one influencing factor may be that binge eating requires an abundance of food, and can only take place in geographical areas where food is abundant, while starvation can take place in any context (Keel & Klump, 2003). In addition, purging to prevent weight gain is more likely to occur in cultures that value thinness. Therefore, the combination of binging and purging is more likely to occur in specific cultural contexts, while restrictingtype AN can exist in numerous contexts. The implication of these findings is that the genetic predisposition to BN is likely expressed differently depending upon the cultural context (e.g., may be expressed as a somatoform disorder in a different cultural context) while the genes related to AN seem to produce AN regardless of the cultural context (Keel & Klump, 2003).

In addition to societal influences, parent-child relationships and overall family environment have also been identified as important etiological factors in eating disorders (Attie & Brooks-Gunn, 1989; Wertheim et al., 1992). Although there has been less research done in this area, family factors are some of the most widely discussed etiological factors in the field (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). The research that has been done indicates that specific types of family environments are associated with AN and BN individuals (e.g., more enmeshed for AN individuals and more chaotic for BN individuals) (Goldstein, 1981; Humphrey, 1989; Kog &

Vandereycken, 1989). Nonetheless, the dearth of research in this area limits the ability to make conclusions about the role of family factors in the development of eating pathology.

Summary

Overall, there is evidence for various biological, psychological, and social factors in the etiology of eating disorders. However, many studies have examined these factors in isolation rather than in a more integrated manner. This omission makes it difficult to determine relationships among the etiological factors and how they contribute to the development of eating pathology. In addition, family factors have not been examined in any depth, despite considerable theory describing relationships between family factors and eating pathology. Finally, it is important to examine these etiological factors during periods of heightened risk, such as developmental transitions, because risk factors may have unique influences during transitional developmental periods (Klump, McGue, & Iacono, 2000). Adolescence is a particularly important developmental period, as it is the time when eating disorders most commonly begin (APA, 1994). There are many theories about the relationship between this period of development and the onset of eating disorders (e.g., changes associated with puberty, difficulties with separationindividuation) (Koff & Rierdan, 1993; Rhodes & Kroger, 1992), but very little empirical research has investigated these theories.

The current study will examine one of these theories, which postulates that difficulties in the parent-child relationship and separation-individuation difficulties during adolescence are influential in the onset of eating disorders during this developmental period. In addition, the influence of puberty and temperament as moderators of these relationships will be examined. Puberty and temperament have been

selected as additional variables for analysis for several reasons. Across studies, temperament has emerged as one of the most robust predictors of eating pathology.

Thus, it is important to examine how temperament may influence relationships between parent-child relationships and separation-individuation in predicting disordered eating.

Further, puberty has been discussed in relation to eating pathology, however few studies have examined how pubertal development may influence other processes that are prominent during early adolescence, including parent-child conflict and separation-individual difficulties. Thus, examining how these established risk factors may influence relationships between family dynamics and eating pathology will result in a more complete model of disordered eating during adolescence.

Parent-Child Relationships, Separation-Individuation, and Disordered Eating

Theories of Separation-Individuation

The process of separation-individuation is relevant to the parent-child relationship and disordered eating and occurs during the adolescent developmental period.

Separation-individuation describes the normal developmental sequence of achieving a sense of being a separate individual entity (Edward, Ruskin, & Turrini, 1981). There are many theories that examine the process of separation-individuation and its importance in adolescent development and health (including eating disorders). Central theories of separation-individuation include developmental theories (e.g., individuation theory, Youniss & Smollar (1985); sociobiological theory, Steinberg (1990); cognitive-developmental theory, Smetana (1991)), traditional lifespan theories (e.g., Erikson (1968)), feminist theories (e.g., Gilligan (1982)), and Mahler's comprehensive theory of

separation-individuation (1975). These theories will be described and the extent to which the theories can be used to explain the development of eating pathology during adolescence will be discussed.

Developmental, Lifespan, and Feminist Theories

Developmental Theories.

There are many developmental theorists who describe the individuation process. Most agree that adolescents increase their striving for autonomy during adolescence, which leads to some conflict in the family and ultimately to individuation (Pinquart & Silbereisen, 2002). Individuation theory, as delineated by Youniss and Smollar (1985), suggests that both autonomy and connectedness are important in accomplishing individuation during adolescence. Too much closeness or too little closeness with family members can inhibit the individuation process; accomplishing individuation in the family context involves a balance between connectedness and individuality (Youniss & Smollar, 1985). Steinberg's sociobiological theory focuses on the major transformations that occur in the parent-child relationship during adolescence. There is a period of disequilibrium in the parent-child relationship during adolescence, triggered by biological and cognitive changes for the adolescent, leading to a redefinition of the parent-child relationship into one that is more cooperative and reciprocal (Steinberg, 1995).

Finally, Smetana's cognitive-developmental perspective suggests that adolescents individuate in response to the development of advanced levels of social reasoning (Smetana, 1991). Adolescents begin to view issues that they once considered to be social conventions as a matter of personal choice, which causes conflict with parents. In addition, Smetana (1991) explains that adolescents view social conventions as arbitrary

through this period of their cognitive development and this also leads to more conflict with parents. However, this process of disagreement contributes to the development of individuation for the individual (Smetana, 1991). Across these theories, there is consensus that adolescence is a period of disequilibrium characterized by some conflict with parents. It is possible that this process could lead to some distress for the adolescent, which could manifest in eating pathology.

Lifespan Theories.

Traditional lifespan theories, including Blos (1965), Baltes (1999), and Erikson (1968), consider individuation and the establishment of psychological autonomy to be fundamental tasks of adolescence that must be addressed and resolved. For example, according to Erikson's lifespan theory (Erikson, 1968), becoming autonomous is a major life task during toddlerhood. Erikson (1968) suggests that between the ages of 18 months and 3 ½ years, the child begins to develop motor and cognitive abilities that allow them to experience themselves as separate entities from their parents. The battle for autonomy with parents can be shown in stubborn refusal and temper tantrums, as children assert their newfound autonomy. If the child resolves this stage in a healthy way, he/she develops self-certainty, autonomy, and the will to be him/herself. However, if parents have an inability to cope with the budding autonomy of their child and impose too many restrictions or unfair punishment, the child may not be able to fully develop his/her autonomy as a separate entity, which reflects a lack of autonomy resolution. Attributes associated with a lack of autonomy resolution for the child include experiencing shame, self-doubt, dependency, self-consciousness, and meek compliance (Erikson, 1968).

Further, autonomy is also postulated to be important during adolescence when the major task involves identity development, which cannot be accomplished without autonomy from parents. The identity crisis in adolescence is thought to revive earlier unresolved autonomy issues as the adolescent struggles for independence from the family; this experience is proposed to function as a recapitulation of earlier attempts at autonomy. According to Erikson (1968), problems in adolescence, including eating disorders, can be viewed as reflections of difficulties with conflicts from earlier stages (e.g., the toddlerhood stage, where the development of autonomy is the major task). Similar to the developmental theories, Erikson's theory and other lifespan theories (e.g., Blos, 1965) are helpful in understanding this process, but are not clear in their explanation of why disordered eating symptoms might develop (rather than other forms of psychopathology) in response to difficulties with separation-individuation during adolescence.

Feminist Theories.

Feminist theorists tend to focus more on difficulties specific to adolescent females that may contribute to eating pathology rather than on the influence of the parent-child relationship. These theories attempt to explain why girls may have an especially difficult time during this developmental phase (Brown & Gilligan, 1993; Gilligan, 1982; Jordan, 1993; Steiner-Adair, 1990; Taylor, Gilligan, & Sullivan, 1995). The theories suggest that girls are socialized throughout their lives to value intimacy and close relationships, while boys are not socialized in this way. The female personality is thought to develop through attachment to others, while the male personality develops in a more independent manner (Chodorow, 1978).

Difficulties then arise during adolescence when girls learn that society values individualism and independence rather than relationships. While these values fit in with how boys have been socialized, they do not fit with socialization patterns for girls. A relational impasse (i.e., desiring authentic relationships but fearing that honest expression of feelings and thoughts will jeopardize their relationships) may subsequently result for adolescent girls, and they may respond to this by compromising their "true selves" in relationships, resulting in inauthentic relationships (i.e., not bringing the most central aspects of themselves to their relationships; Gilligan, 1982) and a need to deal with the adolescent crisis through problematic behaviors, including the development of eating problems and depression (Gilligan, 1982; Steiner-Adair, 1990).

Although these feminist theories help describe potential reasons for the higher prevalence of certain types of psychopathology (e.g., eating disorders, depression) during adolescence, they do not adequately account for individual differences in eating disorder development. According to these theories, all girls should have eating problems since all are socialized the same way. However, because not all girls develop eating pathology, there must be individual differences that account for some girls developing eating disorders while others do not.

Summary.

Overall, these theories offer different perspectives and ways of understanding the process of individuation during adolescence. Some of them can be used to understand the relationship between separation-individuation and eating pathology, although many of them do not explicitly make this connection. For example, developmental and traditional lifespan theories suggest that eating pathology may develop during adolescence in

response to the difficulties experienced with the task of individuation, but they do not specify why eating pathology would develop versus other forms of psychopathology. Feminist theories take this further, discussing how societal influences affecting females lead to confusion in the development of autonomy during adolescence, which can explain the higher prevalence of some disorders (including eating pathology) in females relative to males. However, it is unclear why some girls develop eating pathology and others do not; the feminist theories do not account for the individual differences in eating disorders that are observed.

Overall, the aforementioned theories do not provide a detailed description of the separation-individuation process, which could be used to more comprehensively understand how eating problems could develop due to unresolved separation-individuation issues. Further, these theories do not provide a strong theoretical rationale for why eating pathology would develop instead of other disorders, and why eating disorders occur in some girls but not all girls during adolescence.

Mahler's Theory

Mahler's theory of separation-individuation, in addition to theorists who have expanded on Mahler's work, addresses the limitations described above and provides a theoretical explanation that can be used to understand the relationship between separation-individuation difficulties and eating pathology. This may explain why Mahler's theory has been the overwhelmingly predominant theory used in the theoretical and empirical literature to explain separation-individuation difficulties in eating disorders. Although Mahler's theory does not explain the greater prevalence of eating disorders in females versus males, it identifies processes that may manifest with

disordered eating among females who also have other risk factors for eating pathology that males do not have (e.g., societal influences). Specifically, cultural contexts that idealize thin body size for women may serve as a risk factor for the development of eating pathology among females (Tiggeman & Pickering, 1996), while this has not been identified as a risk factor for males. The process Mahler describes may manifest with other symptoms (e.g., depression, anxiety) among males with different risk factors.

Therefore, risk factors other than those examined in this study may be responsible for the greater prevalence of eating disorders among females versus males (e.g., societal influences). This possible explanation for the greater prevalence of eating disorders in females is not described in Mahler's theory, however it is an extension of her comprehensive theory of separation-individuation related to the development of eating pathology.

Mahler's theory is based on psychodynamic principles, which suggest that interactions with parents throughout a child's life contribute to the child's ability to successfully individuate. Margaret Mahler (1975) described the process of separation-individuation as something that infants experience during their first three years of life with respect to their parents, especially the mother. Within this theory, separation refers to the child's movement from fusion with the mother toward a sense of being separate from the mother (Mahler, 1975). By contrast, individuation consists of those steps that lead to the development of an individual's own personal and unique characteristics and the sense of self (Edward, Ruskin, & Turrini, 1981). According to Mahler (1975), there is an innate drive toward individuation, but this will only be accomplished when the infant can achieve some sense of autonomy and separation from the mother.

There are four sub-phases that the infant is hypothesized to move through during this process in which specific developmental achievements take place. The sub-phases include differentiation (i.e., infant develops a curiosity of the external world outside of the mother), practicing (i.e., infant physically leaves the mother by crawling or climbing but develops anxiety when separated), rapprochement (i.e., child enjoys social interactions and can appear independent but if separated too long, becomes helpless), and consolidation and object constancy (i.e., increased comfort with mother's absence, an internalized image of the mother, and viewing the mother and self as separate beings in a positive manner) (Mahler, 1975). If a child successfully navigates these four phases, he/she develops the ability to understand him/herself as an entity that is unique and separate from significant others.

According to Mahler's theory, there is individual variation in the resolution of each stage in the separation-individuation process. A critical aspect of this theory is that the mother's parenting behavior influences the infant's progression through the separation-individuation stages (Mahler, 1975). For example, infants whose mothers behave in an overprotective manner may experience increased separation anxiety (i.e., strong fears of losing emotional or physical contact with an important other) and more vulnerability to object loss during the differentiation sub-phase. The lack of resolution of the differentiation sub-phase can then lead to conflict as the infant ages, which could manifest itself through symptoms such as separation anxiety and clinginess towards parental figures (Edward et al., 1981). Alternatively, an infant whose mother is uninvolved and unresponsive could experience premature differentiation from his/her mother and may become tentative and clingy. The clinginess, especially during the

rapprochement phase, may serve to increase the mother's rejection of the child and this infant could appear precocious and overly independent as he/she gets older (Edward et al., 1981).

Although Mahler's theory focused on the separation-individuation process during infancy, she noted that transitions later in life could re-activate problems with individuation from infancy (Mahler, 1975). Many theorists have expanded on Mahler's theory by extending it to adolescence, suggesting that other critical developmental periods also contribute to this developmental milestone. Many theorists consider adolescence to be a second separation-individuation stage (Blos, 1965, 1967; Brandt, 1977; Esman, 1980; Josselson, 1980; Kroger, 1989). During adolescence, it is hypothesized that the task is to separate from the internalized parent, instead of the physical parent in reality, which was the task in infancy. The move is made away from the family unit into the outside world, and the challenge of separation-individuation during adolescence is to become autonomous from parents while still maintaining closeness (Edward et al., 1981). Interestingly, it appears that adolescents who struggle with separation-individuation in relation to their parents have difficulties in all relationships, including a general difficulty with being close to others and anxiety or fear of being taken over by the other person (i.e., fear of engulfment) (Huprich, Stepp, Graham, & Johnson, 2004). Therefore, as children get older, their anxiety with respect to relationships with parents extends to relationships with other significant people in their lives. This issue is especially salient during adolescence, when there is a focus on relationships with peers and distancing from parents.

The separation-individuation process may be particularly vulnerable to difficulties during adolescence, given the physical and societal pressures (i.e., pressure to be more independent and adult-like, with more focus on peers rather than parents) that are present during this period. Further, adolescents who did not progress through separation-individuation during infancy successfully may experience re-activation of earlier conflicts that were unresolved during infancy (Mahler, 1975). This re-activation is compounded by the fact that the parental behavior that prohibited successful separation-individuation in infancy (i.e., overprotectiveness, unresponsiveness, etc.) likely continues to be problematic during adolescence and contributes to difficulty with separation-individuation during this critical developmental period.

Several problem behaviors have been theorized to develop as a result of these parental behaviors and subsequent separation-individuation difficulties during adolescence. For example, increased anxiety and acting out (Edward et al., 1981) during adolescence has been linked to difficulties with the separation-individuation process. In addition, eating disorders have been theorized to result from parental behavior and separation difficulties during adolescence, with overprotective parenting resulting in AN and unresponsive parenting resulting in BN (Bruch, 1974; Johnson, 1991; Marsden, 1997).

Importantly, this study did not directly examine the early parent-infant relationship patterns hypothesized by Mahler to be important. Similar to other investigators (Friedlander & Siegel, 1990; Marsden, Meyer, Fuller, & Waller, 2002; Rhodes & Kroger, 1992; Smolak & Levine, 1993), her theory was used to increase understanding of the origins of separation-individuation difficulties that may be

encountered during adolescence by suggesting how these early patterns could manifest in adolescence.

Relationships to Eating Disorders

Theories

One aspect of the mother-infant relationship that is identified by Mahler (1975) to be an important component of the separation-individuation process is interactions around feeding. Further, many theorists believe that disturbances in mother-infant interactions around infant feeding are present and contribute to difficulties in separation-individuation that manifest themselves as eating pathology during adolescence (Bruch, 1974; Chassler, 1997; Marsden, 1997; Rhodes & Kroger, 1992). These theories will be discussed below to provide important theoretical background for the current study. Many theories have focused on the relationship between AN, parental behavior, and separation-individuation, but the relationship between BN and separation-individuation has been less comprehensively and clearly delineated. Although some aspects of the processes in the two syndromes are similar, there are important differences that warrant separate treatment of these theories.

Anorexia nervosa.

Theories linking AN, parent-child relationships, and separation-individuation have focused entirely on the restricting subtype of this disorder rather than the binge-purge type (Bruch, 1974; Garfinkle et al., 1983; Goldstein, 1981; Palazzoli, 1974). This is likely because the restricting and binge-purge subtypes of AN were not introduced into the psychiatric nomenclature until the DSM-IV (APA, 2000), and most theories about

AN, parent-child relationships, and separation-individuation predate this version of the DSM. Before this subtyping, the only type of AN that was thought to exist was the restricting subtype.

Theories of AN (Bruch, 1974; Garfinkle et al., 1983; Goldstein, 1981; Palazzoli, 1974) have postulated that the mother of an AN infant may not respond sensitively to cues from the infant regarding hunger and instead feeds the infant excessively when the mother feels it is necessary, not necessarily when the infant is hungry. Specifically, the mother is hypothesized to behave in an overly attentive and protective manner towards her infant by feeding the infant in response to all cues (e.g., when the infant is crying) or without any cues at all. This maternal feeding behavior suggests that the mother is overly concerned about her infant such that she insists on responding to her infant in the absence of cues that the infant is hungry or needs her. These feeding behaviors reflect a misunderstanding of the infant's needs. Subsequently, the infant is forced to respond to the mother's decisions about eating, therefore eating when her mother wants her to eat instead of when she feels hungry (Bruch, 1974). According to this theory, these experiences do not allow the infant to develop a sense of herself as separate from her mother because the infant is not differentiated in identifying that her bodily experiences come from within herself rather than determined by the external world. Alternatively, when an infant receives appropriate and regular responses to her hunger needs, the infant will learn to discriminate the self from the mother and develop a sense of body identity (Bruch, 1974). Without these sensitive maternal responses to eating needs, there is interference in the separation-individuation process (Bruch, 1974) and the infant can have difficulty feeling like a separate entity from the mother.

More current theorists, in an extension of Mahler's comprehensive description of the separation-individuation process during infancy in conjunction with a focus on mother-infant feeding patterns, suggest that these separation difficulties experienced during infancy around feeding become re-activated during adolescence when there is pressure for the adolescent to become more independent and to separate from her family (Johnson, 1991; Marsden, 1997). In addition to this increased pressure to separate, the theories postulate that the mother continues to exhibit overprotective parental behavior during adolescence that further complicates the child's underseparation that was present in infancy and continues into adolescence (Johnson, 1991; Marsden, 1997). The combination of overprotective parenting, underseparation from the mother, and new pressures for individuation, are all hypothesized to lead to renewed difficulties with the separation-individuation process.

One way that these difficulties are theorized to become manifested is through overcontrolling food intake. The restriction of food is hypothesized to prevent the adolescent from maturing psychosexually into an adult and allows her to avoid separation from her parents and the development of an autonomous self (O'Kearney, 1996).

Restricting AN symptoms are therefore hypothesized to help girls with AN to remain childlike and thus part of the enmeshed family system. In this theory, the development of disordered eating symptoms can be understood as a retreat from the challenge of the adolescent stage of separation-individuation, by returning to an earlier stage of physical development (Marsden, 1997).

Bulimia nervosa.

The influence of disturbed parent-child relationships and the process of separation-individuation have also been used to explain the development of BN (Marsden, 1997). The mother-infant relationship is hypothesized to be affected in a different way in BN than in AN families. Mothers of BN individuals have been theorized to be more erratic and less dependable in their feeding of their infants than other mothers (Johnson, 1991). For example, they have been theorized to be unresponsive to their infant's cues of hunger, feeding their infant on an unpredictable schedule and not responding when their infants indicate that they are hungry (Johnson, 1991). This inconsistent and uninvolved parenting style is theorized to lead to infants having to care for themselves and becoming conflicted about relying on others for care. This conflict and premature independence are thought to then lead to an overseparation from the mother during infancy (Johnson, 1991).

During adolescence, when separation-individuation demands intensify and parental behavior is postulated to continue to be erratic and distant, BN individuals are theorized to intensify their overseparation from their families (Johnson, 1991). However, they are thought to express this intensification in a different way than AN individuals – namely, by binging and purging rather than restricting their food intake. One theory that has been proposed to explain this link postulates that an individual with BN takes in and expels food at will as a form of control (Marsden, 1997). This is control that she does not have over the people that she needs for support, such as her parents who are only inconsistently available to her (Marsden, 1997). It is theorized that the BN individual comes to rely only upon herself and decides what to eat and purge, taking over what she could not rely on her mother to provide for her during infancy. Individuals who develop

BN are thought to desire and strive for continued independence and overseparation from a family that has been inconsistent and emotionally unsupportive (Smolak & Levine, 1993).

Summary.

In summary, eating disorders are theorized to result from conflicts around parent-child relationships that contribute to disturbances in infant separation-individuation that reemerge during adolescence. Specifically, AN symptoms are hypothesized to emerge to prevent adolescents from becoming more physically mature and thus, to avoid separation from overly involved parents. AN individuals are hypothesized to act out their conflict around separation-individuation by renewing dependence on parents by refusing food and returning to a pre-pubertal state (Marsden et al., 2002). By contrast, BN symptoms, although less extensively discussed in the literature, are thought to emerge as a way for adolescent girls with inconsistent and underinvolved parents to feel in control of some part of their lives, by controlling what food they eat and expel.

Empirical Research

Despite these theories, few studies have examined all aspects of theorized relationships, particularly those linking parenting behaviors to the separation-individuation difficulties that girls with eating disorders are postulated to experience.

There has been extensive research exploring the parent-child relationship in association with eating disorders, which has identified specific types of parent-child relationships that are evident in AN and BN families (Goldstein, 1981; Humphrey, 1989; Kog & Vandereycken, 1989). However, less research has examined the relationship between separation-individuation and eating disorders, and no studies have examined the ways in

which parent-child relationships influence the separation-individuation process and increase risk for eating pathology. Because no studies have integrated these concepts, findings pertaining to parent-child relationships and separation-individuation will be discussed separately to inform the current study's integrative model of these relationships.

Relationships with parents.

AN: Empirical studies have found that parents in families with an AN child tend to be more controlling, overprotective, and enmeshed (Bruch, 1974; Foulkes, 1996; Garfinkle et al., 1983; Goldstein, 1981; Horesh et al., 1996; Palazzoli, 1974). In particular, it appears that mothers of individuals with AN are more controlling and overprotective than mothers of individuals with no eating pathology (Johnson, 1991; Pike & Rodin, 1991; Rhodes & Kroger, 1992; Walters & Kendler, 1995). For example, Calam and colleagues (1990) examined parent-child relationships among 31 women with ANrestricting type and an age-matched group of university students with no eating pathology. The participants completed a set of questionnaires, including the Parental Bonding Instrument (PBI; Parker, Tupling, & Brown, 1979). The PBI assesses the parent-child relationship in the first 16 years of an individual's life and has been used in many studies of eating disorders (Bulik, Wade, & Kendler, 2001; Rhodes & Kroger, 1992). It yields two scales: the Care scale, which measures warmth, empathy, and emotional support, and the Protection scale, which measures intrusion, control, and overprotection. Results indicated that the AN group reported significantly higher parental protection on the PBI than the control group. These findings lend support to

theoretical accounts of the parent-child relationship in AN, which have suggested the presence of an overprotective family system.

One significant limitation of this study and others is that the parent-child relationship is typically assessed using self-report measures completed by a single informant, rather than obtaining multiple perspectives. Studies show that there are often discrepancies between the perspectives of parents and children about the parent-child relationship (Elkins, McGue, & Iacono, 1997), thus it is difficult to determine the nature of the parent-child relationship without examining both parent and child perceptions. In addition, studies have generally not examined differences in the parent-child relationship among various types of eating disorders that could help further support theory which suggests that different types of eating disorders are associated with unique patterns of parent-child relationships. Finally, studies have tended to focus on adult participants, who reported on the parent-child relationship retrospectively instead of during key times when the child was living with the parents. This could lead to retrospective recall biases; specifically, adults with disordered eating problems reflecting back on the parent-child relationship may be more likely to over-report difficulties in the parent-child relationship. This could bias the results, with participants reporting more problems in the parent-child relationship than were actually present.

BN: In contrast to families of AN individuals, families with a BN individual have been found to be more disturbed and less involved than AN families or families with no eating pathology (Humphrey, 1989; Kog & Vandereycken, 1989). BN individuals have been shown to perceive their parental figures as unstable and less likely to provide structure than control subjects (Berg, 2000; Johnson & Flack, 1985; Kog &

Vandereycken, 1989). Research shows that BN individuals perceive their family environment to be more disorganized and less cohesive than control families (Humphrey, 1986; Johnson & Flach, 1985).

