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Mother–Child and Father–Child Connectedness in Adolescence and Disordered Eating Symptoms in Young Adulthood

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Abstract

Purpose—The aim of the study was to examine mother–child connectedness and father–child connectedness in adolescence as potential protective factors against a range of disordered eating symptoms in young adulthood among males and females.

Methods—This study used data from the National Longitudinal Study of Adolescent to Adult Health (N = 13,532). Sex-stratified logistic regression models adjusted for demographic covariates were conducted to examine associations of youth-reported mother–child connectedness and father–child connectedness in adolescence (mean age = 15.4 years) with disordered eating symptoms in young adulthood (mean age = 21.8 years).

Results—In this nationally representative sample of U.S. young adults, 7.2% of participants reported binge eating-related concerns, 3.7% reported compensatory behaviors (e.g., self-induced vomiting) to control weight, and 8.6% reported fasting/skipping meals to control weight. Among females, both higher mother–child connectedness and higher father–child connectedness were associated with lower odds of binge eating–related concerns (mother–child: odds ratio [OR] = .83, 95% confidence interval [CI] = .74–.94; father–child: OR = .79, 95% CI = .69–.91), compensatory behaviors (mother–child: OR = .85, 95% CI = .75–.97; father–child: OR = .81, 95% CI = .69–.95), and fasting/skipping meals (mother–child: OR = .79, 95% CI = .72–.87; father–child: OR = .81, 95% CI = .73–.91). No statistically significant associations were observed for mother–child

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connectedness or father–child connectedness with future disordered eating symptoms among males.

Conclusions—These findings suggest that improving mother–child connectedness and father–child connectedness in adolescence may be valuable targets for eating disorders intervention, particularly among females.

Keywords

Feeding and eating disorders; Parent–child relations; Father–child relations; Mother–child relations; Adolescent; Young adult

Parent–child connectedness, defined as closeness, caring, and satisfaction in Parent–child relationships [1], has emerged as a protective factor across a wide range of adverse outcomes including emotional distress, suicidality, violence, substance abuse, and poor physical health [2,3]. Parent–child connectedness is grounded in attachment theory, which posits that healthy child development depends on an infant's ability to form a lasting emotional bond with at least one primary caregiver [4,5]. Attachment-related experiences guide the development of emotion regulation strategies, such that secure attachment fosters healthy emotion regulation and insecure attachment often contributes to emotion dysregulation [4,6]. Although early-life attachment is crucial, the influence of early-life attachment on continued development of emotion regulation strategies and later outcomes depends, to some extent, on the quality of parental care throughout childhood and adolescence [6]. Parent–child connectedness extends to those interactions between parents and children beyond infancy [1]. Given the importance of attachment in healthy child development [4,5] and the close ties between attachment and Parent–child connectedness [1], it is not surprising that Parent–child connectedness has emerged as a protective factor across numerous domains.

Considering that emotion dysregulation contributes to the onset and maintenance of eating disorders [7], Parent–child connectedness may be a salient protective factor in the domain of eating disorders as well. Disordered eating symptoms– including both cognitive and behavioral symptoms–represent a public health concern, given that they are associated with poor dietary intake [8], increased risk for full-threshold eating disorders [9], increased depressive symptoms [10], and suicidality [11]. Mother–child connectedness and father–child connectedness have cross-sectionally been found to have protective associations with binge eating and extreme weight control behaviors among adolescent boys and girls [12,13], as has family connectedness (i.e., connectedness at the family level rather than the Parent–child dyadic level) [14]. Other family relationship characteristics, including family functioning, family communication, unconditional support, and maternal caring, have also cross-sectionally been found to have protective associations with disordered eating symptoms among adolescents [15,16], and early memories of warmth and safeness have been found to have protective associations with disordered eating symptoms among young adults [17]. However, to our knowledge, no longitudinal studies have examined whether protective associations between Parent–child connectedness and disordered eating symptoms extend beyond adolescence (i.e., past 19 years of age). Children develop independence and separate from their parents during the transition from adolescence to adulthood [18], the same developmental period during which eating disorder risk has been found to increase

[19]. Therefore, understanding how mother–child connectedness and father–child connectedness may shape eating disorder risk beyond adolescence could have important intervention implications.