Further, studies show that individuals with BN experience their parents to be less supportive and involved than control families (Leung, Thomas, & Waller, 2000; Pole et al., 1988). For example, Pole et al. (1988) compared 56 women with BN to a control group without eating pathology on the PBI. Because of theories which suggest that BN parents are less emotionally involved and supportive to their BN daughters (Johnson, 1991; Marsden, 1997), the authors hypothesized that BN individuals would experience their parents as less caring than individuals without eating pathology. In essence, the lack of parental involvement in BN families may lead BN individuals to feel that their parents care less about them.

The study results supported hypotheses; the BN group perceived their parents to be significantly less caring, emotionally involved, and supportive than controls without eating pathology. These results confirm theoretical accounts of the parent-child relationship in BN families. However, this study had similar limitations to the previous ones discussed for AN; in particular, only one perspective of the parent-child relationship was obtained and participants were adults rather than adolescents.

Summary: Taken together, empirical research has confirmed the presence of theorized patterns of parent-child relationships among individuals with AN and BN.

However, one limitation that was evident across studies was the use of adult participants that could lead to biases in recalling past experiences with parents. In addition, most of the studies used only one report of the parent-child relationship, instead of obtaining

multiple perspectives. Further, most studies did not compare parent-child relationships among different types of eating disorders.

Separation-individuation.

As noted above, there have been few studies examining the relationship between separation-individuation and eating disorders. Most of the studies that have been conducted examined general disordered eating symptoms rather than eating disorder diagnoses per se, and few have looked at differences between AN and BN sufferers in this area. In addition, none of these studies focused on the adolescent period, but instead examined college students. This is a significant limitation because adolescence is the developmental period when significant separation-individuation difficulties are hypothesized to occur and when disordered eating most commonly begins. The current study will address this limitation by being the first to examine the separation-individuation process during the critical adolescent period.

Even though the studies focused on college populations, they provide some insight into whether separation-individuation issues are related to eating pathology. Studies that have focused on eating disorders symptoms in general have found that greater difficulty with separation-individuation (i.e., excessive responsibility to parents, excessive need for parental approval), more dependency in relationships with others (i.e., anxiety about being separated from significant others and seeking nurturance from others), and a diminished sense of individuality, are predictive of disordered eating behaviors in female college students (Friedlander & Siegel, 1990; Marsden, Meyer, Fuller, & Waller, 2002; Rhodes & Kroger, 1992). However, the lack of differentiation between AN- and BN-like syndromes make these results difficult to interpret. Thus,

although the findings attest to the predictive significance of separation-individuation difficulties for disordered eating, they do not provide critical information about the types of difficulties most predictive of AN and BN.

Only one study has compared separation-individuation difficulties in women with AN and BN. Smolak and Levine (1993) examined four groups of college women: 1) women with BN (N = 19) who reported three or four symptoms of BN and two or fewer AN symptoms; 2) women with restricting AN (RAN) (N = 8) who reported three or more AN symptoms but two or fewer BN symptoms; 3) women with "bulimia anorexia" (BULAN) (N = 9) who reported three or more bulimic symptoms and three or more AN symptoms; and 4) control women without eating pathology. Findings revealed that the BULAN group was similar to the BN group on measures of separation-individuation (e.g., having more denial about their dependency on others and more extreme independence in their values and beliefs). Thus, the two groups were combined for analyses. This suggests that individuals with AN binge-eating/purging type are more similar to BN individuals than individuals with AN restricting type in terms of their separation-individuation difficulties.

The results generally supported the theories of separation-individuation described above; the RAN group showed more dependency in general than the BN/BULAN or control groups, while the BN/BULAN group showed greater overseparation in how much their beliefs, values, and attitudes differed from their parents'. The BN/BULAN group also reported more guilt, mistrust, and resentment about being in relationships with other people. Findings from this study are significant in providing empirical support for theories postulating that individuals with BN symptoms

show over-separation from parents, while individuals with AN symptoms show underseparation. Although the sample sizes were relatively small within each group, group differences still emerged that suggest that effects were large and clinically significant.

In summary, despite extensive theory, very little research has examined separation-individuation and eating disorders. No research has examined these relationships in adolescence, the developmental period in which disordered eating most often begins. Most studies have not examined differences among types of disordered eating symptoms, and the one study that did examine these differences (Smolak & Levine, 1993) had small sample sizes, although predicted differences emerged between groups. Finally, these studies did not examine how relationships with parents impact the separation-individuation process, despite extensive theory regarding these relationships.

Summary.

Taken together, there is empirical evidence that parents in AN families are experienced as more overprotective, less caring, and more enmeshed than control families. BN families are found to be less caring and more erratic than control families. Research also shows that individuals with eating disorders have more separation-individuation difficulties than individuals without eating disorders. In addition, there is some preliminary evidence that AN women show more difficulties with under-separation and that BN women show more difficulties with over-separation. However, no research has examined whether the aforementioned parenting behaviors contribute to these patterns of over- and under-separation.

Previous research examining the parent-child relationship and eating pathology were constrained by several limitations that need to be addressed in future studies. These

studies gathered information from a single informant, rather than obtaining multiple perspectives of the parent-child relationship. In addition, few studies have examined parent-child relationships in both AN and BN, to see if theorized differences exist.

Finally, the studies utilized adult populations, instead of child or adolescent populations that could have led to retrospective recall biases in reports of parent-child relationships.

Research investigating associations between separation-individuation and eating pathology has been equally plagued with problems. All of this research was conducted with a college-aged rather than an adolescent population. Consequently, no research has been done to either support or fail to support theory that separation-individuation difficulties during adolescence are associated with eating pathology. Further, with the exception of one study, research has not examined differences in separation-individuation among AN and BN. Finally, studies have examined separation-individuation, the parent-child relationship, and disordered eating separately instead of examining the combined effects of the variables, despite theory which suggests that specific parenting behaviors lead to the development of eating pathology through their effects on the separation-individuation process. It is critical to examine these variables together in one model in order to gain a complete and comprehensive understanding of the role of parents and separation-individuation in the development of eating pathology.

Effects of Pubertal Development

An additional limitation of previous research is that studies have not examined the impact of puberty. Research has consistently shown that puberty contributes to the development of eating disorders during adolescence (Dorn, Crokett, & Petersen, 1988).

Although direct links between puberty and disordered eating have been hypothesized

(i.e., increases in body fat during puberty lead to increases in disordered eating; Koff & Rierdan, 1993), indirect links that operate through parent-child relationships and separation-individuation processes may be equally important. For example, it may be that puberty influences the development of eating disorders by moderating relationships between the parent-child relationship and separation-individuation. This may occur because pubertal development is a marker of adolescence and the need to individuate. Girls who are more advanced in pubertal development likely experience greater needs to individuate and may consequently become more involved in conflictual parent-child relationships. These stressors, in combination with previous separation-individuation difficulties during infancy, may increase the chances that a girl will develop AN or BN. In essence, pubertal development may act to intensify already existing conflicts as well as the need to resolve separation-individuation difficulties.

To date, no studies have examined the moderating role of puberty on associations between parent-child relationships, separation-individuation, and eating pathology.

However, research in other areas has confirmed the significant influence of pubertal development on parent-child relationships as well as the separation-individuation process.

Puberty and Parent-Child Relationships

Numerous studies have examined associations between puberty and the parent-child relationship in general (Hill, Holmbeck, Marlow, Green, & Lynch, 1985; Savin-Williams & Small, 1986; Steinberg, 1987). One trend has emerged across studies; more advanced pubertal development has been associated with increased distance and less cohesion in the parent-child relationship (Hill et al., 1985; Larson & Richards, 1991; Steinberg, 1988; Steinberg, 2001). For example, Steinberg (1988) conducted a

longitudinal study that included 157 families with a firstborn adolescent child between 11 and 16 years of age. Parents and their adolescent children were assessed initially and then again one year later. Closeness in the parent-child relationship was assessed using parent and adolescent reports on several measures. Pubertal development was measured by research assistants, who rated adolescents on visible signs of secondary sex characteristics such as facial shape, body proportion, and chest and hip development. Results showed that pubertal maturation was associated with more distance in the parent-child relationship, based on parent and adolescent reports. Adolescents who were further in pubertal development and their parents reported less calm communication and cohesion than those who were less advanced in pubertal development. Findings from this study suggest that there are significant changes in the parent-child relationship that take place during puberty; specifically, more emotional distance is associated with pubertal maturation.

In addition to research that shows the parent-child relationship becomes more distant with advanced pubertal development, studies show that parental behavior may change during puberty. Specifically, research shows that adolescents experience their parents as behaving in a more controlling and restrictive manner as the adolescent advances in pubertal development (Steinberg, 1987). This change in parental behavior may occur as parents sense the impending separation of their adolescent and/or parents worry about rebellious, risky behaviors that their adolescent children might start engaging in. Taken together, it seems that despite parental attempts to exert more control over their adolescent children, there is emotional distancing in the parent-child relationship during puberty.

It is important to consider the implications of these findings for adolescent eating disorders. Because research shows that parents of adolescents behave in a more controlling and restrictive way during puberty (Steinberg, 1987), mothers of AN daughters who generally behave in an overprotective manner (i.e., they are excessively controlling and intrusive) before the daughter reaches puberty are likely to behave in an even more controlling way during puberty. This likely increases adolescent difficulties with separation, and adolescents may consequently develop AN as a way to prevent the natural distance in the parent-child relationship that occurs during this period.

There is also evidence that more distance in the parent-child relationship is associated with pubertal maturation (Hill et al., 1985; Larson & Richards, 1991; Steinberg, 1988). It is likely that in families with less involved parents, distance increases during puberty, which could lead to more overseparation and the development of BN symptoms. Therefore, pubertal development may exacerbate parenting practices, which may strain the parent-child relationship and increase difficulties with separation-individuation and disordered eating symptoms.

Puberty and Separation-Individuation

Empirical research has not examined the relationship between puberty and separation-individuation specifically, despite some theories suggesting that puberty drives the separation-individuation process through its effect on the parent-child relationship (see review above and Blos, 1967; Steinberg, 1987). However, studies have focused on the relationship between puberty and constructs related to separation-individuation, such as autonomy from parents. Indeed, more advanced pubertal development has been associated with greater behavioral and emotional autonomy

(Savin-Williams & Small, 1986; Steinberg, 1987; Steinberg, 1988). For example, Steinberg (1987) examined a sample of 204 families with a child between the ages of 10 and 15 to examine the impact of puberty on adolescent autonomy. Mothers reported on autonomy in the parent-child relationship, while adolescents reported on their own emotional autonomy.

Results showed that maternal reports of autonomy were positively associated with pubertal maturation, such that greater pubertal development was associated with more autonomy within the parent-child relationship. Adolescent reports showed that pubertal maturation was associated with more emotional autonomy. Specifically, adolescents who were more advanced in pubertal development reported more behavioral and emotional autonomy.

Findings provide some empirical evidence that pubertal maturation is associated with greater autonomy for the adolescent. Because becoming autonomous is a crucial part of the separation-individuation process, findings linking puberty and autonomy suggest that pubertal development is salient to separation-individuation as well.

Individuals who are vulnerable to difficulties with separation-individuation will likely face more intense conflict in this area during puberty, because it is a developmental period associated with becoming more autonomous from parents.

Summary

Puberty is a developmental period that is associated with the need to become more psychologically and physically separate from parents and family, which could strain parent-child relationships and be associated with conflict around separation-individuation. Research suggests that pubertal development is associated with more

emotional distance in the parent-child relationship, in addition to more parental control and restricting behaviors (Hill et al., 1985; Steinberg, 1987). Therefore, with more advanced pubertal development, existing parental tendencies may become exacerbated, which likely strains the parent-child relationship and increases conflict around separation-individuation during this developmental period. In addition, there is evidence that more advanced pubertal development is associated with more emotional and behavioral autonomy (Savin-Williams & Small, 1986; Steinberg, 1987). This suggests that pubertal development affects processes related to separation-individuation and may increase pre-existing conflicts for individuals who are vulnerable to separation difficulties. This increased conflict around separation-individuation could consequently influence the development of eating pathology, with AN symptoms developing as a way to prevent separation and BN symptoms as a way to maintain overseparation and gain control.

However, the studies described above have not examined separation-individuation difficulties per se, and have not examined relationships with eating pathology.

Additional research is therefore needed to help understand the effects of puberty on the development of eating pathology through its influence on the parent-child relationship and separation-individuation. The current study will address this need by examining parent-child relationships, separation-individuation, disordered eating, and pubertal development together in one model.

Effects of Temperament

Temperament and personality are also important factors in understanding the relationship between separation-individuation and eating pathology during adolescence.

As noted above, personality and temperament have been identified as significant risk

factors for eating disorders (Clark, Watson, & Mineka, 1994; Leon et al., 1999; Lilenfeld et al., 2006; Watson, Clark, & Harkness, 1994). While direct links have been made between temperament and eating pathology, temperament has not been examined as a moderating factor between separation-individuation and disordered eating. It is possible that temperamental style may influence whether individuals who are experiencing intense separation-individuation difficulties during adolescence develop disordered eating symptoms.

Temperament and Eating Pathology

Particular temperamental styles have been associated with the development of eating pathology. More negative affectivity has been associated with the development of eating pathology (Keel et al., 1997; Leon et al., 1995; Lilenfeld et al., 2006). For example, Leon et al. (1999) conducted a prospective longitudinal study with adolescents ages 13 to 18. They found that boys and girls who reported high levels of negative affectivity (i.e., negative feelings, emotions, or moods, increased anxiety, high stress reactivity) were most likely to develop disordered eating attitudes and behaviors over a period of 2 years. Further, temperament was more predictive of the development of disordered eating than other variables, such as other forms of psychopathology. In other related studies, impulsivity was also shown to predict the development of BN symptoms (Stice & Agras, 1998; Wonderlich, Connolly, & Stice, 2004).

Cross-sectional studies show that AN and BN are associated with higher levels of negative affect and harm avoidance, and lower levels of positive affect (Bulik, Sullivan, Weltzin, & Kaye, 1995; Leon et al., 1999; Lilenfeld et al., 2000; Pryor & Wiederman, 1996). Women with AN have been characterized as having a more controlled

temperamental style, by being more rigid, constricted, and perfectionistic (Casper, Hedeker, & McClough, 1992; Kleifield, Sunday, Hurt, & Halmi, 1994; Klump et al., 2000; Pillay & Crisp, 1977; Pryor & Wiederman, 1996; Sohlberg & Strober, 1994; Tomotake & Ohmori, 2002). BN women have been found to be more impulsive than individuals with AN and women without eating pathology (Bulik et al., 1995; Casper et al., 1992; Fassino et al., 2002; Klump et al., 2003; Lilenfeld et al., 2000; Tomotake & Ohmori, 2002). Overall, it appears that individuals with certain personality characteristics or temperamental styles are more likely to develop eating disorders.

Temperament and Separation-Individuation

Research has not examined the influence of personality characteristics or temperament on the separation-individuation process. It has been suggested that the functional significance of temperament is its influence on responses to experiences that are stressful for the individual (Strelau, 2001). Therefore, children who have high levels of negative affectivity may have greater difficulty dealing with stressful situations than children who have low levels of negative affectivity as they may have more trouble modulating their emotional arousal (Compas, Connor-Smith, & Jaser, 2004). Further, responses to the intensification of conflict around separation-individuation during adolescence may be affected by temperament. Specifically, difficulties with separation-individuation may be more likely to lead to disordered eating during adolescence if the individual has a certain type of temperamental style (i.e., more negative affect, less positive affect). To date, no studies have examined whether temperament influences the relationship between separation-individuation and disordered eating.

Summary

Overall, studies show that personality characteristics and temperament are associated with the development of eating pathology. Specifically, control and rigidity are associated with AN and impulsivity is associated with BN (Casper et al., 1992; Kleifield et al, 1994; Klump et al., 2000; Pillay & Crisp, 1977; Pryor & Wiederman, 1996; Sohlberg & Strober, 1994; Tomotake & Ohmori, 2002). More negative affect, less positive affect, and harm avoidance are associated with all forms of eating pathology (Bulik et al., 1995; Leon et al., 1999; Lilenfeld et al., 2000; Pryor & Wiederman, 1996).

In addition to direct effects, temperament may also influence the development of eating pathology indirectly through its influence on the relationship between separation-individuation difficulties and disordered eating. It is possible that during adolescence, when separation-individuation conflicts intensify, individuals having difficulty with separation-individuation who also have a certain temperamental style (e.g., more negative affect and harm avoidance, less positive affect) would be more likely to develop disordered eating symptoms than individuals with a different temperamental style. Importantly, studies examining relationships between temperament and eating pathology have not examined potential indirect effects of temperament on these relationships.

Conclusions

Overall, theories suggest that parent-child relationships influence the process of separation-individuation which may lead to the development of eating pathology (Johnson, 1991). Within AN families, the process of separation-individuation may be inhibited due to the enmeshment of familial relationships, and this could result in underseparation of the AN individual (Johnson, 1991; Newton, 2005). Within BN families, the BN individual may lean towards over-separation from a hostile and chaotic family

environment, but may remain bound up in conflicted parent-child relationships. The BN individual may find that the most effective way to individuate is through over-separation, as a way to distance herself from her family (Smolak & Levine, 1992). These separation-individuation difficulties likely become prominent during adolescence, when pressures for individuation increase and internal conflicts that were unresolved in infancy become prominent. These difficulties may then lead to different types of disordered eating symptoms; restricting and overcontrolling food intake symptoms in girls with AN and binging and purging symptoms in girls with BN.

Additional research is needed to support these theories and to further delineate the link between separation-individuation, parent-child relationships, and disordered eating. Little empirical work has been done in this area, and no studies have examined these processes in adolescence. In addition, the role of pubertal development and temperament in these relationships has not been examined, despite their potentially important role in moderating relationships between the parent-child relationship, separation-individuation, and disordered eating.

Study Overview

This study addresses limitations of previous research by examining the relationship between separation-individuation, mother-child relationships, disordered eating, puberty, and temperament in adolescent girls. Participants included 202 girls between the ages of 10 and 14 who participated in an on-going adolescent twin study. Relationships among the variables were examined using structural equation modeling (SEM) that allowed for associations between variables to be examined in one integrative model

Based on theories described above, this study tested a full mediation model where mother-child relationships led to difficulties with separation-individuation, which then led to disordered eating. A mediator is defined as a variable that explains the relationship between other variables of interest (Mash & Wolfe, 2002). In this model, separationindividuation was proposed to be a mediator because the mother-child relationship is hypothesized to influence separation-individuation difficulties for the child that during adolescence, may result in disordered eating. Moreover, puberty was postulated to moderate relationships between the mother-child relationship and separationindividuation, and between separation-individuation and disordered eating, in the prediction of disordered eating. A moderator is defined as a variable that influences the strength of the relationship between variables of interest (Mash & Wolfe, 2002). Therefore, puberty was proposed to be a moderator in the model by exacerbating existing dynamics in the mother-child relationship, which could lead to increased conflict around separation-individuation for the adolescent. Puberty was also examined as a moderator in the relationship between separation-individuation and disordered eating; conflict around separation-individuation may intensify with more pubertal maturation, which could result in more disordered eating attitudes and behaviors.

Finally, temperament was proposed to moderate relationships between separation-individuation and disordered eating, by influencing how separation-individuation difficulties are managed and expressed; for individuals who are experiencing separation-individuation difficulties, having a particular temperamental style could make it more likely for these individuals to develop eating pathology. Based on previous prospective

research (see above), temperament was also predicted to have direct effects on disordered eating.

Adolescent reports of disordered eating (i.e., bd, wp, be, rs) were used as the outcome variables, while adolescent (i.e., variables with the "a" prefix) and maternal (i.e., variables with the "m" prefix) reports were used to measure the mother-child relationship (i.e., acare, aprotect, mcare, mprotect), separation-individuation (i.e., aenmesh, aengulf, anurtseek, asepanx, adepden, ahealthsep, maaad), pubertal development (i.e., apuberty, mpuberty), and temperament (i.e., anegaffect, ashy, ainhib, mnegaffect, mshy, minhib).

The dependent variables were disordered eating attitudes and behaviors characteristic of restricting type AN ("AN-like" symptoms - weight preoccupation, body dissatisfaction, and dietary restraint), as well as those associated with BN or the binge purge type of AN ("BN-like" symptoms - binge eating). These symptoms were examined as dependent variables in separate models in order to examine mother-child relationships and difficulties with separation-individuation found among different types of disordered eating. Although weight preoccupation, body dissatisfaction, and dietary restraint are present in individuals with all types of eating disorders, these characteristics are present in a more extreme form in individuals with restricting type AN, and thus are included as AN-like rather than BN-like symptoms.

Taken together, these models and analyses improved upon previous research in this area by examining the relationship between separation-individuation, mother-child relationships, disordered eating, puberty, and temperament in an integrative model. It examined girls during adolescence, which is a time that disordered eating most commonly begins and separation-individuation issues are salient. In addition, reports

were obtained from multiple informants (i.e., adolescent girls and their mothers).

Further, different types of disordered eating were examined; distinct patterns among variables were expected for various types of disordered eating based on theory and research.

Aims and Hypotheses

Aim #1: To examine associations between the mother-daughter relationship, separation-individuation difficulties, and AN-like types of disordered eating.

Hypothesis 1: <u>Under-separation</u> (i.e., more adolescent reported enmeshment, nurturance seeking, separation anxiety; less adolescent reported healthy separation; more maternal reported anxiety about adolescent distancing) will mediate the relationship between an <u>enmeshed</u> mother-daughter relationship (i.e., more adolescent and maternal reported maternal protection) and <u>AN-like disordered eating</u> (i.e., weight preoccupation, body dissatisfaction, and dietary restraint.

Aim #2: To examine associations between the mother-daughter relationship, separation-individuation difficulties, and BN-like types of disordered eating.

Hypothesis 2: Over-separation (more adolescent reported denial about their dependency on others and engulfment anxiety; less adolescent reported healthy separation; more maternal reported anxiety about adolescent distancing) will mediate the relationship

between an <u>uninvolved</u> mother-child relationship (less adolescent and maternal reported maternal care and protection) and <u>BN-like disordered eating</u> (i.e., binge eating).

Aim #3: To examine the impact of puberty on relationships between the mother-child relationship, separation-individuation, and AN- and BN-like disordered eating.

Hypothesis 3a: Puberty will moderate the relationship between the mother-child relationship and separation-individuation in both AN-like and BN-like disordered eating. A more enmeshed mother-child relationship will be associated with more underseparation if puberty is more advanced than if it is less advanced. In addition, more advanced puberty will amplify the effects of an uninvolved mother-child relationship on the development of over-separation.

Hypothesis 3b: Puberty will moderate the relationship between separation-individuation and AN-like and BN-like disordered eating. More advanced puberty will increase the impact of under-separation and will be associated with more AN-like disordered eating. More over-separation will lead to more BN-like disordered eating when pubertal development is more advanced.

Aim #4: To examine direct and moderating effects of temperament on AN-like and BN-like disordered eating.

Hypothesis 4a: Temperament will predict AN-like and BN-like disordered eating. A more controlled temperamental style (more adolescent and maternal reported impulse control, negative affect, and avoidance of harm) will predict more AN-like disordered eating, while a more impulsive temperamental style (more adolescent and maternal reported negative affect and avoidance of harm, and less impulse control) will predict more BN-like disordered eating.

Hypothesis 4b: Temperament will moderate the relationship between separation-individuation and AN-like and BN-like disordered eating. More under-separation will be associated with more AN-like disordered eating when temperament style is more controlled. More over-separation will be associated with more BN-like disordered eating when temperament style is more impulsive.

Aim #5: To compare the effect of puberty and temperament as moderators in the model.

Hypothesis 5: This is viewed as an exploratory aim, however based on previous research that shows temperament to be an extremely robust predictor of eating pathology, temperament is predicted to be a more significant predictor in the model.

METHODS

Participants

Participants included a sample of 202 female twins between the ages of 10 and 14 and their mothers who took part in Dr. Klump's on-going Adolescent Twin Study of Behavioral Adjustment and Development from the Michigan State University Adolescent Twin Registry (Klump & Burt, in press). Twins were used in this study as a convenience sample that was ideal for the current study's hypotheses in terms of the age of twins studied and the inclusion of maternal reports. The age breakdown (Age M=12.48, SD=1.39) of participants was as follows: 38 10-year-olds, 46 11-year-olds, 42 12-yearolds, 38 13-year-olds, 38 14-year-olds. The ethnic breakdown of the sample was as follows: 87.1% Caucasian, 7.9% African-American, 2% Biracial, and 3% "Other". The Hollingshead index of social position (Hollingshead, 1975) was used to calculate socioeconomic status and indicated that the breakdown was as follows: 4.4% Category 1 (unskilled laborers, menial service workers), 4.4% Category 2 (machine operators, semiskilled workers), 28.6% Category 3 (skilled craftsmen, clerical, sales workers), 51.6% Category 4 (medium business, minor professional, technical), and 11% Category 5 (major business and professional). The mean score was in the middle to upper SES range (i.e., M=3.60, SD=.91).

Recruitment of the twins involved collaboration with the Michigan Department of Community Health (MDCH) and the Michigan Bureau of Integration, Information, and Planning Services (MBIIP) to identify twins within the age range in Lansing and the surrounding areas. The MDCH manages birth records and can identify all twins born in the state of Michigan in a given year. The MDCH works with the MBIIP to locate

current addresses for the twins through driver's license information of their parents. The recruitment procedures ensured anonymity of the twins and their parents until they agreed to participate in the study.

The MDCH first identified female twins between the ages of 10 and 14 within 75 miles of the laboratory, and found their parent's current addresses through the MBIIP database. MDCH then mailed recruitment packets provided by our project staff to parents that contained information about the study and a reply postcard indicating their children's interest in participating in the study. Families who were interested were contacted by our project staff and multiple mailings were sent out to encourage a higher response rate. The response rate using this method of recruitment was 35%.

Measures

Adolescents completed measures about themselves, while mothers completed measures separately for both twins. Two of the measures, the SITA and the Eating Disorders Examination Questionnaire (EDEQ; Fairburn & Beglin, 1994), were read to adolescents who were between the ages of 10 and 12, because they may have had some difficulty understanding the items and may have had questions about their content. Table 1 includes internal consistency reliability estimates for subjects in the current study for all of the independent and dependent measures.

Adolescent Report

Disordered Eating

Minnesota Eating Behavior Survey: Adolescent participants completed the 30item Minnesota Eating Behavior Survey (MEBS; Klump, McGue, & Iacono, 2000; von

Ranson et al., 2005) to assess eating attitudes and behaviors. The MEBS¹ is a revised version of the original Eating Disorders Inventory (EDI; Garner, Olmsted, & Polivy, 1983); the original MEBS was modified by Minnesota Twin Family Study (MTFS) researchers to make it suitable for use with preadolescent girls. A previous factor analysis (Klump et al., 2000) of the MEBS yielded four subscales: Body Dissatisfaction (i.e., dissatisfaction with the size and/or shape of one's body), Weight Preoccupation (i.e., preoccupation with dieting, weight, and the pursuit of thinness). Binge Eating (i.e., the tendency to engage in episodes of overeating as well as having attitudes conducive to binge eating), and Compensatory Behavior (i.e., the tendency to use or contemplate using inappropriate compensatory behaviors such as self-induced vomiting and laxatives to control weight). The MEBS also includes a total score, which is the combined score of all 30 items. The MEBS scales are scored in the traditional "pathological" direction with high scores indicating greater degrees of the measured construct. The Compensatory Behavior subscale was not included in analyses because of its low reliability (alpha = .40) and low frequency of item endorsement among early adolescents (Klump et al., 2000).

The reliability and validity of the MEBS have been supported by previous studies (Klump et al., 2000; von Ranson et al., 2005). Specifically, internal consistency and three year test-retest statistics were adequate among the subscales. It is noteworthy that the test-retest statistics remained high over a three-year span, as these values reflect not only

¹ The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory (M-EDI)) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

measurement error but also trait stability over a three-year period when numerous situational changes are occurring (i.e., going to college). These statistics thus attest to the strong reliability of the measure. Previous studies have also supported the ability of the MEBS to discriminate between eating disordered subjects and controls (von Ranson et al., 2005). In the current study, internal consistency ranged from .71-.82 for the subscales.

Restraint Scale: Adolescent participants also completed the Restraint subscale of the Eating Disorders Examination Questionnaire (EDEQ; Fairburn & Beglin, 1994) that assesses levels of dietary restraint. The EDEQ is a self-report measure that was derived from the Eating Disorders Examination (EDE; Fairburn & Cooper, 1993), a semi-structured interview assessing core attitudinal and behavioral aspects of eating disorders. The Dietary Restraint scale measures participants' level of deliberate and planned weight control (e.g., attempting to eat less when feeling concerned about one's weight, trying not to eat between meals because of watching one's weight). The EDEQ focuses on behavior and attitudes of the past four weeks and asks participants to rate their behavior or attitudes on a 7-point scale of severity or frequency.

The psychometric properties of the EDEQ, and of the Restraint scale in particular, have been shown to be excellent across numerous studies (Fairburn & Beglin, 1994; Luce & Crowther, 1999; Mond et al., 2004). The Restraint scale has been shown to have excellent internal consistency reliability (alpha = .84-.85), test-retest reliability over a two-week period (r = .81) (Luce & Crowther, 1999), and adequate temporal stability over approximately 300 days (r = .57) (Mond et al., 2004). Validity of the EDEQ has been established by comparing scores on the EDEQ with scores on the EDE among clinical

and community samples (Fairburn & Beglin, 1994). A high level of agreement is found among these scores, indicating that the use of a self-report measure is comparable to an interview method for assessing restraint and other types of eating pathology. The Restraint scale demonstrated adequate internal consistency in this study (alpha=.74).

Separation-Individuation

Separation-Individuation Test of Adolescence: Levels of separation-individuation among adolescents in relation to their parents were measured with the 103-item

Separation-Individuation Test of Adolescence (SITA; Levine, Green, & Millon, 1986).

Adolescents completed this self-report inventory of statements about relationships with parents, teachers, and peers. Examining relationships in all of these areas, rather than just focusing on the parent-child relationship, enabled a more comprehensive assessment of the construct, since theory suggests that separation-individuation difficulties generalize to relationships other than the parent-child relationship (Edward et al., 1981).

The SITA statements assess levels of emotional closeness to significant others and anxiety in various types of interpersonal relationships associated with the emotional closeness or potential loss of closeness. The statements were generated based on Mahler's work describing separation-individuation difficulties in infancy (1975).

Participants evaluate how true the SITA statements are for themselves using a 5-point Likert scale ranging from "strongly agree or is always true for me" to "strongly disagree or is never true for me." The SITA consists of seven subscales derived through factor analysis: Nurturance-Seeking (i.e., the extent to which a person desires to be taken care of and dependent on others), Enmeshment-Seeking (i.e., the extent to which a person feels that closeness with others requires a merging and fusing with the other person),

Engulfment-Anxiety (i.e., the extent to which a person fears that close interpersonal relationships will cause them to lose their sense of independence), Separation-Anxiety (i.e., the extent to which a person has strong fears of losing emotional or physical contact with an important other), Dependency-Denial (i.e., the extent to which person denies or avoids dependency needs), Self-Involvement (i.e., the extent to which a person possesses a high degree of narcissism and self-centeredness), and Healthy-Separation (i.e., the extent to which a person has progressed successfully toward resolution of the conflicts involved with separation-individuation).

The subscales that were analyzed in this study included the Nurturance Seeking, Separation Anxiety, Enmeshment Seeking, Dependency Denial, Engulfment Anxiety, and Healthy Separation scales because they appeared most relevant to the separation-individuation difficulties typically associated with eating disorders. Higher scores on these SITA subscales indicate more of the construct measured.

Internal consistency was established in the development of the measure (Levine et al., 1986) and follow-up studies investigating construct validity have supported its validity (McClanahan & Holmbeck, 1992). Alpha reliability ranges from .64 - .88 among the subscales (Levine et al., 1986). Findings indicate that the SITA maintains its internal structural properties among a clinical population (Levine & Saintonge, 1994). The SITA has also been found to discriminate among personality types on the Millon Adolescent Personality Inventory (MAPI; Millon, Green, & Meagher, 1982) (Levine et al., 1986). For example, the "Confident-Outgoing" group, defined by the MAPI, scored highest on the Healthy Separation scale, in comparison to other groups such as the "Anxious-

Moody" and "Dependent-Conforming" groups. In the current study, the internal consistency of the SITA scales was excellent and ranged from .78-.94.

Mother Report

Parental Separation Anxiety

Mothers of adolescents completed the Parents of Adolescents Separation Anxiety Scale (PASAS; Hock et al., 2001) that assess the mother's response to the growing independence of her adolescent daughters. Although it would be ideal to use a measure that assesses the mother's view of the child's separation difficulties, this type of measure does not exist. As will be discussed in greater detail below, maternal reports on the PASAS have been found to correlate with adolescent reports of difficulties with differentiation from the parent (Hock et al., 2001). In addition, research indicates that parents who foster independence as well as connectedness in their children provide the optimal environment for identity development during adolescence (Cooper, Grotevant, and Condon, 1983).

The PASAS consists of 34 items and yields two subscales derived from factor analysis: Anxiety about Adolescent Distancing (i.e., the mother's feelings of discomfort or loss associated with her adolescent's decreasing involvement with parents and increasing affiliation with others) and Comfort with Secure Base Role (i.e., the mother's contentment in being accessible and serving as a source of security to her adolescents who are expanding their social and physical worlds). Higher scores on the Anxiety about Adolescent Distancing subscale indicate more hurt or emptiness in response to adolescent distancing, while lower scores on this subscale are indicative of mothers who see the growing independence of the adolescent as more positive and less anxiety provoking for

the mother. Higher scores on the Comfort with Secure Base Role reflect mothers who are comfortable in their role providing support when needed, without feeling hurt when not needed by their adolescent child. Lower scores on this subscale indicate discomfort with being accessible and looking forward to diminished responsibility for providing support to their child.

Both the Anxiety about Adolescent Distancing and the Comfort with Secure Base Role subscales have been shown to have adequate internal consistency (alpha = .86 and .82, respectively). In the current study, the Anxiety about Adolescent Distancing subscale demonstrated higher internal consistency than the Comfort with Secure Base Role subscale (alpha = .88 and .68, respectively). Maternal reports using this measure have been found to correlate highly with adolescent reports of attachment to mother and differentiation from mother (Hock et al., 2001). Specifically, healthier attachment, as reported by adolescents, was associated with lower maternal scores on the Anxiety about Adolescent Distancing subscale and with higher scores on the Comfort with Secure Base Role subscale. In addition, self-other differentiation based on adolescent report was positively associated with higher maternal Comfort with Secure Base Role and to lower maternal Anxiety about Adolescent Distancing (Hock et al., 2001).

Adolescent and Mother Report

Mother-Child Relationships

Mother-child relationships were assessed with the Parental Bonding Instrument (PBI; Parker, Tupling, & Brown, 1979), which measures the quality of the mother-child relationship. There are few measures that enable parents and children to report on aspects of the parent-child relationship that are important in understanding eating

disorders. Many measures of the parent-child relationship access parental and child behavior towards one another, and do not access underlying relational constructs important to the development of eating disorders, such as levels of enmeshment. The PBI has been used in many studies of girls and women with eating disorders because it does access these important constructs (Bulik et al., 2001; Rhodes & Kroger, 1992). It is the only existing measure of the parent-child relationship that can be used to study the construct of enmeshment that is often associated with eating disorders.

The PBI consists of two subscales derived from factor analysis: Protection and Care. The Protection subscale assesses the positive and negative aspects of overprotection. The positive pole of Protection (low scores on the Protection subscale) is characterized by promotion of independence and autonomy and the negative pole (high scores) involves intrusion, excessive contact, and prevention of independent behavior. The positive pole of the Care subscale (high scores on the Care subscale) are defined by affection, emotional warmth, empathy, and closeness. The negative pole (low scores) consists of emotional coldness, indifference, and rejection. Higher scores on both subscales indicate experiencing more of the parental characteristic assessed.

Both adolescent and mother reports were obtained on the PBI, with mothers reporting on their relationship with each adolescent daughter separately, and adolescents reporting on their relationship with their mother. Nonetheless, adjustments to the PBI were necessary for use in this study in order to obtain adolescent and mother reports of the mother-child relationship. First, the PBI was originally designed to be a retrospective measure, with young adult subjects reporting on their experiences while they were growing up. Thus, the PBI was modified in this study to refer to the present. For

example, the statement "Liked me to make my own decisions" was changed to "Likes me to make my own decisions."

In addition, the original form of the PBI is designed for informant reports on the same relationship; typically everyone reports on their relationships with their parents, not on their relationship with their children. In order to attain the mother's perspective on the mother-child relationship, the PBI was adjusted so that the mother could report on her behavior and characteristics in regard to her daughter. Mothers were asked to report on their relationship with each daughter, describing how characteristic the statements are of their relationship with their child at the present time. For example, the statement "Lets me do those things I liked doing" was changed to "I let her do those things she likes to do."

Psychometric properties of the PBI have been found to be sufficient (Parker et al., 1979). Test-retest reliability over a 3-week period yielded coefficients of .63 on the Protection scale and .76 on the Care subscale (Parker et al., 1979). Split-half reliability has also been found to be adequate, with a correlation of .69 for the Protection scale and .88 for the Care subscale (Parker et al., 1979). Internal consistency was .76 for the Care scale and .63 for the Protection scale (Parker et al., 1979). Concurrent validity was assessed by comparing child's scores on the PBI with scores by raters who completed interviews about the relationship between parents and their children. These scores correlated with a coefficient of .78 for the Care scale and .51 for the Protection scale (Parker et al., 1979). In the current study, maternal reported Protection had an alpha reliability of .68, and the rest of the scales had alpha reliabilities above .74.

Pubertal Development

Adolescents and mothers completed the Pubertal Development Scale (PDS; Petersen, Crockett, Richards, & Boxer, 1988). The PDS assesses current pubertal development in the area of secondary sex characteristics including height spurts, pubic hair growth, skin changes, breast development, and initiation of menses. These areas were assessed for the current time. Adolescents reported on themselves and mothers reported on each adolescent daughter. Pubertal development was assessed with average scores (Petersen et al, 1988)

The PDS is a well-validated measure showing good internal consistency with alpha coefficients ranging from .68-.83, among the pubertal indices. In the current study, alpha coefficients for the average PDS score ranged from .82-.86. Criterion validity has been demonstrated, with high correlations between the PDS and physician ratings of pubertal development (.61-.67) (Brooks-Gunn et al., 1987). Scores on the PDS have also been compared with interview ratings. Interviewers can observe some of the adolescent's physical characteristics that can give them more information with which to evaluate pubertal development. Correlations between the interview ratings and scores on the PDS was variable and ranged from .41-.79, providing some evidence for the validity of the PDS (Brooks-Gunn et al., 1987).

Temperament

A short form of the Early Adolescent Temperament Questionnaire – Revised (EATQ-R; Capaldi & Rothbart, 1992) was used to assess adolescent temperament.

Temperament was measured rather than personality due to the young age of the child

participants. Mothers and adolescents completed the EATO-R, which was based on a measure of temperament for adults created by Derryberry and Rothbart (1988). The EATQ-R is comprised of 8 Temperament and 2 Behavior Scales. The Temperament Scales include:: Activation Control (i.e., having the capacity to complete an action despite a strong tendency to avoid it), Affiliation (i.e., desiring warmth and closeness with others), Attention (i.e., having the capacity to focus and shift attention when necessary), Fear (i.e., having negative affect in anticipation of distress), Frustration (i.e., having negative affect in response to the interruption of an ongoing task), High Intensity Pleasure (i.e., deriving pleasure from activities that are very intense or novel), Inhibitory Control (i.e., having the ability to plan and to suppress inappropriate behaviors, having the ability to control impulses), and Shyness (i.e., showing inhibition to novel or challenging situations, being harm avoidant). The Behavior subscales include: Depressed Mood (i.e., having unpleasant affect and lowered mood) and Aggression (i.e., engaging in direct and indirect hostile and aggressive actions). Scale scores consist of the total scores of the items making up each scale, with higher scores representing more of the measured construct. In addition to these individual scales, a Negative Affect factor can also be calculated using scores on the Frustration, Depressed Mood, and Aggression subscales. Only the Negative Affect Factor and the Inhibitory Control and Shyness scales were included in analyses because they tap constructs (negative affectivity, impulsivity, and harm avoidance) that have been associated with eating pathology in previous studies (Bulik et al., 1995; Leon et al., 1999; Lillenfeld et al., 2000; Pryor & Wiederman, 1996). Mothers reported on the temperament of their adolescent daughters, and adolescents reported on their own temperament.

Research has supported the psychometric properties of the EATQ-R (Capaldi & Rothbart, 1992; Ellis & Rothbart, 2001; Kim, Brody, & Murry, 2003). Specifically, internal reliability of the scales ranged from .67 - .79. With the exception of the child report on the Inhibitory Control scale (alpha = .47), internal consistency reliability was adequate in the current study, with alpha coefficients ranging from .69-.88. Convergent validity between parent and adolescent reports was low to moderate on the original EATQ, with correlations ranging from .2 -.4. However, this rose slightly to .3-.5 on the revised version of the EATQ (Ellis & Rothbart, 2001). In addition, the EATQ subscales were highly correlated with other measures assessing parallel constructs (Capaldi & Rothbart, 1992). When developing the measure, items were dropped if they showed high correlations with more than one scale. The result was that each temperament scale was found to measure a different dimension of temperament, which indicates that discriminant validity was sufficient (Capaldi & Rothbart, 1992).

Statistical Analyses

Preliminary Data Analyses:

Missing data were estimated using the computer software program SYSTAT 10 (Systat Software Inc., 2002). The data appeared to be "missing at random", as missing data were due entirely to computer malfunctions when the computers unexpectedly crashed during assessments. The expectation-maximization algorithm was therefore used to impute values. Pearson product correlations were then conducted to examine initial relationships among the variables. In addition to examining relationships among the mother-adolescent relationship, separation-individuation, and disordered eating variables, intraclass correlations were conducted with twin reports of the mother-adolescent

relationship. These additional correlations were included to explore degree of concordance in twin experience with their mother. Hypotheses were subsequently tested using structural equation modeling (SEM) with Amos5 (Arbuckle, 2003) and maximum likelihood estimation.

Structural Equation Models:

The structural integrity of the latent constructs was established using confirmatory factor analyses before conducting tests of the models. Fit indices that were used to examine model fit include: the Chi-Square test, Comparative Fit Index (CFI), Goodness-of-Fit Index (GFI), and the Root-Mean-Square Error of Approximation (RMSEA).

Model fit was considered good if the following were present: a non-significant chi-square, a CFI greater than .90, a GFI equal to or greater than .90, and a RMSEA less than or equal to .05 (Schumacker & Lomax, 1992). Model fit was considered adequate if a RMSEA less than or equal to .08 was obtained. Adjustments were made to the models to improve model fit based on theory and modification indices. Single-group analyses were conducted to test models examining relationships between the mother-child relationship, separation-individuation, and disordered eating; multiple-group analyses were subsequently conducted to test moderation in these models.

Moderator Analyses

Multiple-group analyses were used to test puberty and temperament as moderators.

Puberty: In order to test puberty as a moderator, maternal and adolescent reports of pubertal development using the PDS were summed and then the average was calculated so that both maternal and adolescent perspectives of the adolescent's pubertal

development were weighted equally. Two groups were formed based on these average scores by dividing participants at the median score. By indicating the midpoint among the scores for the different variables, the median split made it possible to create two groups (i.e., more advanced pubertal development and less advanced pubertal development). If more than two groups had been created (e.g., using quartiles), the sample size of each group would have been too small and would have reduced the power to test the models.

Temperament: The child report of Inhibitory Control on the EATQ exhibited relatively low internal consistency reliability. Consequently, analyses with this variable were conducted with only the maternal report of Inhibitory Control. However, both maternal and child reports were used for the other EATQ scales (i.e., Negative Affect and Shyness).

Two approaches were taken to examine temperament as a moderator in the AN and BN models. First, an <u>overall composite</u> temperament variable was created by summing into one score the following scores: 1) the maternal and adolescent reports of the Shyness and Negative Affect subscales; and 2) the maternal reports of the Inhibitory Control subscale. For the AN model, these scales were scored in the traditional manner, with higher scores being indicative of having more of the temperamental characteristic. By contrast, for the BN model, the Inhibitory Control subscale items were reverse scored before summing, as less Inhibitory Control was predicted to lead to more BN behaviors. Two groups were subsequently formed for each model using median splits on these composite temperament scores. Having higher scores of the summed AN or BN temperament was indicative of having more of the temperamental characteristics that

were predicted to be associated with AN- or BN-like disordered eating. However, the interpretation of the composite temperament score is not completely clear and a second approach was taken to examine scores on temperament variables separately.

The second approach was to split groups based on scores for each temperament subscale separately (i.e., Inhibitory Control, Shyness, and Negative Affect). This second approach was utilized in case differences existed for each subscale that were not captured by examining composite temperament scores only. For the Shyness and Negative Affect subscales, maternal and child reports on each subscale were summed and then the average of each temperament variable was calculated. Two groups were formed for each subscale by dividing at the median. Because only maternal reports were used for the Inhibitory Control scale, two groups were created by dividing maternal scores of the Inhibitory Control subscale at the median. One group represented higher scores of Inhibitory Control and the other group consisted of individuals who reported less Inhibitory Control.

Multiple-Group Model Fit Comparisons: Group differences (i.e., advanced versus early pubertal development; more versus less negative affect) in parameter estimates were tested in all of the moderation models using cross-group equality constraints. The unconstrained (i.e., all parameters allowed to vary across puberty or temperament groups) and constrained (i.e., all parameters constrained to be equal across groups) models were compared by calculating the differences in chi-square values, using the difference in degrees of freedom between the two models as the df for the chi-square test. Significant chi-square differences suggested that the parameters could not be constrained to be equal across groups and thus, that there are differences in relationships

between variables in the model, depending on the level of the moderator. These results would suggest that the moderator of interest (i.e., puberty or temperament) moderates relationships among variables in the model (Kline, 2005).

Power Analyses:

Using a p value of .05, the sample of 202 adolescents and 101 mothers (which resulted in 202 maternal reports total) provided relatively low power to test the significance of the entire model (i.e., power = .29-.38) (MacCullum, Browne, & Sugawara, 1996). Thus, the probability of rejecting a model was low. Further, for the multi-group analyses, the power was even lower (i.e., power = .17-.21). These power estimates are lower than those based on the initial hypothesized model, because the removal of observed variables during the confirmatory factor analyses reduced the number of unique input coefficients. Because the power to test the significance of the entire model is based on the degrees of freedom (i.e., the difference between the amount of unique input information and the number of parameters), removing some unique input information reduced the degrees of freedom and the overall power estimate.

Despite the modest power, this study was the first to examine separation difficulties and eating pathology among adolescents. In addition, based on Kline's guidelines (i.e., 10:1 ratio of number of cases to number of free parameters) (Kline, 2005), the sample size was sufficient (i.e., 10:1 ratio) to provide statistical precision in estimating the significance of direct (e.g., the mother-child relationship predicting separation-individuation, separation-individuation predicting disordered eating) and indirect (e.g., the mediating effects of separation individuation and the moderating effects of puberty and temperament) effects in the model. Indeed, power was adequate to reject

some models during analyses (see SEM analyses below). Therefore, the sample was sufficient for estimating the significance of each path within the model. Moreover, these estimates provided valuable information about relationships between mother-child relationships, separation-individuation, temperament, puberty, and disordered eating that can be replicated and extended in future research examining etiological factors for eating pathology.

RESULTS

Correlations

Pearson product moment correlations showed significant relationships between disordered eating and many of the mother-child relationship and separation-individuation variables (see Table 2). Specifically, all of the disordered eating variables (with the exception of Body Dissatisfaction) showed significant positive associations with adolescent-reported maternal Protection. In addition, adolescent-reported maternal Care showed significant negative relationships with Binge Eating. Further, despite two exceptions, the disordered eating variables showed significant positive relationships with all of the separation-individuation variables, suggesting that more disordered eating was associated with more separation-individuation difficulties. However, the Enmeshment Seeking variable only showed significant positive relationships with Dietary Restraint. In addition, Dependency Denial did not show significant associations with Dietary Restraint, although Dependency Denial showed significant positive relationships with the other disordered eating variables.

Significant relationships also emerged between the mother-child relationship and separation-individuation variables. Overall, the general pattern that emerged was that maternal Care showed significant negative relationships with separation-individuation difficulties, while maternal Protection showed significant positive relationships with separation-individuation difficulties. The exception to this pattern was again the Enmeshment Seeking variable, as maternal Care showed significant positive associations with this aspect of separation-individuation.

Further, intraclass correlations examining concordance within twin pairs revealed significant positive correlations on responses of maternal care and protection (Care = .21*; Protection = .23*), which indicates that there was some similarity in experience by twins with the same mother. Although the correlations were significant, their values were very modest, suggesting that there were differences in twin perceptions of maternal behavior.