Evidence suggests that Parent–child connectedness is modifiable [20–22], and if improving Parent–child connectedness during adolescence could reduce the risk of eating disorders down the line, it may be a useful target for eating disorders treatment and prevention. Better understanding the roles of mother–child connectedness and father–child connectedness in relation to eating disorders could, therefore, have important clinical and public health implications. Using data from a large, nationally representative sample in the U.S., the objective of this study was to investigate the extent to which mother–child connectedness and father–child connectedness in adolescence are associated with a range of disordered eating symptoms in young adulthood among males and females.

Methods

Participants

This study used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health) [23]. Systematic sampling methods and implicit stratification were incorporated into the Add Health study design to ensure the sample was representative of U.S. schools with respect to region of country, urbanicity, school size, school type, and ethnicity. Wave 1 data were collected in 1994–1995 when participants were in grades 7–12, Wave 2 data were collected in 1996 when participants were in grades 8–12, and Wave 3 data were collected in 2001–2002 when participants were aged 18–26 years [24]. Of the 15,197 participants interviewed at Wave 3, 875 participants were excluded because of missing sampling weights and 790 participants who did not report either a mother or a father in the household in adolescence were excluded, leaving 13,532 participants available for analyses in the present study. The Add Health protocol was approved by the Institutional Review Board at the University of North Carolina at Chapel Hill [24].

Measures

Parent–child connectedness in adolescence—Mother–child connectedness and father–child connectedness were assessed at Wave 1 with the Relationship with Mother and Relationship with Father subscales of the Youth Asset Survey [25,26]. Five-point Likert-type scales were used for the following items: “How close do you feel to your [mother/father]?,” “Most of the time, your [mother/father] is warm and loving toward you,” “You are satisfied with the way your [mother/father] and you communicate with each other,” and “Overall, you are satisfied with your relationship with your [mother/father].” These items are similar to items used to assess Parent–child connectedness in previous studies [2,12,27,28]. We averaged responses to yield a continuous variable with possible scores ranging from 1 to 5, with higher scores indicating higher levels of mother–child connectedness (Cronbach’s $\alpha = .86$ in this sample) and father–child connectedness (Cronbach’s $\alpha = .90$ in this sample).

Disordered eating symptoms in young adulthood—Disordered eating symptoms were assessed at Wave III via self-report. Participants reporting that they had “eaten so much

in a short period that [they] would have been embarrassed if others had seen [them] do it" and/or "been afraid to start eating because [they] thought [they] would not be able to stop or control [their] eating" in the past 7 days were assigned a positive response for the dichotomous variable for binge eating–related concerns. Participants reporting that they "made [themselves] throw up," "took laxatives," "took weight-loss pills," and/or "used diuretics–that is, water pills" in the past 7 days to lose weight or stay the same weight were assigned a positive response for the dichotomous variable for compensatory behaviors. Participants reporting that they "fasted or skipped meals" in the past 7 days to lose weight or stay the same weight were assigned a positive response for the dichotomous variable for fasting/skipping meals. In addition, participants endorsing binge eating–related concerns, compensatory behaviors, and/or fasting/skipping meals were assigned a positive response for a dichotomous variable for any disordered eating symptoms.

Demographic covariates—The following variables were included as demographic covariates: participant age at Wave I (continuous), participant race/ethnicity (categorical: non-Hispanic white, non-Hispanic black, or other), family structure (categorical: mother and father, mother only, or father only), mother type (dichotomous: biological/adoptive or step/other), father type (dichotomous: biological/adoptive or step/other), highest parental education (categorical: less than high school, high school graduate or equivalent, some college/trade school, or graduated college or above), and percent federal poverty level in adolescence (continuous; calculated using parent-reported household income in 1994, participant-reported household size in 1994 or 1995, and 1994 federal poverty guidelines).