Taken together, these correlations preliminarily confirm hypotheses suggesting that disordered eating shows significant positive relationships with maternal overprotection and separation-individuation difficulties, as well as significant negative relationships with maternal care. Further, characteristics of the mother-child relationship (i.e., more maternal overprotection, less maternal Care) show significant positive associations with separation-individuation difficulties. In addition, twins show some similarity in their experiences with their mother, although the correlations are modest which indicates that twins also differ considerably in their experience with their mother. SEM Analyses

Aim 1: Associations between the mother-daughter relationship, separationindividuation difficulties, and AN-like types of disordered eating.

Confirmatory Factor Analyses

A CFA was conducted to test the fit of latent constructs representing separation-individuation and AN-like disordered eating delineated in the original conceptual model (see Figure 1). The attempt to create a latent factor for separation-individuation resulted in very poor fit, as indicated by a significant chi-square statistic and an RMSEA that was greater than $.08 \ (\chi^2(5) = 55.86; p < .000; RMSEA = .23 \ (.17, .28))$. Another attempt to

create a latent factor for separation-individuation was conducted using all of the indicators except for the Enmeshment Seeking variable. This resulted in acceptable fit indices ($\chi^2(2) = .92$; p = .63; RMSEA = .00 (.00, .11)), however the factor loadings were all very low (i.e., .08, .17, .37), therefore this could not be used as a latent factor either.

In an effort to improve model fit, the analyses were re-run using observed rather than latent constructs for the mother-child relationship and separation-individuation. In addition, the number of scales examined was reduced and only included those scales that have been previously linked empirically or theoretically to AN symptoms. These decisions were based on previous research (Johnson, 1991; Marsden, 1997; Smolak & Levine, 1993), which suggested that girls with AN symptoms would have more difficulties with under-separation from their mothers. Thus, the Enmeshment Seeking and Separation Anxiety subscales were identified as appropriate scales to include in the models. Although both of the PASAS subscales have shown correlations with separation-individuation difficulties in adolescence (Hock et al., 2001), the Anxiety about Adolescent Distancing scale was used because it demonstrated much higher internal reliability than the other PASAS subscale (i.e., Comfort with Secure Base Role) and it provided information about the mother's perspective on her daughter's separationindividuation difficulties. The two SITA subscales and one PASAS subscale were subsequently examined as observed variables in the SEM models, with mother and adolescent reports representing separate variables.

The CFA model for AN-like disordered eating contained all of the indicators (i.e., Weight Preoccupation, Body Dissatisfaction, and Dietary Restraint) and resulted in one latent factor. The model was just identified ($\chi^2(0) = 0$) so it was not possible to

determine the model fit. However, the factor loadings were all high and were not substantially different from one another (Standardized Coefficients: AN → Body Dissatisfaction = .73; AN → Weight Preoccupation = .88; AN → Dietary Restraint = .67). Therefore, an AN-like disordered eating latent factor was used in subsequent analyses.

Structural Equation Model

The initial hypothesized AN model (see Figure 2) was tested and resulted in a poor fit to the data (see Table 3). Thus, an error covariance was added between the errors for the SITA subscales (i.e., Enmeshment Seeking and Separation Anxiety) to account for shared method variance. The model fit improved significantly with the addition of this error covariance. The new model (AN – Model with Correlated Errors) demonstrated many significant regression coefficients and factor loadings (see Table 3; Figure 3). However, all of the paths between the mother-child relationship and the Enmeshment Seeking subscale, and between AN-like disordered eating and the Enmeshment Seeking subscale, were non-significant. Therefore, an alternative model (AN – Reduced Model) was developed by removing the Enmeshment Seeking variable from the model. In addition, other non-significant paths between the mother-child relationship and separation-individuation variables were eliminated, leaving only the significant paths between the mother-child relationship and separation-individuation variables (see Table 3; Figure 4).

The coefficients in this model were all significant and their values indicated that more maternal Protection predicted more under-separation, which was predictive of more AN-like disordered eating. This model explained 10.5% of the variance in AN-like

disordered eating. Although both the AN – Model with Correlated Errors and AN – Reduced Model demonstrated adequate model fit, the AN – Reduced Model was more parsimonious and thus, was chosen as best fitting. Unstandardized coefficients and standard errors for the AN – Reduced Model are presented in Table 4.

In order to test for mediation, direct paths between the mother-child relationship variables and AN-like disordered eating were added separately to the AN – Reduced Model and changes in model fit and significance of direct paths were examined. The addition of direct paths to the model resulted in non-significant relationships between the mother-child relationship variables and AN-like disordered eating. Further, the model fit did not improve significantly in either model when direct paths between the mother-child relationship and AN-like disordered eating were added (see Table 3; Figures 5-6). These results provide evidence that separation-individuation fully mediates relationships between the mother-child relationship and AN-like disordered eating. These findings support the hypothesized mediated model and suggest that the relationship between increased maternal protection and AN-like disordered eating is explained by adolescent difficulties with under-separation.

Aim 2: Associations between the mother-daughter relationship, separationindividuation difficulties, and BN-like types of disordered eating.

Confirmatory Factor Analyses

CFA models were conducted to test latent constructs of the mother-child relationship and separation-individuation for the variables in the BN model (see Figure 7). Similar to the AN model, attempts to create latent constructs for the mother-child

relationship and separation-individuation resulted in very poor model fit. For the mother-child relationship, the mother and adolescent reports could not be included in the same latent factor ($\chi^2(2) = 14.81$; p = .001; RMSEA = .18 (.10, .27)), so all of the subscales were represented with separate observed variables. In addition, a latent factor could not be created for separation-individuation ($\chi^2(2) = 68.59$; p = .01; RMSEA = .13 (.05, .22)), so based on previous research and theory (Johnson, 1991; Marsden, 1997; Smolak & Levine, 1993), subscales intended to assess over-separation on the SITA (i.e., Engulfment Anxiety, Dependency Denial) were selected and used as observed variables in the model (see Figure 8). The Anxiety about Adolescent Distancing from the PASAS was also used in the BN model. Although this is the same PASAS scale that was used in the AN model, as noted previously, this PASAS subscale demonstrated higher internal reliability than the other PASAS subscale. In addition, the content of the two PASAS subscales does not discriminate between over-separation and under-separation, which made it possible to use the same PASAS subscale in both the AN and BN models.

Structural Model

The original structural model that was tested resulted in poor fit (see Table 5; Figure 8). Similar to the AN model, an error covariance was added between SITA subscales to account for shared method variance. This resulted in good model fit based on the modification indices (BN – Model with Correlated Errors; see Table 5; Figure 9). However, certain paths in this model were not significant. Therefore, an alternative model was created that eliminated non-significant paths between the mother-child relationship and separation-individuation variables (BN – Reduced Model; see Table 5; Figure 10). Values of the significant parameter estimates suggested that less maternal

Care and more maternal Protection were predictive of more over-separation, which was predictive of more Binge Eating. The finding that maternal Protection <u>positively</u> predicted separation-individuation difficulties and Binge Eating was contrary to expectations that lower levels of maternal Protection would predict separation individuation difficulties. However, it is possible that the <u>combination</u> of overprotective maternal behavior and low maternal empathy results in an overly punitive parent-child relationship that is then associated with BN types of symptoms (see discussion of this possibility in the Discussion section below).

As with the AN models, the BN – Model with Correlated Errors and the BN – Reduced Model both fit the data adequately, but the BN – Reduced Model was more parsimonious and was thus selected as best fitting. Unstandardized coefficients and standard errors for the BN – Reduced Model are presented in Table 6. This model explained 11.9% of the variance in BN-like disordered eating.

To test for mediation, direct paths were added to the model in order to examine direct effects between the mother-child relationship and BN-like disordered eating, as well as changes in model fit indices when the direct paths were added. Separate models with direct paths added between the mother-child relationship variables and BN-like disordered eating did not differ in any meaningful way from the model with the direct paths added all together and this model is presented in Figure 11. One direct relationship between the mother-child relationship and BN-like disordered eating was significant; specifically, less maternal reported maternal protection was predictive of more BN-like disordered eating while the other direct paths were not significant predictors of BN-like disordered eating. This suggests that separation-individuation difficulties partially

mediate relationships between the mother-child relationship and binge eating. However, the finding that <u>less</u> maternal-reported maternal protection was predictive of <u>more</u> binge-eating as well as <u>less</u> over-separation was unexpected. As with the AN model, the model fit did not improve significantly when direct paths between the mother-child relationship and binge-eating were added (see Table 5; Figure 11), which provided support for the partially mediated model. This suggests that over-separation partially explains the relationship between the mother-adolescent relationship and BN-like disordered eating.

<u>Aim 3: Examining the moderating effects of puberty on associations between</u>

Aim 3: Examining the moderating effects of puberty on associations between mother-child relationships, separation-individuation difficulties, and disordered eating.

AN Model

Puberty was initially tested as a moderator for the AN – Reduced Model. There were no significant differences in chi-square values for the unconstrained and constrained models (see Table 7), suggesting that puberty did not moderate relationships between the mother-child relationship, under-separation, and AN-like disordered eating.

However, the multi-group analyses were subsequently re-run with the AN – Model with Correlated Errors, in case information about relationships between the mother-child relationship and separation-individuation were lost by removing the non-significant paths and the Enmeshment Seeking variable from the model. These analyses revealed significant differences in the chi-square values between the unconstrained and constrained models (see Table 7). Additional constraint models were subsequently tested that constrained some relationships to be equal across groups while allowing others to vary freely. The best fitting model using the AN – Model with Correlated Errors allowed

the relationship between the mother-adolescent relationship and separation-individuation variables to be unconstrained, with the rest of the coefficients in the model constrained (see Table 7). The fit of this model was not significantly worse than the fit of the completely unconstrained model, suggesting that the paths between the separation-individuation variables and AN-like disordered eating could be constrained without any loss of information.

Results from this model provided evidence that puberty moderated relationships between the mother-adolescent relationship and separation-individuation, which was consistent with the hypotheses (see Figure 12 for the standardized parameter estimates across puberty groups and Table 8 for the unstandardized parameter estimates and standard errors). These findings also confirm that important information was lost when the Enmeshment Seeking variable was removed from the model. Nonetheless, contrary to expectations, puberty did not moderate relationships between separation-individuation and AN-like disordered eating, as evidenced by significant chi-square differences between the completely unconstrained model and the model that unconstrained relationships between separation-individuation and AN-like disordered eating only (see Table 7).

In the interpretation of results from multiple-group analyses, the magnitude of coefficients across groups cannot be compared due to the different variance in each group (Kline, 2005). Therefore, differences in sign (i.e., positive/negative) and significance for parameter estimates among the groups were examined to determine the moderating effects of puberty. Overall, differences in the sign and significance of parameter estimates provided some support for the hypothesis that stronger relationships between

overprotection and separation-individuation difficulties were present in girls with more advanced pubertal development. Specifically, adolescent-reported maternal Protection was associated with more maternal Anxiety about Adolescent Distancing for the advanced puberty group. By contrast, this estimate was not significant in the early puberty group and actually showed a negative sign. Further, more maternal-reported Protection was associated with less adolescent Enmeshment Seeking in the early puberty group, while this parameter was not significant in the more advanced puberty group. It is possible that more maternal protection predicted less adolescent enmeshment in the early puberty group because maternal protection is more developmentally appropriate for adolescents who are earlier in their development.

BN Model

Puberty was also tested as a moderator in the BN – Reduced Model. These analyses did not provide evidence for differences between earlier and more advanced stages of pubertal development, as there were no significant differences in chi-square values between the unconstrained and constrained models (see Table 9). However, when these analyses were conducted with the BN – Model with Correlated Errors, significant differences were found. The best model, as evidenced by a lack of significant difference in chi-square values between this model and the unconstrained model (see Table 9), included unconstrained relationships between all of the mother-child relationship and separation-individuation variables. Similar to the previous analyses with AN, information appears to have been lost when paths between these variables were removed. These analyses suggest that pubertal development moderated relationships between the mother-child relationship and separation-individuation, but not between separation-

individuation and disordered eating (see Table 10 and Figure 13 for parameter estimates). Thus, similar to analyses of AN, study hypotheses were only partially supported.

Analyses revealed differences in parameter estimates among the puberty groups. In comparing relationships between adolescent and maternal reported <u>Care</u> and separation-individuation variables, there was some evidence that stronger relationships between less Care and over-separation were present in girls with more advanced pubertal development. Specifically, in the more advanced puberty group, less adolescent-reported maternal Care significantly predicted more Dependency Denial, but this relationship was not found to be significant in the early puberty group. In addition, more maternal reported Care was found to predict more Dependency Denial in the early puberty group, which was contrary to theory and not found to be significant in the advanced puberty group.

In comparing relationships between maternal <u>Protection</u> and separation-individuation variables among the two groups, the results were inconsistent and suggested that different aspects of over-separation difficulties appear to be salient in relation to maternal overprotective behavior, based on the level of pubertal development. For example, adolescent-reported maternal Protection was a significant positive predictor of Dependency Denial in the early puberty group but not in the advanced puberty group. In addition, adolescent-reported maternal Protection significantly positively predicted maternal Anxiety about Adolescent Distancing in the advanced puberty group, but not in the early puberty group.

Exploratory Analyses: Age as moderator

It is possible that examining self- or mother-reported puberty as a moderator in analyses may have decreased the ability of the models to detect significant effects. Using a more objective measure of adolescent development, such as age, could yield more robust results. Two new groups (i.e., older and younger) were formed by dividing the group using a median split (median age = 12.5). There was significant overlap in membership between the age and puberty groups, as exhibited by the significant relationship ($\chi^2(1) = 70.45$, p < .001) between membership in each of these groups (see cross-tabulation results in Table 11). Nonetheless, re-running the models with the two age groups yielded slightly different results than analyses conducted with the puberty groups. Specifically, age was not found to moderate any relationships in the AN model, whereas puberty was found to moderate relationships in the AN model (see Appendix table A1). However, results indicated that for the BN model, age moderated relationship between the mother-child relationship and over-separation, as puberty had done (see Appendix tables A2 and A3).

Aim 4: Direct and moderating effects of temperament.

Initially, the direct and moderating effects of temperament were examined within the Correlated Errors and Reduced Models. However, because results were identical across models, only analyses conducted with the AN- and BN - Reduced Models will be discussed.

AN Model

Moderating Effects: As discussed above, temperament was tested as a moderator using both the composite measure and the individual temperament scales. Using the overall composite AN Temperament variable, significant differences emerged in the fit of

the unconstrained and fully constrained models (see Table 12). The best fitting model showed differences in factor loadings of the observed variables comprising the AN-like disordered eating latent variable, rather than differences in predictive relationships among the variables. These differences in the factor loadings suggested that the factor structure was different among the individuals comprising the two temperament groups. However, the factor loadings were all significant and all had positive signs in both groups (see Table 13; Figure 14), and moderation of factor loadings was not of primary interest in this study.

Using the individual temperament subscales in separate models (i.e., high versus low Inhibitory Control, high versus low Shyness, and high versus low Negative Affect), results showed that there were no significant differences in chi-square values of the unconstrained and constrained models for any of the individual temperament subscales (see Table 12). These findings suggest that the parameter estimates can be constrained across the temperament groups, and that there are not significant differences in predictive relationships by temperament type. Temperament therefore did not emerge as a significant moderator in these analyses.

Direct Effects: The overall composite AN temperament variable and individual temperament variables were subsequently added to the AN - Reduced Model separately to test whether they had direct effects on disordered eating (see Table 14). Results showed that the model fit was poor when the overall composite AN Temperament variable was included as a direct effect. When the Shyness variable was examined, the model fit was adequate but the path between Shyness and AN-like Disordered Eating was not significant. Inhibitory Control and Negative Affect each showed direct effects in

Affect predicted more AN-like Disordered Eating (see Table 15; Figure 15). Contrary to expectations, however, <u>more</u> Inhibitory Control was predictive of less AN-like Disordered Eating (see Table 15).

BN Model

Moderating Effects: Temperament was also tested as a moderator in the BN – Reduced Model. The <u>overall composite</u> BN temperament variable and the individual temperament variables were examined separately as moderators in the model. There were not significant chi-square differences in the unconstrained and constrained models for the overall composite BN temperament or the individual temperament subscales (see Table 16). This suggests that the overall composite BN temperament variable and the individual temperament variables did not moderate relationships in the model.

Direct Effects: As with the AN model, temperament was also tested as a direct effect in the BN model (see Table 17). Results showed that the model fit was poor when the overall composite BN Temperament or Negative Affect variables were added separately as direct effects. Although the model fit the data adequately when Shyness and Inhibitory Control were added separately as direct effects, the Shyness and Inhibitory Control parameter estimates were not significant (see Table 18).

Aim 5: Effects of puberty and temperament as moderators in the model.

Puberty appeared to be a stronger moderator than temperament in the AN and BN models. For the AN and BN models, puberty moderated relationships between the maternal-adolescent relationship and separation-individuation, while temperament did not moderate any predictive relationships among the variables.

DISCUSSION

The main goal of this study was to examine relationships between separation-individuation and disordered eating during adolescence. These relationships had been described theoretically but had not been examined empirically. Hypotheses proposing that specific dynamics within the mother-child relationship would predict more separation-individuation difficulties and more AN- and BN-like disordered eating, were generally supported. Specifically, more maternal overprotection predicted more adolescent under-separation which, in turn, predicted AN-like symptoms. By contrast, overprotective maternal behavior and less maternal care predicted more adolescent overseparation, which was associated with BN-like symptoms. These findings support theories suggesting that characteristics of the mother-adolescent relationship are associated with an adolescent's ability to separate and individuate, which is in turn associated with eating pathology.

Additional aims were to explore the effects of other variables (i.e., pubertal development, temperament) on relationships among the variables. Puberty was found to moderate relationships between the mother-child relationship and separation-individuation, such that more advanced pubertal development was associated with stronger relationships between mother-child relationship variables (i.e., more maternal overprotection and less care) and adolescent under/over separation. However, temperament was not found to moderate any predictive relationships among the variables in the models.

Importantly, this study improved upon previous research in a number of ways. First, it examined the joint influence of the mother-child relationship and separation-individuation difficulties on disordered eating, in one integrative model. Second, adolescent rather than adult participants were examined, which allowed for the investigation of separation-individuation difficulties during the salient developmental period when these difficulties are theorized to contribute to eating disorders. Third, both maternal and child reports were obtained. Consistent with previous research, relatively low correlations were found between maternal and adolescent reports in the current study and thus, they were represented by separate constructs in analyses. Finally, relationships with different types of eating pathology (AN- and BN-like symptoms) were examined in separate models, resulting in the identification of important differences in predictive relationships between mother-child relationships, separation-individuation difficulties, and disordered eating.

Relationships among the Mother-Child Relationship, Separation-Individuation, and Disordered Eating

AN-Like Disordered Eating

Results supported the hypothesized mediated model, where the influence of maternal overprotection on AN types of disordered eating was found to be mediated through adolescent under-separation. These associations were found for Body Dissatisfaction, Weight Preoccupation, and Dietary Restraint, which were the three variables comprising the AN-like disordered eating latent variable. These results generally support previous research conducted with adults that found relationships

between separation-individuation difficulties and AN symptoms (Friedlander & Siegel, 1990; Marsden, Meyer, Fuller, & Waller, 2002; Rhodes & Kroger, 1992). However, these findings extend previous research by examining these constructs during adolescence, which is the developmental period when they are salient, and by investigating the extent to which parenting influences under-separation and AN-like disordered eating. Taken together, results from this study suggest that adolescents with mothers who are overprotective have more anxiety about separating and are more likely to exhibit AN-like symptoms than adolescents without these characteristics.

Our results are broadly consistent with theories described previously which link parenting behaviors, individuation, and AN-types of disordered eating (Bruch, 1974; Goldstein, 1981; Garfinkle et al., 1983; Palazzoli, 1974). These theories postulate that overprotective maternal behavior may hinder an adolescent's efforts to individuate by encouraging an adolescent to stay dependent on parents, rather than helping the adolescent develop a sense of independence and separateness from the family. Difficulty becoming independent during adolescence is then postulated to lead to decreased separation from the family and increased levels of AN-like symptoms.

Surprisingly, in our study, the Enmeshment Seeking scale (which most closely approximates the processes described above) did not significantly predict AN-like disordered eating. Previous empirical research has found relationships between enmeshment and AN types of symptoms (Friedlander & Siegel, 1990; Marsden, Meyer, Fuller, & Waller, 2002; Rhodes & Kroger, 1992), however these studies focused on enmeshment within the mother-child relationship, while the SITA Enmeshment Seeking scale examines the extent to which the adolescent becomes enmeshed in relationships in

general. Although these constructs are likely related, their differences may explain why significant effects were not found for Enmeshment Seeking in this study. Rhodes and Kroger (1992) used the SITA with a late adolescent sample (mean age = 19) and did not find the Enmeshment Seeking scale to be predictive of disordered eating. Results from their study and the current study suggest that this scale may not capture the enmeshment that has been commonly associated with eating pathology. Enmeshment within the mother-child relationship, rather than enmeshment across relationships in general, may be most related to AN-like disordered eating. Additional research comparing findings from the SITA and other measures of enmeshment will clarify the role of enmeshment in AN.

It will also be important to identify the specific processes accounting for relationships between under-separation and AN disordered eating. Theory postulates that adolescents who are conflicted about separation-individuation may develop AN symptoms in an attempt to avoid the normal developmental process of separation from the mother and family (Johnson, 1991; Marsden, 1997). In essence, the AN symptoms may function as an attempt to keep the adolescent in a more childlike physical state that will delay adolescence and thus, delay the need to separate from parents. However, research using instruments that could access some of these underlying motivations and internal processes is needed to confirm this theory. Moreover, future research should investigate other potential reasons for the maternal overprotection/adolescent under-separation relationship. For example, it is possible that maternal overprotection and adolescent under-separation may create a general vulnerability to psychopathology that manifests as AN symptoms for some girls, depending on the presence of other risk factors (e.g., genetic predisposition for eating disorders, teasing about body

shape/weight). In girls without these additional risk factors, maternal protection and adolescent under-separation may manifest as other forms of psychopathology that are also prevalent in girls during adolescence (e.g., depression or anxiety).

Nonetheless, it is important to note that the variables in the AN model explained only a small percentage of the variance in AN-like disordered eating (10.5%). Therefore, the mother-child relationship and separation-individuation difficulties may represent small but significant factors that are associated with disordered eating symptoms. Incorporating other risk factors discussed previously (e.g., genetic factors, depression, anxiety) into the models is likely to provide a more complete picture of the development of AN-like disordered eating during adolescence.

BN-Like Disordered Eating

Results also supported the mediated model for BN-like disordered eating; specifically, over-separation partially explained the relationship between the mother-child relationship (i.e., low maternal Care and high maternal Protection) and binge eating. These results confirm previous research conducted with adults that found relationships between difficulties with separation-individuation and BN (Friedlander & Siegel, 1990; Marsden, Meyer, Fuller, & Waller, 2002; Rhodes & Kroger, 1992; Smolak & Levine, 1994). Results significantly extend this research by showing that associations between over-separation and BN-like disordered eating are influenced by the mother-child relationship during the critical adolescent time period.

As predicted, less maternal Care was predictive of more over-separation. Several processes could potentially account for this finding. For example, it is possible that girls

who experience less maternal care may fear that they will not receive the care they need and so instead, they may withdraw from their mothers and over-separate in an attempt to take care of themselves. Alternatively, less maternal care may be indicative of a more chaotic family environment that may then lead to a desire to separate from the family. In essence, the adolescent may seek separation from a chaotic family environment in an attempt to gain or maintain internal stability. Research somewhat supports the second explanation; families who have a daughter with BN have been shown to exhibit less maternal care and support and more disorganization (Hedlund, Fichter, Quadflieg, & Brandl, 2003; Johnson & Flach, 1985). More research is needed to understand both of these processes in greater depth as well as their potential for explaining the relationship between less maternal care and adolescent over-separation.

Contrary to hypotheses, <u>more</u> maternal Protection predicted more separation-individuation difficulties in the BN model. This study's hypotheses were based on previous research that generally found parents of women with BN to <u>be less involved</u> and more unstable than parents in control families (Berg, 2000; Humphrey, 1989; Johnson & Flack, 1985; Kog & Vandereycken, 1989). However, one possible influencing factor is that both maternal and child reports were obtained in this study, while previous studies did not include maternal reports. It is possible that mothers may report more involvement in their children's lives than children report, which may lead to higher Protection scores than has been found in previous work. Indeed, in a post-hoc analysis, a paired t-test showed that there were significant differences between mother (M = 31.65; SD = 3.78) and daughter (M = 29.55; SD = 5.53) Protection scores, with mothers reporting significantly more protection than daughters (t(200) = -5.24; p = <.001). This may

explain why maternal overprotection was found to be associated with over-separation and BN symptoms in this study, but not in other studies.