Statistical analysis—All analyses were conducted with SAS 9.4, using SAS Survey Procedures with U.S. census region as the stratum variable, school as the cluster variable, and sampling weights to account for the complex sampling design in Add Health [29].

Descriptive statistics—We computed univariate statistics for mother–child connectedness, father–child connectedness, disordered eating symptoms, and demographic covariates. We also computed bivariate statistics by participant sex.

Multiple imputation—Data were missing at the following rates: 21% for percent federal poverty level, 4% for highest parental education, and less than 1% for mother–child connectedness (among participants reporting a mother in the household), father–child connectedness (among participants reporting a father in the household), disordered eating symptoms, age, sex, and race/ethnicity. To preserve sample size, we conducted multiple imputation with the assumption that data were missing at random. We created 20 imputed datasets using the fully conditional specification method in the MI procedure in SAS 9.4 [30]. In sensitivity analyses, we conducted analyses with only demographic covariates imputed and using complete case data only.

Logistic regression—On each imputed dataset, we ran logistic regression models examining associations of mother–child connectedness and father–child connectedness in adolescence with disordered eating symptoms in young adulthood, adjusted for demographic covariates. We ran separate models for any disordered eating symptoms, binge eating–related concerns, compensatory behaviors, and fasting/skipping meals. There is theoretical

support for distinct relationships between mother–son, mother–daughter, father–son, and father–daughter dyads [31–33], as well as empirical support for sex differences in associations between family relationship characteristics and mental health outcomes [34,35]. In addition, several demographic covariates differed by participant sex in the present sample (Table 1). For these reasons, all models were stratified by participant sex a priori.

Combining inference from multiply imputed datasets—Results from logistic regression analyses were combined and summarized, using both within-imputation and between-imputation variance to reflect uncertainty because of the missing data [36].

Results

Summary characteristics of the study population

Descriptive statistics are presented in Table 1. In young adulthood (mean age = 21.75 years), 16.6% of participants reported any disordered eating symptoms, with 7.2% reporting binge eating–related concerns, 3.7% reporting compensatory behaviors, and 8.6% reporting fasting/skipping meals. Prevalence estimates for each type of disordered eating symptom differed by sex, with females reporting higher prevalence than males (all ps < .001). Mean (standard error) mother–child connectedness and father–child connectedness levels in adolescence were 4.33 (.01) and 4.13 (.02), respectively, with higher levels of both among males than females (both ps < .001). Mother–child connectedness and father–child connectedness were positively correlated (r = .47; p < .001).

Associations between parent–child connectedness and disordered eating symptoms

Sex-stratified, demographics-adjusted associations of mother–child connectedness and father–child connectedness with disordered eating symptoms are presented in Table 2. Among females, higher mother–child connectedness and higher father–child connectedness were associated with lower odds of any disordered eating symptoms (mother–child: odds ratio [OR] = .82, 95% confidence interval [CI] = .76–.89; father–child: OR = .80, 95% CI = .72–.89), binge eating–related concerns (mother–child: OR = .83, 95% CI = .74–.94; father–child: OR = .79, 95% CI = .69–.91), compensatory behaviors (mother–child: OR = .85, 95% CI = .75–.97; father–child: OR = .81, 95% CI = .69–.95), and fasting/skipping meals (mother–child: OR = .79, 95% CI = .72–.87; father–child: OR = .81, 95% CI = .73–.91). No statistically significant associations were observed for mother–child connectedness or father–child connectedness among males. Results were not substantially different in sensitivity analyses using complete cases only and imputing only demographic covariates.