However, maternal-reported maternal Protection showed a significant negative relationship with binge-eating when that direct effect was added to the model. It appears that for <u>maternal reports</u> of maternal protection, less protection has some direct effects on binge-eating symptoms that are not explained by the influence of maternal protection on separation-individuation difficulties. Alternatively, relationships between adolescent reports of maternal protection and binge-eating were fully explained by their influence on separation-individuation difficulties. These findings suggest that the adolescent experience of maternal behavior is influential in the development of binge eating through its impact on the adolescent separation-individuation process, whereas the maternal perspective of maternal behavior influences adolescent binge eating directly as well as indirectly through separation-individuation. Therefore, separate and distinct family processes may be present for adolescents and mothers that influence adolescent binge eating, although further research is necessary to confirm this. This study's discrepancies between mother and daughter reports of maternal protection also highlight the importance of including both mother and child reports in order to understand their differential and joint effects.

Further, whereas <u>more</u> maternal and adolescent reported maternal protection was predictive of more over-separation and more binge-eating, <u>less</u> maternal-reported maternal protection was predictive of more binge-eating. It is possible that maternal-reported maternal protection has some mediated effects through over-separation that are different than its direct effects. However, it is also possible that when the PBI was

adjusted in this study to obtain maternal reports, the psychometric properties of the measure were affected. Indeed, the alpha reliability for maternal reported maternal protection was low (.68), and this may have affected the results. Therefore, the validity and reliability of the PBI for mothers may not be as strong, leading to this unusual finding.

Nonetheless, the identification in this study of adolescent-reported maternal overprotection as a correlate of BN symptoms is interesting and suggests that problematic parent-child relationships for these symptoms might be marked by the combination of low maternal Care with high maternal Protection. This pattern has been previously termed "affectionless control" (Parker, 1983). Children who experience this type of parenting have been theorized to experience their parents as controlling, but not in a way that is directed towards ensuring their welfare. Rather, the controlling parental behavior is experienced as a way for the parents to assert control over their child to satisfy the parents' own needs f authority and power. This parenting style is different from one characterized by high protection and high care, where the parents may be experienced as controlling, but the parents are also viewed as having the child's best interests at heart (Parker, 1983).

Importantly, this "affectionless control" parenting style has been associated with other types of psychopathology in children; specifically, it has been found to be prevalent among children with depression (Parker, 1983; Patton et al., 2001; Pilowsky, Wickramaratne, Nomura, & Weissman, 2006). The specific link between this type of control and BN remains to be elucidated. However, the two dimensions of affectionless control (i.e., high control and low affection) comprising this parenting style appear to be

related to several types of bulimic symptoms. Specifically, binge eating may develop as a way for the adolescent to take control of her life, retaliating against parental control, by making her own decisions about what food to eat and expel. Indeed, research indicates that individuals with BN feel like many areas of their life are out of their control and that binging and purging can function to gain control back (Kayrooz, 1995). In addition, experiencing less maternal affection may lead individuals to feel like their needs are not being met. Individuals at risk for BN may cope with this feeling by filling themselves with food during binges. More research is needed to examine these possibilities and understand the processes underlying the link between parent-child relationships and BN.

Interestingly, in terms of separation individuation variables, Dependency Denial was the significant predictor of binge eating. Both of the other separation-individuation variables, Engulfment Anxiety and maternal Anxiety about Adolescent Distancing, only showed a trend towards being positive predictors of binge eating. Dependency Denial may have emerged as the most significant predictor because it has the clearest theoretical connection to BN symptoms. Possibly related to experiencing the "affectionless control" parenting style described above, individuals with high scores on Dependency Denial do not allow themselves to trust and depend on others, and this leaves them with needs that are not met. Having a need for closeness with others that is not filled may cause them to fill themselves up with food during binges. The Engulfment Anxiety subscale assesses the fear of losing one's independence by having close interpersonal relationships, while maternal Anxiety about Adolescent Distancing assesses the mother's discomfort with her daughter's growing independence. While both of these scales may be related to adolescent over-separation, they may be less likely than Dependency Denial to manifest

in BN types of symptoms because their content is less related to the experience of being unable to rely on others for care and support that may be influential in the development of BN symptoms.

Importantly, as with the AN model, it is also the case with BN that characteristics of the mother-child relationship and over-separation may increase vulnerability to psychopathology in general, and that other risk factors may account for the development of BN type pathology versus other types of psychopathology. Indeed, similar to the AN model, only a small percentage of the total variance in BN types of pathology was explained (11.9 %). The inclusion of other risk factors (i.e., genetic factors, depression, anxiety) in future models of BN may result in a more comprehensive picture of the development of BN during adolescence.

Moderators

Effects of Puberty, Age, Temperament

Overall, findings for the moderators indicated that during adolescence, puberty is more influential in the development of separation-individuation difficulties than are age or temperament. Age did show some indirect effects on separation-individuation in the BN model, but not in the AN model. Further, temperament does appear to have more general, direct effects on AN (but not BN) types of eating pathology, supporting previous research showing that temperament is a strong predictor of AN regardless of developmental period (Bloks, Hoek, Callewaert, & van Furth, 2004; Keel, Fulkerson, & Leon, 1997; Wonderlich, Connolly, & Stice, 2004).

Puberty: For both the AN and BN models, mother-child relationship difficulties were more predictive of separation-individuation difficulties in girls with more advanced pubertal development. These findings support study hypotheses as well as previous theory and research showing that more advanced pubertal development may increase parental overprotective behavior during a time when the adolescent feels more of a need to individuate (Steinberg, 1987). In essence, the changes associated with pubertal development may lead mothers to become more overprotective as they sense the impending separation of their adolescent and/or worry about risky behaviors that their daughters may begin to engage in. Pubertal development could therefore intensify conflictual mother-child relationships and increase difficulties with separationindividuation for the adolescent. Adolescents with AN-like pathology may experience even more anxiety about separating as they advance in puberty and their mothers become more overprotective. This appears to result in more under-separation for the adolescent that is then associated with more AN symptoms. Alternatively, adolescents with BN-like pathology may increase their need to distance themselves from their family in an extreme way as their pubertal development increases and strains the mother-child relationship.

Surprisingly, puberty did not moderate relationships between separation-individuation and AN- or BN-like disordered eating. This suggests that the relationship between separation-individuation difficulties and disordered eating may not be affected by differences in pubertal status; instead, puberty may only influence the development of disordered eating through affecting associations between parenting and separation-individuation difficulties. These findings are significant in indicating that some of the effects of puberty on eating pathology are indirect, influencing the processes that underlie

the development of disordered eating rather than directly influencing disordered eating symptoms.

Age: In the exploratory analyses including age as a moderator, significant findings emerged in the BN model suggesting that age, like puberty, moderated relationships between the mother-child relationship and separation-individuation difficulties. Being older seemed to strengthen relationships between maternal overprotection and less care and over-separation, although this was not found for relationships among all variables. However, age was not found to moderate predictive relationships in the AN model. It is unclear why age and puberty did not show similar effects in the AN model. It is possible that for these types of mother-child relationship dynamics and separation-individuation difficulties, seeing the actual bodily changes of puberty is more influential in exacerbating conflictual mother-child relationships than changes in age. According to theory (Marsden, 1997; O'Kearney, 1996), individuals who develop AN symptoms may be trying to slow the physical changes associated with puberty to prevent separation and individuation. Therefore, when these pubertal changes occur, they may have more psychological influence on the development of adolescent under-separation than age alone. Further, observing physical changes in their daughters may influence mothers to feel more of a pull to keep them close. This may lead to more overprotective maternal behavior which may then increase the adolescent's efforts to avoid separation and lead to AN symptoms. Importantly, all of these hypotheses are speculative, as more research is needed to confirm that there are differences in the moderating effects of age and puberty and to examine the processes underlying the differential effects.

Temperament: Temperament did not show any moderating effects on the variables in the AN or BN models, which suggests that it does not influence relationships between the mother-child relationship, separation-individuation difficulties, and AN- and BN- like disordered eating. It is unclear why these moderating effects were not observed, as it would seem likely that individuals who have more negative affect or impulsivity would be more likely to develop disordered eating symptoms if they were experiencing separation-individuation difficulties. It may be that difficulties with separationindividuation are difficult for anyone, regardless of their temperament, thus temperament does not make it more likely that disordered eating would develop. It is also possible that temperament influences relationships between separation-individuation difficulties and forms of psychopathology other than eating pathology (e.g., depression, anxiety). Since no previous studies have examined the extent to which temperament moderates relationships between separation-individuation difficulties and eating pathology, it is difficult to determine whether temperament does not moderate these relationships or whether its moderating effects did not emerge in results, possibly for methodological reasons. Future studies are needed to clarify the role of temperament as a moderator of relationships between separation-individuation and eating pathology.

Nonetheless, temperament did show some direct effects on AN-like disordered eating, although no direct effects were observed for BN symptoms. The direct effects of temperament on AN-like disordered eating included positive associations between negative affect, impulsivity, and disordered eating. These findings are consistent with previous research (e.g., Klump et al., 2000) that has shown that adolescents who experience more negative affect tend to be anxious and dysphoric and are more likely to

display AN-like disordered eating symptoms. However, contrary to expectations, more impulsivity (i.e., low Inhibitory Control) was predictive of more AN-like disordered eating. These findings conflict with research which has found that impulsivity is more strongly associated with BN than AN (Bulik, Sullivan, Weltzin, & Kaye, 1995; Casper, Hedeker, & McClough, 1992; Fassino et al., 2002; Klump et al., 2003; Lilenfeld et al., 2000; Tomotake & Ohmori, 2002).

One possible explanation for the discrepant findings is that the use of only maternal reports of Inhibitory Control affected the results. As noted previously, there are low correlations between maternal and child reports of adolescent temperament and thus, it is possible that different results would have been obtained had we had a reliable measure of child reported Inhibitory Control. Specifically, findings may have conformed more closely to previous results had we used child reports, as previous studies examining impulsivity and eating pathology have not included parent reports.

Another way that this study differed methodologically from many other studies of impulsivity and eating pathology is that an adolescent population was used in this study, rather than an adult population. It is possible that impulsivity is related to AN and BN symptoms differently during adolescence and adulthood. Adolescents have been found to be more impulsive than adults (Vaidya, Grippo, Johnson, & Watson, 2004), thus this study's sample may be more impulsive overall than adult samples, which may have led to findings that differ from other studies. However, the extent to which impulsivity is associated with AN and whether impulsivity shows different relationships in AN and BN in adolescence versus adulthood awaits further research.

Moreover, the lack of direct effects of temperament on BN is contrary to findings of previous research (Bulik, Sullivan, Weltzin, & Kaye, 1995; Casper, Hedeker, & McClough, 1992; Fassino et al., 2002; Klump et al., 2003; Lilenfeld et al., 2000; Tomotake & Ohmori, 2002). It was particularly surprising that no direct effects were found for impulsivity and BN, whereas they were found between impulsivity and AN. Although it is possible that temperament influences AN more than BN, it is also possible that some of the methodological issues described previously (i.e., use of mother reports only or mother and child, use of adolescent versus adult populations) could also account for the unusual results regarding temperament and BN. Specifically, the use of both maternal and child reports of temperament rather than only child reports as well as using adolescent rather than adult participants was different than other studies that have examined links between temperament and eating pathology.

In addition, the use of the EATQ may have impacted results in this study. Indeed, in an adolescent population, Wonderlich and colleagues (2004) found that impulsivity measured with traditional temperament or personality scales was not associated with BN symptoms; however, behavioral constructs associated with impulsivity (e.g., delinquency) did significantly predict BN symptoms. Wonderlich and colleagues (2004) suggest that generic measures of trait impulsivity may not be specific enough to disordered eating behaviors, in an adolescent population. Therefore, the use of the EATQ in this study may have contributed to the general lack of direct effects that were found for BN, as there is some indication that more sensitive indicators of temperament and personality constructs may be important to use when examining relationships between temperament and eating pathology in an adolescent population (Wonderlich et al., 2004).

Given these methodological assessment issues, more research that compares effects across different measures is necessary to examine the extent to which temperament is associated with BN symptoms.

Another possible reason for differences between this study's findings and previous research on personality and eating pathology is that the mother's expectations about her child's temperament (which we did not measure) influenced results. Lerner's goodness-of-fit model of person-context interaction (1983) emphasizes the importance of the relationship between an individual's personality and the corresponding characteristics of significant others (e.g., mothers) who constitute their social context. Lerner (1983) suggests that at a certain point in development, the most important predictor of adaptive functioning is the match between the children's attributes and the social context, including parental expectations of their child's temperament. Thus, it is possible that if maternal expectations for her adolescent's temperament had also been assessed, the "goodness of fit" between the mother's general expectations about temperament and the mother's assessment of the adolescent's temperament could have emerged as more significant predictors of disordered eating than the temperament variables examined. It is possible that in adolescence, the "goodness of fit" might be a better predictor of both AN and BN, which may partially account for the unusual findings regarding temperament and disordered eating in this study. This possibility is purely speculative, and thus, additional research is needed to investigate the influence of the "goodness of fit" of temperament on separation-individuation and disordered eating.

Finally, temperamental characteristics are believed to show some stability across time and setting. However, the magnitude of the stability is modest when single time

points are considered (Seifer, 2000). Therefore the use of one method of assessing temperament at one time point may have affected this study's results and offers another possible way to understand the unusual findings with temperament in this study. Specifically, temperament could have been a measure of the state of the adolescent when completing the assessment materials that could have fluctuated throughout the day, which may have affected the stability of the temperament results. This possibility is speculative and more research is needed, assessing temperament more comprehensively (i.e., state and trait aspects) to determine the direct and indirect effects of temperament on eating pathology.

Limitations and Future Directions

It is important to note several limitations of this study. First, due to the relatively small sample size, the power was low to test model fit and thus, there was a low probability of rejecting a false model. However, this was the first study to examine relationships between separation-individuation and eating pathology among adolescents and thus, it was an important first step in understanding significant factors that may influence the rise in disordered eating behavior during adolescence. Future research should attempt to replicate the results of this study in a larger sample.

Second, the sample was predominantly Caucasian and representative of the middle- to upper-level socioeconomic groups. Research shows that eating disorders are more prevalent in Caucasians of middle-to-upper socioeconomic status (Dolan, 1991; Striegel-Moore et al., 2003), although some studies find that eating disorders are becoming more prevalent among minority populations and those of lower socioeconomic

status (le Grange, Telch, & Tibbs, 1998; Pernick et al., 2006; Toro et al., 2006; Wassenaar et al., 2000). Therefore, future research should examine whether our results generalize to more diverse samples.

Third, although one of this study's strengths was its use of reports from multiple informants, there were no reports obtained from fathers. Research has increasingly provided support for the importance of the father-daughter relationship in the development of eating pathology (Wonderlich, Ukestad, & Perzacki, 1994). Therefore, future research should include paternal reports and measures of the father-daughter relationship in order to investigate its relationship with separation-individuation and disordered eating.

Fourth, this study did not use a clinical sample, and thus, it is unclear whether the relationships observed would extend to individuals with clinical eating disorders. It is possible that the relationships would be stronger in clinical populations, due to the greater severity of disordered eating. However future research is needed to examine this question. Fifth, twins were used as a convenience sample in this study. Future research should attempt to replicate the results of this study with singletons to ensure that the findings of the current study were not affected by the use of twins.

Sixth, attempts in this study to differentiate between bulimic symptoms and restrictor AN may not have been wholly successful. The variables comprising the AN-like disordered eating latent factor in this study (i.e., weight preoccupation, body dissatisfaction, and dietary restraint) are also present in BN individuals. Because the data for both models were from the same sample, individuals who reported high levels of binge eating may have also reported high levels of the variables making up the AN latent

factor. However, because there were different associations between the mother-child relationship and separation-individuation difficulties (e.g., less care and more protection predicting more over-separation and more protection predicting more under-separation) in the AN and BN models, there were also likely individuals in this sample with high scores on AN-like disordered eating symptoms (e.g., high weight preoccupation, body dissatisfaction, and dietary restraint scores) who did not report high scores on binge eating. Confirming this hypothesis, a post-hoc confirmatory factor analysis was conducted to examine one disordered eating latent variable comprised of body dissatisfaction, weight preoccupation, dietary restraint, and binge eating. Results showed that the latent factor did not fit well ($\chi^2(2) = 9.36$; p = .01; RMSEA = .14 (.06, .23), and that binge eating had the lowest factor loading among the variables. These results provide some evidence that binge eating is a different type of eating pathology than the variables comprising the AN-like disordered eating factor.

Finally, because of the cross-sectional design of this study, it was not possible to demonstrate direction of effects among the variables. Although this study was important in providing empirical support for theory that difficulties with the maternal-child relationship and separation-individuation difficulties are associated with disordered eating in adolescence, future research should examine the extent to which parent-child relationship variables cause separation-individuation difficulties, which subsequently cause disordered eating. In addition, it is possible that child behavior could lead to mother-child conflict, rather than maternal behavior driving conflict in the mother-child relationship. For example, a child who consistently pushes limits set by parents may lead mothers to behave in an overly protective manner, in an effort to be firm and protect the

child from harm. Because of the cross-sectional design of this study, it is unclear whether the mother's over-protective behavior leads to or is the result of the child's behavior. Analyses revealed that twin experiences with their mother were positively correlated with their co-twin's experience therefore there was some similarity across children with the same mother. However, the concordance between twin reports on their mother's behavior was not perfect, and variation is likely related to differences in the child's behavior and corresponding response by the mother. Therefore, future research is needed to further disentangle how much of the mother-child interaction is explained by parent versus child behavior.

Further, although the foundations of this study were based on the work of Mahler (1975) which focused on infancy, this study focused on adolescence. Future research should determine whether the early infant-parent interactions described by Mahler (1975) predict separation-individuation difficulties during adolescence and lead to eating pathology. Although a longitudinal study of this scope would be difficult to conduct, it is imperative for understanding whether early maternal-child relationships and feeding patterns are related to the later development of eating pathology. As an alternative approach, maternal reports of infant feeding behavior could be obtained retrospectively and related to separation-individuation and eating difficulties in adolescence. Although the validity of these reports remains a question (see Finkel & McGue, 1993), they would provide a good first step towards examining whether parenting behaviors and early feeding behaviors are associated with the later development of eating pathology.

Conclusions and Treatment Implications

This study provides initial support for theories (Johnson, 1991; Marsden, 1997; Newton, 2005; O'Kearney, 1996) which postulate that difficulties in the mother-child relationship lead to separation-individuation difficulties during adolescence which, in turn increase risk for disordered eating. Results extend these theories by showing that pubertal development moderates relationships between the mother-child relationship and separation-individuation.

This study's findings could help explain the increase in disordered eating behavior during adolescence and thus contribute to research examining the etiology of eating disorders. However, longitudinal research is necessary to confirm the status of these variables (i.e., separation-individuation, the mother-child relationship, puberty) as risk factors for the development of eating pathology. This longitudinal research could examine the variables included in this study as well as variables that were not, such as genetic factors and other forms of psychopathology (e.g., anxiety, depression). This type of prospective research could help demonstrate causality among the variables as well as provide information about which variables are most influential in the development of disordered eating during adolescence. Further, this research could attempt to assess interrelationships among the variables, to elucidate the underlying mechanisms that lead to eating pathology.

The results of this study also have implications for treatment and prevention efforts. School and community prevention programs that target adolescence could be enhanced by incorporating results from this study that highlight the importance of the mother-adolescent relationship and separation-individuation. Specifically, existing prevention programs could include education for parents and their adolescent daughters

about how difficulties becoming independent could put an adolescent at risk for developing an eating disorder. Parents and their adolescent daughters could be encouraged to discuss their views on their relationship and the process of the adolescent becoming independent. By encouraging expression of these issues within the family, the adolescent may feel supported in the task of becoming independent or of becoming interdependent (i.e., being able to tolerate needing to depend on family members) and less likely to internalize conflicted feelings about the separation-individuation process so that they do not manifest in eating pathology. Therefore, helping families at risk to become aware of the influence that family relationships and separation-individuation difficulties can have on the development of eating disorders during adolescence may aid in preventing their occurrence. Adding this component to existing programs could make them more effective in preventing the development of eating pathology.

This study also has implications for the treatment of individuals with eating disorders. Specifically, the efficacy of existing family treatment programs (e.g., le Grange, Lock, & Dymek, 2003; Lock & le Grange, 2005; Lock, le Grange, Agras, & Dare, 2001) might be enhanced if interventions focus on parent-child conflict around separation-individuation difficulties. The family treatment programs that have been found to be most effective for treating eating disorders have been based on the Maudsley method (Lock et al., 2001). The Maudsley approach to treating girls with eating disorders emphasizes the inclusion of the family in treatment. The adolescent is viewed as being "regressed" at a younger age and needing the parents to be involved and to provide help in taking care of his/her needs. Further, this approach advocates that parents

must increase their control over the adolescent's eating and must be involved in the refeeding process of the child (Lock et al., 2001).

The principles of the Maudsley approach may appear to conflict with this study's findings; specifically, this study suggests that parental overprotective behavior prevents the adolescent's autonomy which leads to more disordered eating, while the Maudsley approach seems to recommend increasing the child's dependence on the parent. However, also crucial to the Maudsley approach is that parents are encouraged to be involved in the treatment in a respectful way, with regard for the adolescent's point of view and experience. The parents are seen as needing to assist their adolescent with the developmental task of autonomy – one way to do so is to temporarily be in charge of the adolescent to help reduce the hold of the eating disorder on the child's life, until the adolescent is back to a more normal weight and eating routine. There is also attention towards general family dynamics, as well as teaching parents about how to be empathic towards their children and to promote tasks of adolescence. Therefore, this study's findings are actually more congruent with the Maudsley family treatment approach than they appear to be. Parents are assisted in becoming empathic and caring to help their child establish healthy weight and eating routines, and are subsequently encouraged and taught skills to help their children with the developmental process of adolescence. The process of teaching parents to be caring and directive with their adolescents initially, and then to learn to respect their adolescent's growing autonomy is consistent with implications from this study.

In conclusion, results from this study provide the first empirical support for theories (Johnson, 1991; Marsden, 1997; Newton, 2005; O'Kearney, 1996) which

describe relationships among the mother-child relationship, separation-individuation difficulties, puberty, and eating pathology during adolescence. These findings contribute to the understanding of factors associated with the development of eating disorders during adolescence and this information can be used in the prevention and treatment of eating disorders.

FIGURES

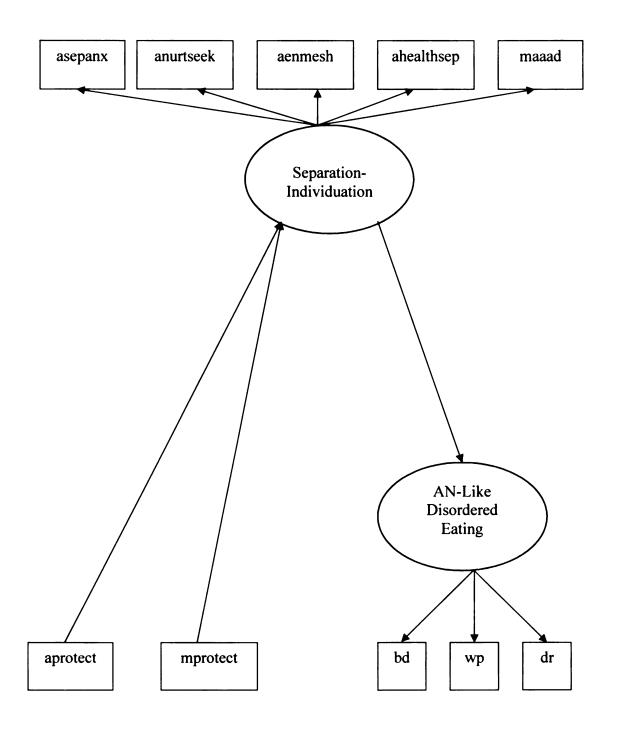


Figure 1. Conceptual and structural model of relationships between the mother-child relationship, separation-individuation, and AN-like disordered eating

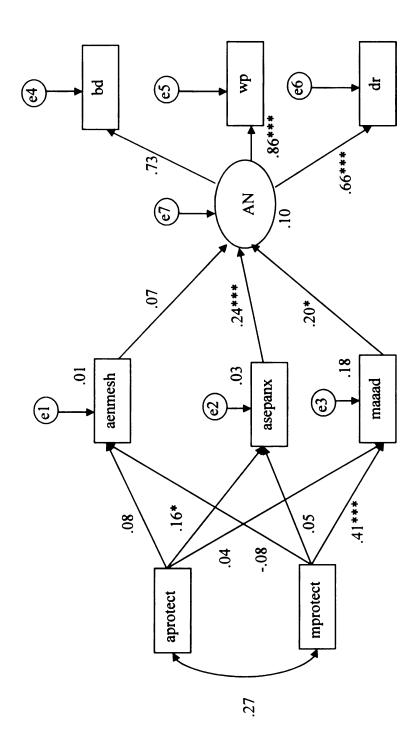


Figure 2. Latent Variable Structural Equation Model of AN - Original Model (includes all paths and variables).

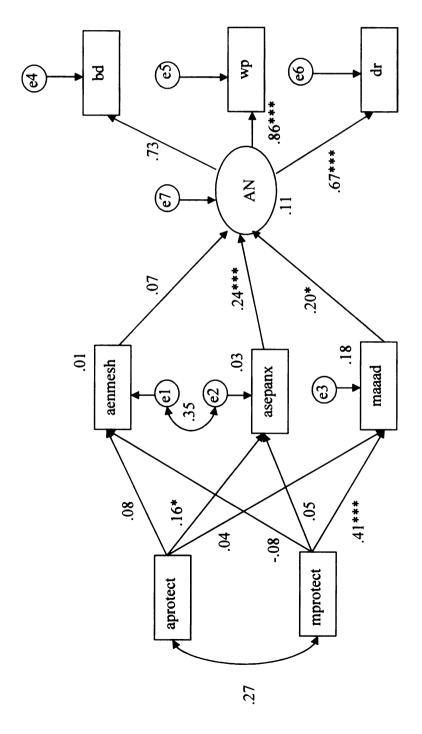


Figure 3. Latent Variable Structural Equation Model of AN - Model with Correlated Errors (i.e., e1 and e2).

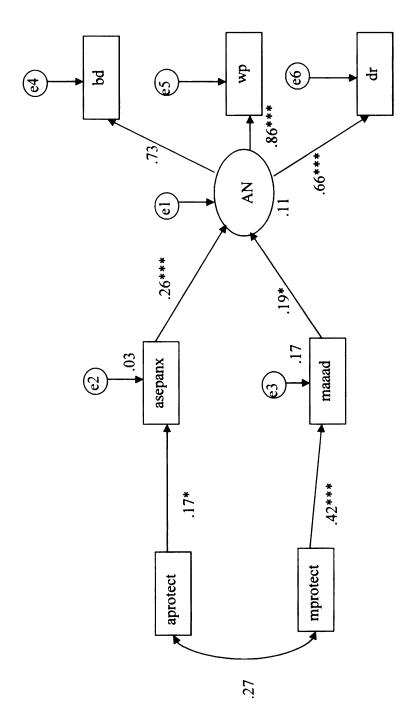


Figure 4. Latent Variable Structural Equation Model of AN- Reduced Model (without aenmesh (Enmeshment Seeking) and nonsignificant paths between mother-child relationship and separation-individuation variables).

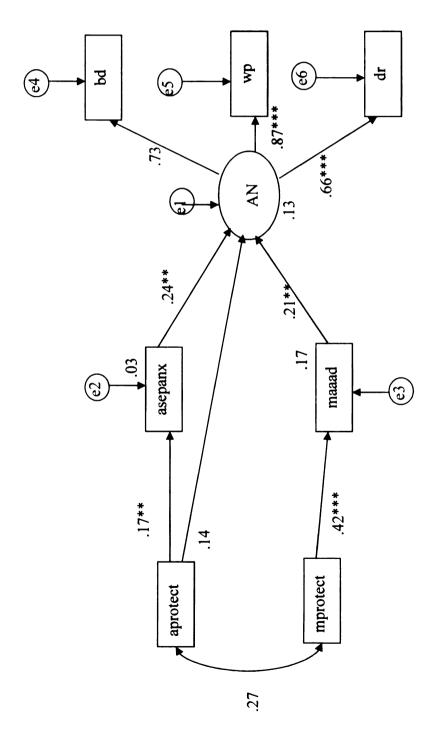


Figure 5. AN-Reduced Model with direct effect between adolescent reported maternal protection and AN-like disordered eating

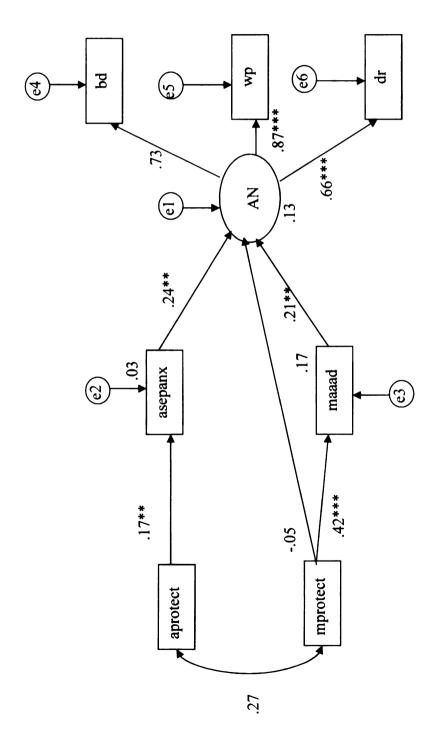


Figure 6. AN-Reduced Model with direct effects between maternal reported maternal protection and AN-like disordered eating

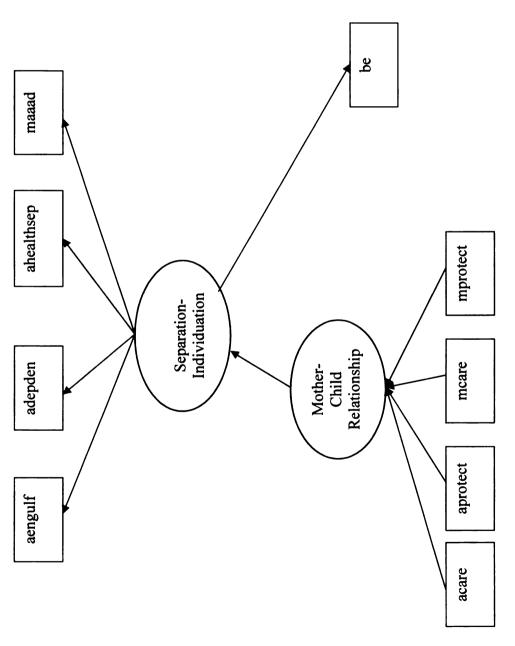


Figure 7. Conceptual and structural model of relationships between the mother-child, relationship, separation-individuation, and BN-like disordered eating.

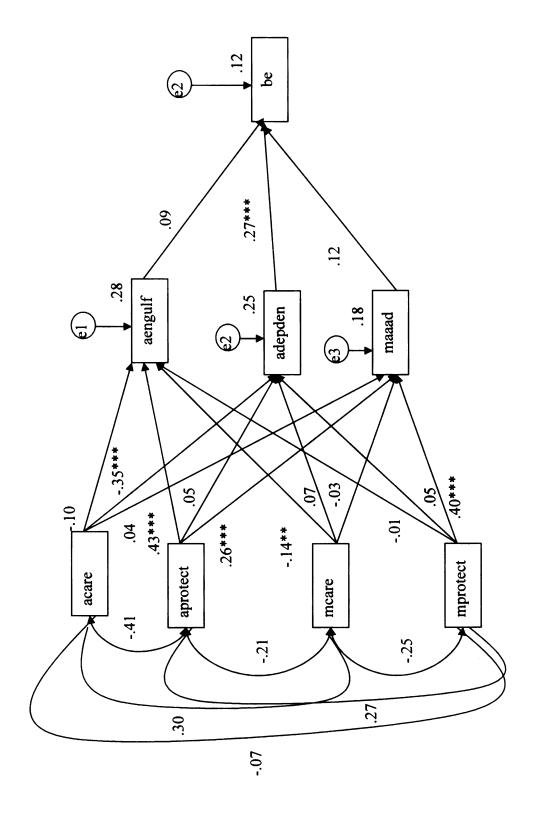


Figure 8. Structural model of BN - Original Model (includes all paths and variables).

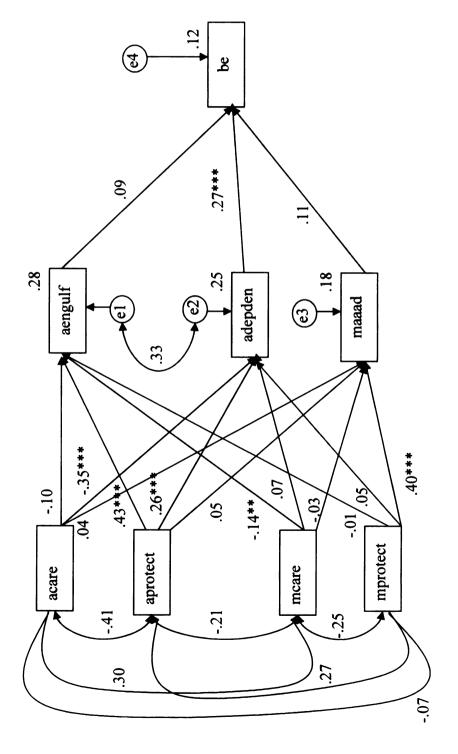


Figure 9. Structural model of BN - Model with Correlated Errors (i.e., e1 and e2).

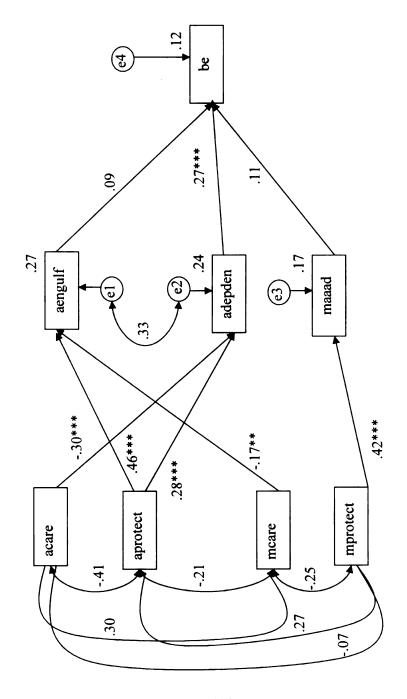


Figure 10. Structural model of BN – Reduced Model (without nonsignificant paths between mother-child relationship and separation-individuation variables).

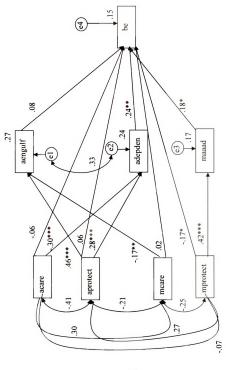


Figure 11. BN - Reduced Model with direct effects between the mother-child relationship variables and binge

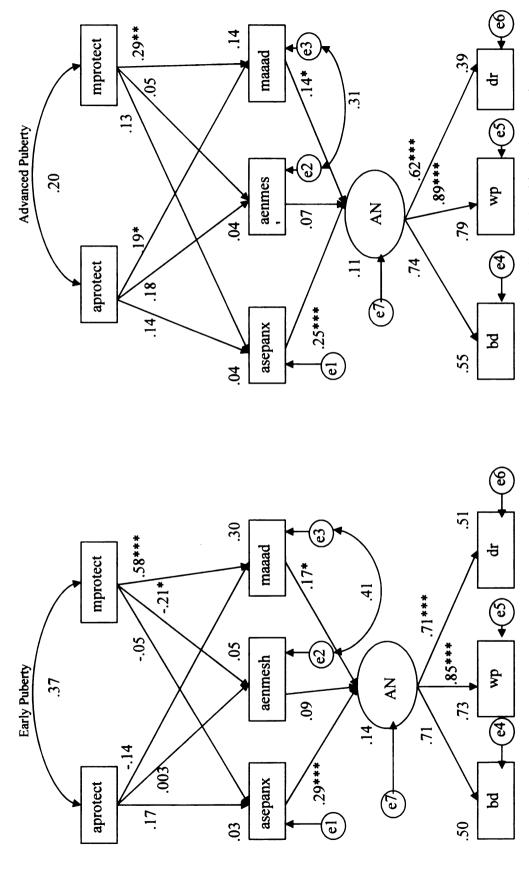


Figure 12. AN-Reduced Model with puberty moderating relationships between the mother-child relationship and separationindividuation variables.

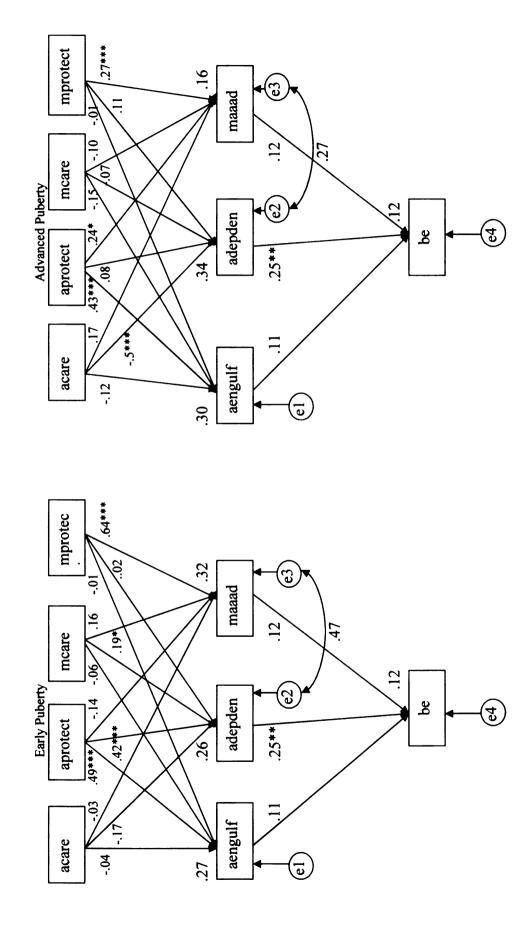


Figure 13. BN - Reduced Model with puberty moderating relationships between the mother-child relationship and separationindividuation variables (correlation values for exogenous variables not shown).

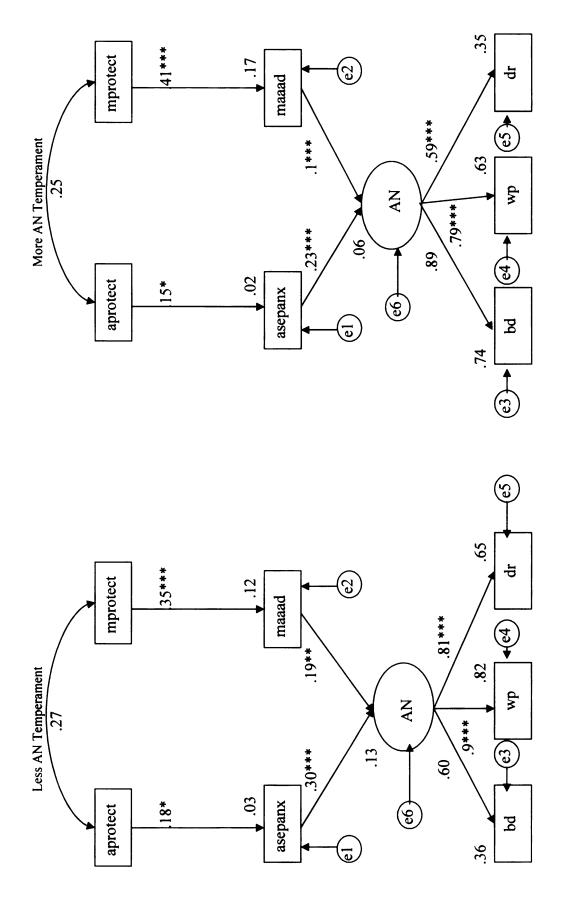


Figure 14. AN - Reduced Model with AN temperament moderating relationships among factor loadings of AN-like disordered eating.

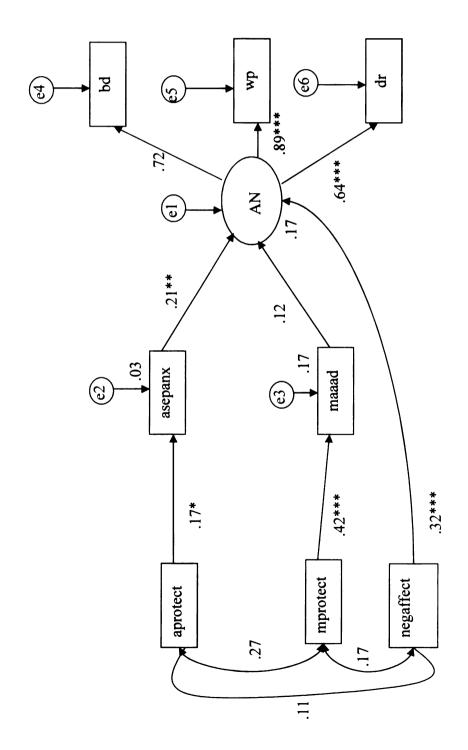


Figure 15. Latent Variable Structural Equation Model of AN- Reduced Model with Negative Affect added as a direct effect.

TABLES

Table 1. Internal consistency reliability estimates for subscales used in analyses.

Variable	Adolescent Report	Mother Report
MEBS Scales		
Weight Preoccupation	.78	
Body Dissatisfaction	.82	
Binge Eating	.71	
EDEQ Scale		
Restraint	.74	
PBI Scales		
Maternal Care	.85	.75
Maternal Protection	.76	.68
SITA Scales		
Enmeshment Seeking	.94	
Separation Anxiety	.78	
Nurturance Seeking	.76	••
Healthy Separation	.94	
Engulfment Anxiety	.91	••
Dependency Denial	.94	••
PASAS Scale		
Maternal Anxiety about		.88
Adolescent Distancing		
PDS Scale		
Pubertal Development	.82	.86
EATQ Scales		
Negative Affect	.84	.87
Inhibitory Control	.47	.69
Shyness	.78	.88

Note. Alpha coefficients estimates are internal consistency coefficients.

Table 2. Pearson product correlations between PBI, SITA, PASAS, MEBS, and EDEQ scores.

Variable	Disordered Eating	weight preoccupation (a)	2. body dissatisfaction (a)	3. dietary restraint (a)	4. binge eating (a)	PBI Scores	5. maternal care (a)	6. maternal protection (a)	7.maternal care (m)	8. maternal protection (m)	Separation Individuation	9. enmeshment seeking (a)	10. separation anxiety (a)	11. engulfment anxiety (a)	12. dependency denial (a)	13. maternal anxiety about adolescent
_			.64**	**85.	.44**	1	12	**61.	05	90.	,	Π.	.25**	.28**	.24**	.20**
2			1	.48**	.44**	1	.02	.10	02	.03	1	.07	.24**	.15*	.15*	.17*
c.					.21**	1	10.	.16*	10.	90.	1	.21**	.20**	.21**	11.	.22**
4					:	:	20**	.20**	03	04	1	.05	**0£	.23**	.32**	.14*
S							1	41**	.30**	07	ı	.25**	.13	31**	44**	02
9								1	.21**	.27**	1	90.	.17*	**65.	.40**	.15*
7									1	.15*	1	.15*	.14*	.25**	T.	13
00										:	i	90:-	60.	.15*	.13	.42**
6												1	.35**	.12	05	.01
10													1	**61.	.28**	.23
=														1	.47**	.12
12															1	.07
13																1

Table 3. Goodness of fit indices for single-group analyses of models with AN-like disordered eating.

Model	χ^2 (df)	χ^2 diff	CFI	GFI	RMSEA (CI)
1. AN – Original Model	50.017 (15)		.883	.945	.108 (.076, .142)
2. AN – Model with Correlated Errors	23.472 (14)	26.545 a *	.968	.972	.058 (.00, .098)
3. AN – Reduced Model	17.140 (13)		.985	.977	.040 (.00, .086)
3a. AN – Reduced Model with direct path between aprotect and AN	13.66 (12)	4.56 b	.994	.981	.026 (.00, .079)
3b. AN- Reduced Model with direct path between mprotect and AN	16.82 (12)	.32 °	.977	.982	.045 (.00, .09)

Note. $\chi^2=$ model chi-square statistic. $\chi^2_{\rm diff}{}^a=$ compared to model 1. $\chi^2_{\rm diff}{}^b=$ compared to model 3. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high). M-C Rel = mother-child relationship. AN – Model with Correlated Errors = includes all paths, variables, and correlated errors. AN – Reduced Model = eliminates the Enmeshment Seeking variable and nonsignificant paths. AN – Reduced Model with direct path between M-C Rel and AN = model 3 with direct paths between the mother-child relationship and AN-like disordered eating. * *p < 0.05.

Table 4. Unstandardized coefficients for AN - Reduced Model.

Paths	Unstandardized Coefficients (SE)
aprotect → asepanx	.21* (.08)
mprotect → maaad	1.21*** (.19)
asepanx → AN-like disordered eating	.05*** (.02)
maaad → AN-like disordered eating	.02* (.01)
AN-like disordered eating → bd	1.00 ()
AN-like disordered eating → wp	1.44*** (.16)
AN-like disordered eating → dr	.50*** (.06)

Note. Dashes (--) = standard error not estimated because coefficient was fixed to 1.0. * p < .05; ** p < .01; *** p < .001.

Table 5. Goodness of fit indices for single-group analyses of BN-like disordered eating.

Model	χ^2 (df)	χ^2 diff	CFI	GFI	RMSEA (CI)
1. BN	30.062 (7)		.916	.965	.128 (.08, .18)
model					
2. BN –	7.480 (6)	25.95 a*	.995	.991	.035 (.00, .103)
Model with					
Correlated					
Errors					
2a. BN –	.69 (2)	6.79 ^b	.999	1.000	.000 (.00, .10)
Model with					
Correlated		•			
Errors and					
direct paths					
between M-					
C Rel and					
BE		, -			
3. BN –	11.557		1.00	.986	.00 (.00, .063)
Reduced	(13)				
Model					
3a. BN –	4.77 (9)	6.787 ^c	.994	1.000	.00 (.00, .04)
Reduced					
Model with					
direct paths					
between M-					
C Rel and					
BE					

Note. χ^2 = model chi-square statistic. $\chi^2_{\text{diff}}{}^a$ = compared to model 1. $\chi^2_{\text{diff}}{}^b$ = compared to model 2. $\chi^2_{\text{diff}}{}^c$ = compared to model 3. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high). M-C Rel = mother-child relationship. BE = binge eating. BN – Model with Correlated Errors = all paths, variables, and correlated errors. BN – Model with Correlated Errors and direct path between M-C Rel and BE = model 2 with direct paths between the mother-child relationship and binge eating. BN – Reduced Model = eliminates insignificant paths between mother-adolescent relationship and separation-individuation variables. BN – Reduced Model with direct paths between M-C Rel and BE = model 3 with direct path between the mother-child relationship and binge eating.

^{*} *p* < .05.

Table 6. Unstandardized coefficients for BN – Reduced Model.

Paths	Unstandardized Coefficients (SE)
acare → adepden	28*** (.06)
aprotect → aengulf	.46*** (.06)
aprotect → adepden	.25*** (.06)
mcare → aengulf	25** (.09)
mprotect → maaad	1.21*** (.19)
aengulf → be	.03 (.02)
adepden → be	.08*** (.02)
maaad → be	.02 (.01)

^{*} p < .05; ** p < .01; *** p < .001.

Table 7. Goodness of fit indices for multiple-group analyses of AN models examining puberty as a moderator.

Model	χ^2 (df)	χ^2 diff	CFI	GFI	RMSEA (CI)
AN – Reduced					, ,
Model					
1. Unconstrained	41.560 (26)		.946	.948	.055 (.02, .09)
2. Fully	46.323 (32)	4.763	.950	.943	.047 (.00, .08)
Constrained					
AN - Model with					
Correlated					
Errors					
1. Unconstrained	41.418 (28)		.958	.954	.049 (.00, .08)
2. Fully	63.054 (39)	21.636*	.925	.932	.056 (.03, .08)
Constrained					
3. SI \rightarrow AN paths	57.489 (36)	16.071*	.933	.937	.055 (.03, .08)
unconstrained					
4. M-C Rel → SI	47.220 (33)	5.802	.956	.947	.047 (.00, .075)
paths					
unconstrained					

Note. χ^2 = model chi-square statistic. χ^2_{diff} = compared to respective model 1. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high). Fully Constrained = all regression coefficients and factor loadings constrained. M-C Rel = mother-child relationship. SI = separation individuation. SI \rightarrow AN paths unconstrained = paths between separation-individuation variables and AN factor unconstrained; all other regression coefficients and factor loadings constrained. M-C Rel \rightarrow SI paths unconstrained = paths between the mother-child relationship and separation-individuation variables unconstrained; all other regression coefficients and factor loadings constrained.

^{*} p < .05.

Table 8. Unstandardized coefficients for AN – Model with Correlated Errors among puberty groups.