Discussion

This study examined associations of mother–child connectedness and father–child connectedness during adolescence with a range of disordered eating symptoms during young adulthood among males and females. We found that both higher mother–child connectedness and higher father–child connectedness in adolescence were associated with lower odds of binge eating–related concerns, compensatory behaviors, and fasting/skipping meals in young adulthood among females, but neither mother–child connectedness nor father–child

connectedness in adolescence were associated with any of these disordered eating symptoms in young adulthood among males. These results suggest that improving mother–child connectedness and father–child connectedness in adolescence may be valuable targets for eating disorders intervention among girls; however, there may be other processes operating for boys.

Our results build on previous cross-sectional findings that mother–child connectedness and father–child connectedness are associated with lower odds of disordered eating symptoms among both adolescent boys and girls [13] by providing evidence that these associations extend into young adulthood for females but not males. Although the pattern by sex we observed is incongruent with cross-sectional findings among adolescents [13], there is previous evidence to suggest that associations between Parent–child connectedness and favorable outcomes may be more enduring for girls versus boys. For example, higher Parent–child connectedness has been found to be associated with increases in body satisfaction over time among adolescent girls but not boys [37]. The incongruence between cross-sectional and longitudinal findings may be related to sex differences in the way relationships with parents change throughout adolescence. Although perceived parental support declines from early to middle adolescence for both boys and girls, it has been found to increase for girls but stabilize for boys between middle and late adolescence [38]. Similarly, girls have been found to need more emotional support from their parents than boys during the process of separating from their parents in late adolescence [39]. Therefore, receiving emotional support during this time may be particularly important for girls. These differences may help explain why favorable outcomes associated with both mother–child connectedness and father–child connectedness appear to be more enduring for females than males.

Associations between Parent–child connectedness and disordered eating symptoms may be mediated by factors similar to those that have been found to help explain associations between insecure attachment and disordered eating symptoms. Recent meta-analytic findings suggest that maladaptive emotion regulation and depressive symptoms are strong mediators of associations between insecure attachment and disordered eating symptoms, whereas body dissatisfaction, neuroticism, perfectionism, mindfulness, and social comparison are weaker mediators [40]. Future research would be necessary to determine whether or not maladaptive emotion regulation and depressive symptoms also mediate associations between Parent–child connectedness and disordered eating symptoms, but it is plausible that higher Parent–child connectedness may lead to less maladaptive emotion regulation and depressive symptoms, which may, in turn, lead to less disordered eating symptoms.

A key strength of this study is the availability of data from a large, nationally representative sample of participants in the U.S. followed from adolescence into young adulthood. Young adulthood is a critical period, as eating disorder risk has been found to increase during this period [19]. Another strength of this study was the use of Parent–child connectedness measures with established reliability. Furthermore, this study assessed father–child relationships, which have been understudied relative to mother–child relationships.

This study also had limitations, which included the use of single-item measures with a 7-day assessment time frame to assess disordered eating symptoms. Moreover, disordered eating symptoms were not assessed in adolescence; therefore, we were unable to control for pre-existing disordered eating symptoms or examine whether the relationship between Parent–child connectedness and disordered eating symptoms is bidirectional. Given the observational nature of the data, observed associations cannot be interpreted causally. Observed associations may instead reflect correlation; for example, participants with greater levels of general psychological well-being may experience higher Parent–child connectedness and also experience fewer disordered eating symptoms. In addition, because of the manner in which Add Health data were collected, we were unable to examine associations for same-sex parents. Finally, given that data from adolescence were collected in 1994–1995 and data from young adulthood were collected in 2001–2002, it is possible that findings may not be as relevant for adolescents and young adults today. Regardless, findings from this study offer important contributions to understanding how mother–child connectedness and father–child connectedness may influence the development of eating disorders.

Given that associations were not found for males in the present study, more research is needed to better understand what factors might be associated with lower eating disorder risk in males. For example, peer factors may be more salient than family factors in relation to eating disorder risk among males. Conversely, the findings from this study suggest that for females, improving mother–child connectedness and father–child connectedness in adolescence may help reduce subsequent eating disorder risk. As Parent–child connectedness is a reciprocal construct influenced by both the parent and the child, interventions to improve Parent–child connectedness should focus not only on the parents but also on the children and/or family-level factors (e.g., improving family-level communication). Furthermore, given that Parent–child connectedness has been established as a protective factor across a wide range of domains, effective interventions to increase Parent–child connectedness could have widespread positive impact beyond reducing the burden of eating disorders.

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IMPLICATIONS AND CONTRIBUTION

This study suggests that mother–child connectedness and father–child connectedness in adolescence may protect against future disordered eating symptoms for females but not males. These results can help guide eating disorders prevention and early intervention efforts.

Table 1

Sample characteristics, overall and by participants' sex

	Overall (N = 13,532)	Males (N = 6,432)	Females (N = 7,100)	*p*
	Sampled frequency (weighted percent)			
Race/ethnicity				
Non-Hispanic white	7,376 (68.1)	3,488 (67.6)	3,888 (68.5)	.09
Non-Hispanic black	2,734 (14.8)	1,205 (14.4)	1,529(15.3)	
Other	3,381 (17.1)	1,726(18.0)	1,655 (16.2)	
Percent federal poverty level				
<100%	1,656(15.8)	764 (15.3)	892 (16.2)	.69
100%—199%	2,223 (20.5)	1,082 (20.8)	1,141 (20.2)	
200%—399%	3,957 (38.6)	1,899 (39.2)	2,058 (38.1)	
≥400%	2,568 (25.1)	1,236 (24.7)	1,332 (25.5)	
Highest parental education				
Less than high school	1,615 (11.9)	7,24(11.7)	891 (12.1)	.12
High school graduate or equivalent	3,768 (31.1)	1,788 (31.1)	1,980 (31.1)	
Some college/trade school	2,785 (21.6)	1,275 (20.7)	1,510(22.5)	
Graduated college or above	4,906 (35.4)	2,395 (36.5)	2,511 (34.2)	
Family structure				
Mother and father	9,507 (71.1)	4,590 (71.9)	4,917 (70.3)	<.001
Mother only	3,523 (25.1)	1,567 (23.8)	1,956 (26.6)	
Father only	502 (3.7)	275 (4.3)	227 (3.1)	
Mother type				
Biological/adoptive	12,720 (97.7)	5,990 (97.2)	6,730 (98.1)	.006
Step/other	310(2.3)	167 (2.8)	143 (1.9)	
Father type				
Biological/adoptive	8,828 (88.1)	4,306 (88.3)	4,522 (87.9)	.63
Step/other	1,181 (11.9)	559(11.7)	622 (12.1)	
Any disordered eating symptoms	2,347 (16.6)	798 (11.4)	1,549 (22.0)	<.001
Binge eating—related concerns	1,017 (7.2)	399 (5.7)	618 (8.8)	<.001
Compensatory behaviors	549 (3.7)	108 (1.5)	441 (6.0)	<.001

	Overall (N = 13,532)	Males (N = 6,432)	Females (N = 7,100)	*p*
	Sampled frequency (weighted percent)			
Fasting/skipping meals	1,225 (8.6)	373 (5.3)	852 (12.0)	<.001
	Mean (standard error)			*p*
Age at baseline (y)	15.37 (.12)	15.46 (.12)	15.29 (.12)	<.001
Age at follow-up (y)	21.75 (.12)	21.84 (.12)	21.66 (.12)	<.001
Mother—child connectedness in adolescence	4.33 (.01)	4.41 (.02)	4.24 (.02)	<.001
Father—child connectedness in adolescence	4.13 (.02)	4.20 (.02)	4.05 (.02)	<.001

Table 2

Associations between parent—child connectedness in adolescence and disordered eating symptoms in young adulthood

	Any disordered eating symptoms, OR (95% CI)	Binge eating—related concerns, OR (95% CI)	