Paths	Unstandardized Coefficients (SE) Early Puberty	Unstandardized Coefficients (SE) Advanced Puberty
aprotect →	.23 (.13)	.16 (.12)
asepanx	.23 (.13)	.10 (.12)
aprotect →	.004 (.13)	.19 (.11)
aenmesh	.001 (.13)	,
aprotect →	32 (21)	.39* (.20)
maaad		
mprotect → asepanx	08 (.17)	.20 (.16)
mprotect → aenmesh	34* (.17)	.08 (.14)
mprotect → maaad	1.67 *** (.26)	.82** (.27)
asepanx → AN- like disordered eating	.05*** (.02)	.05*** (.02)
aenmesh → AN- like disordered eating	.02 (.02)	.02 (.02)
maaad → AN- like disordered eating	.02* (.01)	.02* (.01)
AN-like disordered eating → bd	1 ()	1 ()
AN-like disordered eating → wp	1.48*** (.17)	1.48*** (.17)
AN-like disordered eating → dr	.50*** (.06)	.50*** (.06)

Note. Paths between the mother-child relationship and separation-individuation variables unconstrained; all other regression coefficients and factor loadings constrained. Dashes (--) = standard error not estimated because coefficient was fixed

to 1.0.

^{*} p < .05; ** p < .01; *** p < .001.

Table 9. Goodness of fit indices for multiple-group analyses of BN models examining puberty as a moderator.

Model	χ^2 (df)	χ^2 diff	CFI	GFI	RMSEA (CI)
BN – Reduced Model					
1. Unconstrained	47.005 (26)		.946	.935	.064 (.03, .09)
2. Fully Constrained	61.133 (34)	14.128	.930	.916	.063 (.04, .09)
BN – Model with Correlated Errors					
1. Unconstrained	26.385 (12)		.955	.969	.078 (.04, .12)
2. Fully Constrained	57.353 (27)	30.968*	.906	.936	.075 (.05, .10)
 SI → BE paths unconstrained 	52.773 (24)	26.388*	.911	.942	.078 (.05, .11)
 M-C Rel → SI paths unconstrained 	30.965 (15)	4.58	.950	.964	.073 (.04, .11)

Note. χ^2 = model chi-square statistic. χ^2_{diff} = compared to respective model 1. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high). Fully Constrained = all regression coefficients constrained. M-C Rel = mother-child relationship. SI = separation individuation. BE = binge eating. SI \rightarrow BE paths unconstrained = paths between separation-individuation variables and binge eating unconstrained; all other regression coefficients constrained. M-C Rel \rightarrow SI paths unconstrained = paths between the mother-child relationship and separation-individuation variables unconstrained; all other regression coefficients constrained.

Table 10. Unstandardized coefficients for BN – Model with Correlated Errors among puberty groups.

Paths	Unstandardized	Unstandardized
	Coefficients	Coefficients
	(SE)	(SE)
	Early puberty	Advanced puberty
acare→ aengulf	05 (.11)	11 (.09)
acare→ adepden	18 (.10)	41*** (.08)
acare→ maaad	07 (.24)	.33 (.21)
Aprotect → aengulf	.51*** (.10)	.41*** (.09)
Aprotect→ adepden	.40*** (.10)	.07 (.08)
Aprotect→ maaad	32 (.22)	.51* (.22)
mcare→ aengulf	10 (.15)	21 (.13)
mcare→ adepden	.28* (.14)	09 (.11)
mcare→ maaad	.55 (.32)	31 (.31)
mprotect → aengulf	02 (.13)	01 (.11)
mprotect→ adepden	.02 (.12)	.13 (.10)
mprotect→ maaad	1.83*** (.28)	.76** (.27)
aengulf → be	.03 (.02)	.03 (.02)
Adepden → be	.08** (.02)	.08** (.02)
maaad → be	.02 (.01)	.02 (.01)

Note. Paths between the mother-child relationship and separation-individuation variables unconstrained; all other regression coefficients constrained. * p < .05; *** p < .01; **** p < .001.

Table 11. Cross-tabulation table examining composition of age and puberty groups.

		Age Groups		
		Younger Age	Older Age	Total
Puberty Groups	Early Puberty	79	20	99
-	Late Puberty	21	81	102
Total		100	101	201

Table 12. Goodness of fit indices for multiple-group analyses of AN – Reduced Model

examining temperament as a moderator. Model X^{2} (df) χ^2 diff CFI GFI RMSEA (CI) AN - Reduced Model AN Temperament 1. Unconstrained 23.354 (26) 1.00 .968 .00 (.00, .049) 2. Fully 46.958 (32) 26.304* .947 .937 .047 (.00, .08) Constrained 41.340 (30) 17.986* .957 .943 3. $SI \rightarrow AN$ paths .044 (.00, .07) unconstrained 4. M-C Rel → SI 45.295 (30) 21.941* .943 .939 .051 (.01, .08) paths unconstrained 5. Factor loadings .964 26.204 (30) 2.85 1.00 .00 (.00, .04) unconstrained Shyness 1. Unconstrained 30,413 (26) .984 .960 .029 (.00, .07) 2. Fully 38.074 (32) 7.661 .977 .948 .031 (.00, .06) Constrained Inhibitory Control 1 Unconstrained .955 33,948 (26) .969 .039 (.00, .07) 2. Fully 44.958 (32) 11.01 .949 .943 .045 (.00, .07) Constrained Negative Affect 1. Unconstrained .025 (.00, .06) 29.318 (26) 986 .961 2. Fully 37.548 (32) 8.23 .976 .948 .030 (.00, .06)

Note, χ^2 = model chi-square statistic. χ^2_{diff} = compared to respective model 1. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high).

Fully Constrained = all regression coefficients and factor loadings constrained. M-C Rel = mother-child relationship. SI = separation individuation. SI \rightarrow AN paths unconstrained = paths between separation-individuation variables and AN factor

unconstrained; all other regression coefficients and factor loadings constrained. M-C Rel \rightarrow SI paths unconstrained = paths between the mother-child relationship and separation-individuation variables unconstrained; all other regression coefficients and factor loadings constrained.

Constrained

^{*} p < .05

Table 13. Unstandardized coefficients for AN - Reduced Model among summed AN Temperament groups.

Paths	Unstandardized Coefficients (SE)	Unstandardized Coefficients (SE)
	Less AN Temperament	More AN Temperament
aprotect → asepanx	.19* (.08)	.19* (.08)
mprotect → maaad	1.07*** (.19)	1.07*** (.19)
asepanx → AN-like disordered eating	.05*** (.02)	.05*** (.02)
maaad → AN-like disordered eating	.02** (.09)	.02** (.09)
AN-like disordered eating → bd	1.00 ()	1.00 ()
AN-like disordered eating → wp	1.76***(.29)	1.10* (.17)
AN-like disordered eating → dr	.89*** (.15)	.32*** (.06)

Note. Factor loadings unconstrained; regression coefficients constrained. Dashes (--) = standard error not estimated because coefficient was fixed to 1.0.

^{*} p < .05; ** p < .01; *** p < .001.

Table 14. Goodness of fit indices for single-group analyses of AN – Reduced Model examining direct effects of temperament.

Model	χ^2 (df)	CFI	GFI	RMSEA (CI)
AN Temperament	45.35 (17)	.91	.95	.09 (.06, .12)
Shyness	30.18 (17)	.95	.96	.06 (.02, .10)
Inhibitory Control	28.33 (17)	.96	.97	.06 (.01, .09)
Negative Affect	35.30 (17)	.94	.96	.07 (.04, .11)

Note. χ^2 = model chi-square statistic. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high).

Table 15. Unstandardized and standardized coefficients for AN - Reduced Model with direct effects of temperament variables.

Paths	Unstandardized Coefficients (SE)	Standardized Coefficients
Negative Affect as Direct Effect		
aprotect → asepanx	.21* (.08)	.17*
mprotect → maaad	1.21*** (.19)	.42***
asepanx → AN-like disordered eating	.04** (.01)	.21**
maaad → AN-like disordered eating	.01 (.01)	.12
negaffect → AN-like	.82*** (.20)	.32***
disordered eating		
AN-like disordered eating → bd	1.00 ()	.72
AN-like disordered eating → wp	1.51*** (.17)	.89***
AN-like disordered eating → dr	.50*** (.06)	.64***
Inhibitory Control as Direct Effect		
aprotect → asepanx	.21* (.08)	.17*
mprotect → maaad	1.21*** (.19)	.42***
asepanx → AN-like disordered eating	.06*** (.02)	.28***
inhib → AN-like disordered eating	41* (.20)	15*
AN-like disordered eating → bd	1.00 ()	.74
AN-like disordered eating → wp	1.42*** (.16)	.86***
AN-like disordered eating → dr Shyness as direct effect	.50*** (.06)	.66***
aprotect → asepanx	.21* (.08)	.17*
mprotect → maaad	1.21*** (.19)	.42***
asepanx → AN-like disordered eating	.05*** (.02)	.26***
maaad → AN-like disordered eating	.02* (.01)	.19*
shy → AN-like disordered	03 (.20)	01
eating	` ,	
AN-like disordered eating → bd	1.00 ()	.73
AN-like disordered eating → wp	1.44*** (.16)	.86***
AN-like disordered eating → dr	.50*** (.06)	.66***

Note. Dashes (--) = standard error not estimated because coefficient was fixed to 1.0. p < .05; ** p < .01; *** p < .001.

Table 16. Goodness of fit indices for multiple-group analyses of BN – Reduced Model examining temperament as a moderator.

Model	χ^2 (df)	X ² diff	CFI	GFI	RMSEA (CI)
BN Temperament					
1. Unconstrained	26.189 (26)		.999	.969	.006 (.00, .06)
2. Fully	40.720 (34)	14.531	.971	.954	.032 (.00, .06)
Constrained					
Shyness					
1. Unconstrained	30.040 (26)		.986	.964	.028 (.00, .06)
2. Fully	41.299 (34)	11.259	.964	.951	.033 (.00, .06)
Constrained		1			
Inhibitory Control					
1. Unconstrained	27.339 (26)		.994	.967	.016 (.00, .06)
2. Fully	45.275 (34)	17.936*	.951	.947	.041 (.00, .07)
Constrained					
Negative Affect					
1. Unconstrained	28.477 (26)		.989	.967	.022 (.00, .06)
2. Fully	35.341 (34)	6.954	.994	.959	.014 (.00, .05)
Constrained					

Note. χ^2 = model chi-square statistic. χ^2_{diff} = compared to respective model 1. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high). Fully Constrained = all regression coefficients constrained. M-C Rel = mother-child relationship. SI = separation individuation. BE = binge eating. M-C Rel \rightarrow SI paths unconstrained = paths between the mother-child relationship and separation-individuation variables unconstrained; all other regression coefficients constrained. SI \rightarrow BE paths unconstrained = paths between separation-individuation variables and binge eating unconstrained; all other regression coefficients constrained. p < .05.

Table 17. Goodness of fit indices for multiple-group analyses of BN models examining direct effects of temperament.

Model	χ^2 (df)	CFI	GFI	RMSEA (CI)
BN Temperament	43.53 (16)	.92	.96	.09 (.06, .13)
Shyness	27.68 (16)	.96	.97	.06 (.02, .10)
Inhibitory Control	21.05 (16)	.98	.98	.04 (.00, .08)
Negative Affect	45.28 (16)	.91	.95	.10 (.06, .13)

Note. χ^2 = model chi-square statistic. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high).

Table 18. Unstandardized and standardized coefficients for BN – Reduced Model with Inhibitory Control and Shyness as direct effects.

Paths	Unstandardized Coefficients (SE)	Standardized Coefficients		
Inhibitory Control as				
Direct Effect				
acare → adepden	28*** (.06)	30***		
aprotect → aengulf	.46*** (.06)	.46***		
aprotect → adepden	.25*** (.06)	.28***		
mcare → aengulf	25** (.09)	17**		
mprotect → maaad	1.21*** (.19)	.42***		
inhib → be	.08 (21)	.02		
aengulf → be	.02 (.02)	.07		
adepden → be	.07** (.02)	.24**		
maaad → be	.01 (.01)	.08		
Shyness as Direct Effect				
acare → adepden	28*** (.06)	30***		
aprotect → aengulf	.46*** (.06)	.46***		
aprotect → adepden	.25*** (.06)	.28***		
mcare → aengulf	25** (.09)	17**		
mprotect → maaad	1.21*** (.19)	.42***		
shy → be	23 (.22)	07		
aengulf → be	.02 (.02)	.08		
adepden → be	.09*** (.02)	.28***		
maaad → be	.02 (.01)	.13		

^{*} *p* < .05; ** *p* < .01; *** *p* < .001.

APPENDICES

APPENDIX A: Examining age as a moderator in the AN and BN models

Table A1. Goodness of fit indices for multiple-group analyses of AN models examining age as a moderator.

Model	χ^2 (df)	χ^2 diff	CFI	GFI	RMSEA (CI)
AN – Reduced					
Model					
1. Unconstrained	31.715 (26)		.979	.957	.033 (.00, .07)
2. Fully Constrained	35.132 (32)	3.417	.988	.951	.022 (.00, .06)
AN - Model with					
Correlated Errors					
1. Unconstrained	41.238 (28)		.957	.953	.049 (.00, .08)
2. Fully Constrained	54.220 (39)	12.982	.936	.950	.044 (.00, .07)

Note. χ^2 = model chi-square statistic. χ^2_{diff} = compared to respective model 1. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high). Fully Constrained = all regression coefficients and factor loadings constrained.

Table A2. Goodness of fit indices for multiple-group analyses of BN models examining age as a moderator.

Model	χ^2 (df)	χ ² diff	CFI	GFI	RMSEA (CI)
BN - Reduced Model					
1. Unconstrained	35.041 (26)		.971	.960	.042 (.00, .07)
2. Fully Constrained	52.079 (34)	17.038*	.941	.940	.052 (.02, .08)
3. SI → BE paths unconstrained	48.080 (31)	13.039*	.945	.944	.053 (.02, .08)
4. M-C Rel → SI paths unconstrained	39.039 (29)	3.998	.967	.955	.042 (.00, .07)
BN - Model with					
Correlated Errors					
1. Unconstrained	24.042 (12)		.961	.972	.071 (.03, .11)
2. Fully Constrained	48.488 (27)	24.446	.930	.956	.063 (.03, .09)

Note. $\chi^2=$ model chi-square statistic. $\chi^2_{\rm diff}=$ compared to respective model 1. CFI = comparative fit index. GFI= goodness-of-fit index. RMSEA= root mean square error of approximation. CI = 90% Confidence Interval (low, high). Fully Constrained = all regression coefficients constrained. M-C Rel = mother-child relationship. SI = separation individuation. BE = binge eating. SI \rightarrow BE paths unconstrained = paths between separation-individuation variables and binge eating; all other regression coefficients constrained. M-C Rel \rightarrow SI paths unconstrained = paths between the mother-child relationship and separation-individuation variables unconstrained; all other regression coefficients constrained.

^{*} p < .05.

Table A3. Unstandardized and standardized coefficients for BN - Reduced Model among age groups.

Paths	Unstandardized	Standardized	Unstandardized	Standardize	
	Coefficients (SE)	Coefficients	Coefficients	d	
			(SE)	Coefficients	
	Youn	ger	Older		
acare →	08 (.11)	07	42*** (.07)	54***	
adepden					
aprotect → aengulf	.45*** (.10)	.43***	.47*** (.08)	.50***	
aprotect→ adepden	.45*** (.10)	.44***	.08 (.26)	.10	
mcare→ aengulf	19 (.14)	12	29* (.12)	21*	
mprotect→ maaad	1.43*** (.29)	.45***	1.14*** (.25)	.42***	
aengulf → be	.02 (.02)	.09	.02 (.02)	.08	
adepden → be	.09** (.02)	.33**	.09*** (.02)	.27***	
maaad → be	.02 (.01)	.14	.02 (.01)	.11	

Note. Paths between the mother-child relationship and separation-individuation variables unconstrained; all other regression coefficients constrained. * p < .05; ** p < .01; *** p < .001.

REFERENCES

REFERENCES

- Allison, D.B., Kalinsky, L.B., & Gorman, B.S. (1992). The comparative psychometric properties of three measure of dietary restraint. *Psychological Assessment*, 4, 391-398.
- American Psychiatric Association (1994). Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). American Psychiatric Association, Washington, DC.
- Arbuckle, J., & Wothke, W. (1999). AMOS 4.0 User's Guide. Chicago, IL: Smallwaters Corporation.
- Attie, I., & Brooks-Gunn, J. (1989). Development of eating problems in adolescent girls: A longitudinal study. *Developmental Psychology*, 25, 70-79.
- Baltes, P., Staudinger, U., & Lindenberger, U. (1999). Lifespan psychology: Theory and application to intellectual functioning. *Annual Review of Psychology*, 50, 471 -507.
- Baron, R., & Kenny, D. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic and statistical considerations. Journal of Personality and Social Psychology, 51, 1173-1182.
- Berg, M.L., Crosby, R.D., Wonderlich, S.A., & Hawley, D. (2000). The relationship of temperament and perceptions of nonshared environment in bulimia nervosa. *International Journal of Eating Disorders*, 28, 148-154.
- Bloks, H., Hoek, H., Callewaert, I., & van Furth, E. (2004). Stability of personality traits in patients who received intensive treatment for a severe eating disorder. *The Journal of Nervous and Mental Disease*, 192, 129-138.
- Blos, P. (1965). On adolescence: A psychoanalytic interpretation. New York,
 Free Press.
- Blos, P. (1967). The second individuation process of adolescence. *Psychoanalytic Study of the Child*, 22, 162-186.
- Brandt, D. (1977). Separation and identity in adolescence: Erikson and Mahler some similarities. *Contemporary Psychoanalysis*, 13, 507-518.
- Brookings, J., & Wilson, J. (1994). Personality and family-environment predictors of self reported eating attitudes and behaviors. *Journal of Personality Assessment*, 63, 313-326.

- Brooks-Gunn, J., & Warren, M. (1985). Measuring physical status and timing in early adolescence: A developmental perspective. *Journal of Youth and Adolescence*, 14, 163-184.
- Brooks-Gunn, J., Warren, M.P., Rosso, J., & Gargiulo, J. (1987). Validity of self report measures of girls' pubertal status. *Child Development*, 58, 829-841.
- Brown, L., & Gilligan, C. (1992). Meeting at the crossroads: Women's psychology and girls' development. Cambridge, MA: Harvard University Press.
- Bruch, H. (1974). Eating Disorders: Obesity, Anorexia Nervosa, and the Person Within. London: Routledge & Kegan Paul.
- Bulik, C., Sullivan, P., Fear, J., & Pickering, A. (2000). The outcome of anorexia nervosa: eating attitudes, personality, and parental bonding. *International Journal of Eating Disorders*, 28, 139-147.
- Bulik, C., Sullivan, P., & Kendler, K. (1998). Heritability of binge-eating and broadly-defined bulimia nervosa. *Biological Psychiatry*, 44, 1210-1218.
- Bulik, C., Sullivan, P., Wade, T., & Kendler, K. (2000). Twin studies of eating disorders: A review. *International Journal of Eating Disorders*, 27, 1-20.
- Bulik, C., Sullivan, P., Weltzin, T., & Kaye, W. (1995). Temperament in eating disorders. *International Journal of Eating Disorders*, 17, 251-261.
- Bulik, C., Wade, T., & Kendler, K. (2001). Characteristics of monozygotic twins discordant for bulimia nervosa. *International Journal of Eating Disorders*, 29, 1-10.
- Calam, R., Waller, G., Slade, P., & Newton, T. (1990). Eating disorders and perceived relationships with parents. *International Journal of Eating Disorders*, 9, 479-485.
- Capaldi, D., & Rothbart, M. (1992). Development and validation of an early adolescent temperament measure. *Journal of Early Adolescence*, 12, 153-173.
- Casper, R., Hedeker, D., & McClough, J. (1992). Personality dimensions in eating disorders and their relevance for subtyping. *Journal of the American Academy of Child and Adolescent Psychiatry*, 31, 830-839.
- Chassler, L. (1997). Understanding anorexia nervosa and bulimia nervosa from an attachment perspective. *Clinical Social Work Journal*, 25, 407-423.

- Childress, A., Brewerton, T., Hodges, E., & Jarrell, M. (1993). The Kids' Eating Disorders Survey (KEDS): A study of middle school students. *Journal of the American Academy of Child and Adolescent Psychiatry*, 32, 843-850.
- Chodorow, N. (1974). Family structure and feminine personality. In M. Rosaldo & L. Lamphere (Eds.), *Women: Culture and Society*. Stanford, CA: Stanford University Press.
- Christenson, R., & Wilson, W. (1985). Assessing pathology in the separation individuation process by an inventory: A preliminary report. *Journal of Nervous and Mental Disease*, 179, 561-565.
- Clark, L., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, 103, 103-116.
- Cohen, J. (1992). A power primer. Psychological Bulletin, 112, 153-159.
- Compas, B., Connor-Smith, J., & Jaser, S. (2004). Temperament, stress reactivity, and coping: Implications for depression in childhood and adolescence. *Journal of Clinical Child and Adolescent Psychology*, 33, 21-31.
- Cooper, C., Grotevant, H., & Condon, S. (1983). Individuality and connectedness in the family as a context for adolescent identity formation and role-taking skill. In H. Grotevant & C. Cooper (Eds.), *Adolescent development in the family (pp. 43-60)*. San Francisco: Jossey-Bass.
- Derryberry, D., & Rothbart, M. (1988). Arousal, affect, and attention as components of temperament. *Journal of Personality and Social Psychology*, 55, 958-966.
- Dolan, B. (1991). Cross-cultural aspects of anorexia nervosa and bulimia: A review. *International Journal of Eating Disorders*, 10, 67-79.
- Dorn, L., Crokett, L., & Petersen, A. (1988). The relations of pubertal status to intrapersonal changes in young adolescents. *Journal of Early Adolescence*, 8, 405 -419.
- Edward, J., Ruskin, N., & Turrini, P. (1981). Separation-individuation: Theory and application. New York: Gardner.
- Elkins, I., McGue, M., & Iacono, W. (1997). Genetic and environmental influences on parent-son relationships: Evidence for increasing genetic influence during adolescence. *Developmental Psychology*, 33, 351-363.
- Ellis, L., & Rothbart, M. (2005). Revision of the Early Adolescent Temperament Questionnaire. Manuscript in preparation.

- Erikson, E. (1963). Childhood and society (2nd ed.). New York: Norton.
- Erikson, E. (1968). Identity: Youth and crisis. New York: Norton.
- Essman, A. (1980). Adolescent psychopathology and the rapprochement phenomenon. *Adolescent Psychiatry*, 8, 320-331.
- Fairburn, C., & Beglin, S. (1994). Assessment of eating disorders: Interview or self report questionnaire? *International Journal of Eating Disorders*, 16, 363-370.
- Fassino, S., Abbate-Dagga, G., Amianto, F., Leombruni, P., Boggio, S., & Rovera, G. (2002). Temperament and character profile of eating disorders: a controlled study with the Temperament and Character Inventory. *International Journal of Eating Disorders*, 32, 412-425.
- Faul, F., & Erdfelder, E. (1992). GPOWER: A priori, post-hoc, and compromise power analyses for MS-DOS [Computer program]. Bonn, FRG: Bonn University, Department of Psychology.
- Finkel, D., & McGue, M. (1993). Twenty-five year follow-up of child-rearing practices: Reliability of retrospective data. *Personality and Individual Differences*, 15, 147-154.
- Finn, S., Hartman, M., Leon, G., Lawson, L. (1986). Eating disorders and sexual abuse: Lack of confirmation for a clinical hypothesis. *International Journal of Eating Disorders*, 5, 1051-1060.
- Foulkes, P.V. (1996). Eating disorders, family, and therapy. *Australian Journal of Psychotherapy*, 15, 28-42.
- Friedlander, M., & Siegel, S. (1990). Separation-individuation difficulties and cognitive-behavioral indicators of eating disorders among college women. *Journal of Counseling Psychology*, 37, 74-78.
- Garfinkel, P., Garner, D., & Rose, J. (1983). A comparison of characteristics in families of patients with anorexia nervosa and normal controls. *Psychological Medicine*, 13, 821-828.
- Garner, D., Garfinkel, P., Schwart, D., & Thompson, M. (1980). Cultural expectations of thinness in women. *Psychological Reports*, 47, 483-491.
- Garner, D.M., Olmsted, M.P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders*, 2, 15-34.
- Ghaderi, A., & Scott, B. (2000). The big five and eating disorders: A prospective study in the general population. *European Journal of Personality*, 14, 311 -323.

- Gilligan, C. (1982). In a different voice. Cambridge, MA: Harvard University Press.
- Goldstein, M. (1981). Family factors associated with schizophrenia and anorexia nervosa. *Journal of Youth and Adolescence*, 10, 385-405.
- Goldsmith, H., Buss, A., Plomin, R., Rothbart, M., Thomas, A., Chess, C., Hinde, R., & McCall, R. (1987). Roundtable: What is temperament? Four approaches. *Child Development*, 58, 505-529.
- Graber, J., & Brooks-Gunn, J. (1996). Transitions and turning points: Navigating the passage from childhood through adolescence. *Developmental Psychology*, 32, 768-776.
- Green, M., Rogers, P., Elliman, N., & Gatenby, S. (1994). Impairment of cognitive performance associated with dieting and high levels of dietary restraint. *Physiology and Behavior*, 55, 447-452.
- Grice, D., Halmi, K., Fichter, M., Strober, M., Woodside, D., Treasure, J., et al. (2002). Evidence for a susceptibility gene for anorexia nervosa on Chromosome 1. American Journal of Human Genetics, 70, 787-892.
- Heaven, P., Mulligan, K., Merrilees, R., Woods, T., & Fairooz, Y. (2001). Neuroticism and conscientiousness as predictors of emotional external, and restrained eating behaviors. *International Journal of Eating Disorders*, 30, 161-166.
- Hedlund, S., Fichter, M., Quadflieg, N., & Brandl, C. (2003). Expressed emotion, family environment, and parental bonding in bulimia nervosa: A 6-year investigation. *Eating and Weight Disorders*, 8, 26-35.
- Herzog, D. B., Dorer, D. J., Keel, P. K., Selwyn, S. E., Ekeblad, E. R., & Richards, A. (1999). Recovery and relapse in anorexia and bulimia nervosa: A 7.5-year follow up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 38, 829-837.
- Hill, J., Holmbeck, G., Marlow, L., Green, T., & Lynch, M. (1985) Pubertal status and parent-child relations in families of seventh-grade boys. *Journal of Early Adolescence*, 5, 31-44.
- Hock, E., Eberly, M., Bartle-Haring, S., Ellwanger, P., & Widaman, K. (2001). Separation anxiety in parents of adolescents: Theoretical significance and scale development. *Child Development*, 72, 284-298.
- Hoffman, J. (1984). Psychological separation of late adolescents from their parents. *Journal of Counseling Psychology*, 31, 170-178.

- Hollingshead, A. (1975). Four Factor Index of Social Status. Unpublished manuscript, Yale University, New Haven, CT.
- Horesh, N., Apter, A., Ishai, J., Danzinger, Y., Miculincer, M., Stein, D., Lepkifker, E., & Minouni, M. (1996). Abnormal psychosocial situations and eating disorders in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35(7), 921-927.
- Hsu, L. (1990). Eating Disorders. New York: Guilford Press.
- Hsu, L. K. G. (1996). Epidemiology of the eating disorders. *Psychiatric Clinics of North America*, 19,681-700.
- Humphrey, L.L. (1989). Observed family interactions among subtypes of eating disorders using structural analysis of social behavior. *Journal of Consulting and Clinical Psychology*, 57, 206-214.
- Huprich, S., Stepp, S., Graham, A., & Johnson, L. (2004). Gender differences in dependency, separation, object relations and pathological eating behavior and attitudes. *Personality and Individual Differences*, 36, 801-811.
- Johnson, C. (1991). Treatment of eating-disordered patients with borderline and false self/narcissistic disorders. In C.Johnson (Ed.), *Psychodynamic treatment of anorexia nervosa and bulimia* (pp.165-193). New York: Guilford Press.
- Johnson, J., Cohen, P, Kasen, S., & Brook, J. (2003). Risk factors and outcomes associated with adolescent eating disorders: Findings of the children in the community study. In P. Swain (Ed.), Focus on eating disorders research (pp. 1-30). New York: Nova Biomedical Books.
- Johnson, C., & Flach, A. (1985). Family characteristics of 105 patients with bulimia. *American Journal of Psychiatry*, 142, 1321-1324.
- Josselson, R. (1980). Ego development in adolescence. In J. Adelson (Ed.) *Handbook of Adolescent Psychology* (pp. 188-210). New York: Wiley.
- Kaye, W. (1997). Anorexia nervosa, obsessional behavior, and serotonin. *Psychopharmacology Bulletin, 33,* 335-344.
- Kaye, W., Gwirtsman, H., George, D., & Ebert, M. (1991). Altered serotonin activity in anorexia nervosa after long-term weight restoration: Does elevated cerebrospinal fluid hydroxyindoleacetic acid level correlate with rigid and obsessive behavior? *Archives of General Psychiatry*, 48, 556-562.

- Kaye, W., Gwirtsman, H., George, D., Jimerson, D., & Ebert, M. (1988). CSF 5 HIAA concentration in anorexia nervosa: Reduced values in underweight subjects normalize after weight gain. *Biological Psychiatry*, 23, 102-105.
- Kayrooz, C. (1995). A thematic guide to bulimia nervosa. Australian and New Zealand Journal of Family Therapy, 16, 64-72.
- Keel, P, & Klump, K. (2003). Are eating disorders culture-bound syndromes? Implications for conceptualizing their etiology. *Psychological Bulletin*, 129, 747 -769.
- Keel, P., Leon, G., & Fulkerson, J. (2001). Vulnerability to eating disorders in childhood and adolescence. In R. Ingram (Ed.), *Vulnerability to psychopathology: Risk across the lifespan (pp.389-411)*. New York: Guilford Press.
- Keel, P., Mitchell, J., Miller, K., Davis, T., & Crow, S. (1999). Long-term outcome of bulimia nervosa. *Archives of General Psychiatry*, 56, 63-69.
- Kendler, K., MacLean, C., Neale, M., Kessler, R., Heath, A., Eaves, L. (1991). The genetic epidemiology of bulimia nervosa. *American Journal of Psychiatry*, 148, 1627-1637.
- Killen, J., Taylor, C., Hayward, C., & Wilson, D. (1994). Pursuit of thinness and onset of eating disorder symptoms in a community sample of adolescent girls: A three year prospective analysis. *International Journal of Eating Disorders*, 16, 227-238.
- Kim, S., Brody, G., & Murry, V. (2003). Factor structure of the early adolescent temperament questionnaire and measurement invariance across gender. Journal of Early Adolescence, 23, 268-294.
- Kleifield, E.I., Sunday, S., Hurt, S., & Halmi K.A. (1994). The Tridimensional Personality Questionnaire: An exploration of personality traits in eating disorders. *Journal of Psychiatric Research*, 28, 413-423.
- Kline, R. (2005). Principles and practice of structural equation modeling 2nd edition. New York: Guilford Press.
- Klump, K.L, & Burt, S.A. (in press). The Michigan State University Twin Registry (MSUTR): Genetic, environmental, and neurobiological influences on behavior across development. Twin Research in Human Genetics.
- Klump, K.L., McGue, M., & Iacono, W.G. (2000). Age differences in genetic and environmental influences on eating attitudes and behaviors in preadolescent and adolescent female twins. *Journal of Abnormal Psychology*, 109, 239-251.

- Klump, K.L., McGue, M., & Iacono, W. (2003). Differential heritability of eating attitudes and behaviors in prepubertal versus pubertal twins. *International Journal of Eating Disorders*, 33, 287-292.
- Klump, K. L., Miller, K. B., Keel, P. K., McGue, M., & Iacono, W. G. (2001). Genetic and environmental influences on anorexia nervosa syndromes in a population-based twin sample. *Psychological Medicine*, 31, 737-740.
- Klump, K. L., Wonderlich, S., Lehoux, P., Lilenfeld, L. R., & Bulik, C. M. (2002). Does environment matter? A review of nonshared environment and eating disorders. *International Journal of Eating Disorders*, 31, 118 135.
- Kocourkova, J., & Koutek, J. (2003). Eating disorders in childhood and adolescence. In P. Swain (Ed.), *Focus on eating disorders research* (pp.31-44). New York: Nova Biomedical Books.
- Koff, E., & Rierdan, J. (1993). Advanced pubertal development and eating disturbance in early adolescent girls. *Journal of Adolescent Health*, 14,433-439.
- Kog, E., & Vandereycken, W. (1989). Family interaction in eating disorder patients and normal controls. *International Journal of Eating Disorders*, 8, 11-23.
- Koronyo-Hamaoui, M., Danzinger, Y., Frisch, A., Stein, D., Leor, S., Laufer, N., et al. (2002). Association between anorexia nervosa and the hsKCa3 gene: A family based and case control study. *Molecular Psychiatry*, 7, 82-85.
- Kortegaard, L., Hoerder, J., Joergensen, J., Gillberg, C., & Kyvik, K. (2001). A preliminary population-based twin study of self-reported eating disorder. *Psychological Medicine*, 31, 361-365.
- Kroger, J. (1989). *Identity in Adolescence: The Balance between Self and Other*. London: Routledge.
- Larson, R., & Richards, M. (1991). Daily companionship in late childhood and early adolescence: Changing developmental contexts. *Child Development*, 62, 284 -300).
- Lask, B., & Bryant-Waugh, R. (1992). Early-onset anorexia nervosa and related eating disorders. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 33, 281-300.
- Le Grange, D., Lock, J., & Dymek, M. (2003). Family-based therapy for adolescents with bulimia nervosa. *American Journal of Psychotherapy*, 57, 237-251.

- Le Grange, D., Telch, C., & Tibbs, J. (1998). Eating attitudes and behaviors in 1,435 South African Caucasian and non-Caucasian college students. *American Journal of Psychiatry*, 155, 250-254.
- Leon, G., Fulkerson, J., Perry, C., & Early-Zald, M. (1995). Prospective analysis of personality and behavioral vulnerabilities and gender influences in the later development of disordered eating. *Journal of Abnormal Psychology*, 104, 140 -149.
- Leon, G., Fulkerson, J., Perry, C., Keel, P., & Klump, K. (1999). Three to four year prospective evaluation of personality and behavioral risk factors for later disordered eating in adolescent girls and boys. *Journal of Youth and Adolescence*, 28, 181-196.
- Lerner, R. (1983). A "goodness of fit" model of person-context interaction. In D. Magnusson & V.L. Allen (Eds.), *Human Development: An Interactional Perspective.* New York: Academic Press.
- Levine, J., Green, C., Millon, T. (1986). The Separation-Individuation Test of Adolescence. *Journal of Personality Assessment*, 50, 123-137.
- Levine, J., & Saintonge, S. (1993). Psychometric properties of the Separation Individuation Test of Adolescence within a clinical population. *Journal of Clinical Psychology*, 49, 492-507.
- Leung, N., Thomas, G., & Waller, G. (2000). The relationship between parental bonding and core beliefs in anorexic and bulimic women. *British Journal of Clinical Psychology*, 39, 205-213.
- Lilenfeld, L., Stein, D., Bulik, C., Strober, M., Plotnicov, K., Pollice, C., Rao, R., Merikangas, K., Nagy, L., & Kaye W. (2000). Personality traits among currently eating disordered, recovered, and never ill first-degree female relatives of bulimic and control women. *Psychological Medicine*, 30, 1399-1410.
- Lilenfeld, L., Wonderlich, S., Riso, L., Crosby, R., & Mitchell, J. (2006). Eating disorders and personality: A methodological and empirical review. *Clinical Psychology Review*, 26, 299-320.
- Lock, J., & le Grange, D. (2005). Family-Based Treatment of Eating Disorders. *International Journal of Eating Disorders*, 37, 64-67.
- Lock, J., le Grange, D., Agras, S., & Dare, C. (2001). Treatment manual for anorexia nervosa: A family-based approach. New York: Guilford Press.

- Luce, K., & Crowther, J. (1999). The reliability of the eating disorder examination -self-report questionnaire version (EDE-Q). *International Journal of Eating Disorders*, 25, 349-351.
- MacCullum, R.C., Browne, M.W., & Sugawara, H.M. (1996). Power analysis and determination of sample size for covariance structure modeling. *Psychological Methods*, 1(2), 130-149.
- Maddock, J., & Rossi, J. (2001). Statistical power of articles published in three health psychology related journals. *Health Psychology*, 20, 76-78.
- Mahler, M. (1967). "On Human Symbiosis and the Vicissitudes of Individuation." II: 77 -97.
- Mahler, M. (1975). "On the Current Status of the Infantile Neurosis." II: 189-193.
- Marsden, P., Meyer, C., Fuller, M., & Waller, G. (2002). The relationship between eating psychopathology and separation-individuation in young nonclinical women. *Journal of Nervous and Mental Disease*, 190, 710 713.
- Mash, E. & Wolfe, D. (2002). Abnormal Child Psychology, 2nd Edition. Belmont, CA: Wadsworth.
- McClanahan, G., & Holmbeck, G. (1992). Separation-individuation, family functioning, and psychological adjustment in college students: A construct validity study of the separation-individuation test of adolescence. *Journal of Personality Assessment*, 59, 468-485.
- McGue, M., Elkins, I., Walden, B., & Iacono, W. (2005). Perceptions of the parent adolescent relationship: A longitudinal investigation. *Developmental Psychology*, 41, 971-984.
- Millon, T., Green, C., & Meagher, R. (1982). Adolescent Personality Inventory, manual (2nd ed.). Minneapolis, MN: National Computer Systems.
- Mond, J., Phillipa, H., Rodgers, B., Owen, C., & Beumont, P. (2004). Temporal stability of the eating disorder examination questionnaire. *International Journal of Eating Disorders*, 36, 195-203.
- Newton, M. (2005). Exploring the psychopathology of anorexia nervosa: A Mahlerian standpoint. *Perspectives in Psychiatric Care*, 41, 172-180.
- NINM: Eating disorders. NIH publication No. 93-3477. Rockville, MD, 1994.

- Ohannessian, C., Lerner, R., Lerner, J., & von Eye, A. (2000). Adolescent-parent discrepancies in perceptions of family functioning and early adolescent self competence. *International Journal of Behavioral Development*, 24, 362-372.
- O'Kearney, R. (1996). Attachment disruption in anorexia nervosa and bulimia nervosa: A review of theory and empirical research. *International Journal of Eating Disorders*, 20, 115-127.
- Palazolli, M. (1978). Self-starvation: From individual to family therapy in the treatment of anorexia nervosa. New York: Jason Aronson.
- Palmer, R., Oppenheimer, R., Marshall, R. (1988). Eating-disordered patients remember their parents: A study using the parental-bonding instrument. *International Journal of Eating Disorders*, 7, 101-106.
- Parker, G. (1983). Parental affectionless control as an antecedent to adult depression: A risk factor delineated. *Archives of General Psychiatry*, 40, 956-960.
- Parker, G., Tupling, H., & Brown, L. B. (1979). A parental bonding instrument. *British Journal of Medical Psychology*, 52, 1-10.
- Patton, G., Carlin, J., Shao, Q., & Hibbert, M. (1997). Adolescent dieting: Healthyweight control or borderline eating disorder? *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 38, 299-306.
- Patton, G., Coffey, C., Posterino, M., Carlin, J., & Wolfe, R. (2001). Parental "affectionless control" in adolescent depressive disorder. *Social Psychiatry and Psychiatric Epidemiology*, 36, 475-480.
- Pelton, J., & Forehand, R. (2001). Discrepancy between mother and child perceptions of their relationship: Consequences for adolescents considered within the context of parental divorce. *Journal of Family Violence*, 16, 1-15.
- Pernick, Y., Nichols, J., Rauh, M., Kern, M., Ming, J., Lawson, M., & Wilfley, D. (2006). Disordered eating among a multi-racial/ethnic sample of female high-school athletes. *Journal of Adolescent Health*, 38, 689-695.
- Petersen, A., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. *Journal of Youth and Adolescence*, 17, 117-133.
- Pike, K., & Rodin, J. (1991). Mothers, daughters, and disordered eating. *Journal of Abnormal Psychology*, 100, 198-204.
- Pilowsky, D., Wickramartne, P., Nomura, Y., & Weissman, M. (2006). Family discord, parental depression, and psychopathology in offspring: 20-year follow

- -up. Journal of the American Academy of Child and Adolescent Psychiatry, 45, 452-460.
- Placanica, J. L., Faunce, G. J., & Job, R. F. S. (2002). The effect of fasting on attentional biases for food and body shape/weight words in high and low eating disorder inventory scorers. *International Journal of Eating Disorders*, 32, 79-90.
- Podar, I., Hannus, A., & Allik, J. (1999). Personality and affectivity characteristics associated with eating disorders: A comparison of eating disordered, weight preoccupied, and normal samples. *Journal of Personality Assessment*, 73, 133 -147.
- Pole, R., Waller, D., Stewart, S., & Parkin-Feigenbaum, L. (1988). Parental caringversus protection in bulimia. *International Journal of Eating Disorders*, 7,601-606.
- Polivy, J., & Herman, C. (1985). Dieting and bingeing: A causal analysis. *American Psychologist*, 40, 193-201.
- Pryor, T., & Wiederman, M. (1996). Measurement of non-clinical personality characteristics of women with anorexia nervosa and bulimia nervosa. *Journal of Personality Assessment*, 67, 414-421.
- Rhodes, B., & Kroger, J. (1992). Parental bonding and separation-individuation difficulties among lateadolescent eating disordered women. *Child Psychiatry and Human Development*, 22, 249-263.
- Ricca, V., Nacmias, B., Cellini, E., Di Bernado, M., Rotella, C., & Sorbi, S. (2002). 5 HT2A receptor gene polymorphism and eating disorders. *Neuroscience Letters*, 323, 105-108.
- Rothbart, M., Ahadi, S., & Evans, D. (2000). Temperament and personality: Origins and outcomes. *Journal of Personality and Social Psychology*, 78, 122-135.
- Rothbart, M., & Bates, J. (1998). Temperament. In W. Damon (Series Ed.) & N. Eisenberg (Vol. Ed), Handbook of child psychology: Vol 3. Social, emotional, and personality development (5th ed., pp. 105-176). New York: Wiley.
- Russell, G., Szmukler, G., Dare, C., & Eisler, I. (1987). An evaluation of family therapy in anorexia nervosa and bulimia nervosa. *Archives of General Psychiatry*, 44, 1057-1056.
- Savin-Williams, R., & Small, S. (1986). The timing of puberty and its relationship to adolescent and parent perceptions of family interactions. *Developmental Psychology*, 22, 342-347.

- Schmidt, F. (1996). Statistical significance testing for cumulative knowledge psychology: Implications for the training of researchers. *Psychological Methods*. 1, 115-129.
- Seifer, R. (2000). Temperament and goodness of fit: Implications for developmental psychopathology. In A. Sameroff, M. Lewis, & S. Miller (Editors), Handbook of Developmental Psychopathology (2nd ed., pp. 257-273). New York: Kluwer Academic/Plenum Pulishers.
- Sights, J.R., & Richards, H.C. (1984). Parents of bulimic women. *International Journal of Eating Disorders*, 3, 3-13.
- Smolak, L., & Levine, M. (1993). Separation-individuation difficulties and the distinction between bulimia nervosa and anorexia nervosa in college women. *International Journal of Eating Disorders*, 14, 33-41.
- Steinberg, L. (1987). Impact of puberty on family relations: Effects of pubertal status and pubertal timing. *Developmental Psychology*, 23, 451-460.
- Steinberg, L. (1988). Reciprocal relation between parent-child distance and pubertal maturation. *Developmental Psychology*, 24, 122-128.
- Steinberg, L. (2005). Adolescence. New York, NY: McGraw Hill.
- Steinberg, L., & Morris, A. (2001). Adolescent development. *Annual Review of Psychology*, 52, 83-110.
- Steiner-Adair, C. (1990). The body politic: Normal female adolescent development and the development of eating disorders. In C. Gilligan, N.P. Lyons & T.J. Hammer (Eds.), *Making connections: The relational worlds of adolescent girls at Emma Willard School* (pp. 162-182). Cambridge, MA: Harvard University Press.
- Stice, E., & Agras, W. (1998). Predicting onset and cessation bulimic behaviors during adolescence: A longitudinal grouping analysis. *Journal of Abnormal Psychology*, 107, 671-675.
- Strelau, J. (2001). The role of temperament as a moderator of stress. In T. Wachs & G. Kohnstamm (Eds.), *Temperament in context* (pp. 153-172). Mahwah, NJ: Lawrence Erlbaum Associates, Inc.
- Striegel-Moore, R., Dohm, F., Kraemer, H., Taylor, C., Daniels, S., Crawford, P., & Schreiber, G. (2003). Eating disorders in white and black women. *American Journal of Psychiatry*, 160, 1326-1331.
- Strober, M., Freeman, R., & Morrell, W. (1997). The long-term course of severe anorexia nervosa in adolescents: Survival analysis of recovery, relapse, and

- outcome predictors over 10-15 years in a prospective study. *International Journal of Eating Disorders*, 22, 339-360.
- Strober, M., Freeman, R., Lampert, C., Diamand, J., & Kaye, W. (2000). Controlled family study of anorexia nervosa and bulimia nervosa: Evidence of shared liability and transmission of partial syndromes. *American Journal of Psychiatry*, 157, 393-401.
- Taylor, J., Gilligan, G., & Sullivan, A. (1995). Between voice and silence: Women and girls, race and relationships. Cambridge, MA: Harvard University Press.
- Tiggeman, M., & Pickering, A. (1996). Role of television in adolescent women's body dissatisfaction and drive for thinness, *International Journal of Eating Disorders*, 20, 199-203.
- Tomotake, M., & Ohmori, T. (2002). Personality profiles in patients with eating disorders. *Journal of Medical Investigation*, 49, 87-96.
- Torgersen, S. (2000). Genetics. In M. Hersen & A. Bellack (Eds.), *Psychopathology in Adulthood* (pp. 55-76), Boston, MA: Allyn and Bacon.
- Toro, J., Gomez-Peresmitre, G., Sentis, J., Valles, A., Casula, V., Castro, J., Pineda, G., Leon, R., Platas, S., & Rodriguez, R. (2006). Eating disorders and body image in Spanish and Mexican female adolescents. *Social Psychiatry and Psychiatric Epidemiology*, 41, 556-565.
- Truby, H., & Paxton, S. (2002). Development of the Children's Body Image Scale. British Journal of Clinical Psychology, 41, 185-204.
- Vaidya, J., Grippo, A., Johnson, A., & Watson, D. (2004). A comparative developmental study of impulsivity in rats and humans: The role of reward sensitivity. *Annals of the New York Academy of Sciences*, 1021, 395-398.
- Vandereycken, W., Kog, E., & Vanderlinden, J. (1989). The family approach to eating disorders. New York: PMA.
- Vervaet, M., van Heeringen, C., & Audenaert, K. (2004). Personality-related characteristics in restricting versus binging and purging eating disordered patients. *Comprehensive Psychiatry*, 45, 37-43.
- Vitousek, K., & Manke, F. (1994). Personality variables and disorders in anorexia and bulimia nervosa. *Journal of Abnormal Psychology*, 103, 137-147.
- von Ranson, K.M., Klump, K.L., Iacono, W.G., & McGue, M. (2005). Development and validation of the Minnesota Eating Disorder Inventory: A brief measure of disordered eating attitudes and behaviors. *Eating Behaviors*, 6, 373-392.

- Wade, T.D., Bulik, C.M., Neale, M., & Kendler, K.S. (2000). Anorexia nervosa and major depression: An examination of shared genetic and environmental risk factors. *American Journal of Psychiatry*, 29, 925-934.
- Wade, T., Martin, N., & Tiggerman, M. (1998). Genetic and environmental risk factors for the weight and shape concerns characteristic of bulimia nervosa. *Psychological Medicine*, 28, 761-771.
- Walters, E., & Kendler, K. (1995) Anorexia nervosa and anorexic-like syndromes in a population-based female twin sample. *American Journal of Psychiatry*, 152, 64 -71.
- Wardle, J., & Beales, S. (1987). Restraint and food intake: an experimental study of eating patterns in the laboratory and in everyday life. *Behavior Research and Therapy*, 25, 179-185.
- Wassenaar, D., le Grange, D., Winship, J., & Lachenicht, L. (2000). The prevalence of eating disorder pathology in a cross-ethnic population of female students in South Africa. *European Eating Disorders Review*, 8, 225-236.
- Watson, D., Clark, L., & Harkness, A. (1994). Structures of personality and their relevance to psychopathology. *Journal of Abnormal Psychology*, 103, 18-31.
- Wertheim, E., Paxton, S., Maude, D., Szmukler, G., Gibbons, K., & Hiller, L. (1992). Psychosocial predictors of weight loss behaviors and binge eating in adolescent girls and boys. *International Journal of Eating Disorders*, 12, 151-160.
- Wiseman, C., Gray, J., Mosimann, J., & Ahrens, A. (1992). Cultural expectations of thinness in women: An update. *International Journal of Eating Disorders*, 11, 85-89.
- Wonderlich, S., Connolly, K., & Stice, E. (2004). Impulsivity as a risk factor for eating disorder behavior: Assessment implications with adolescents. *International Journal of Eating Disorders*, 36, 172-182.
- Wonderlich, S., Brewerton, T., Jocic, Z., Dansky, B., & Abbott, D. (1997). Relationship of childhood sexual abuse and eating disorders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 1107-1115.
- Wonderlich, S., Ukestad, L., & Perzacki, R. (1994). Perceptions of nonshared childhood environment in bulimia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, 33, 740-747.

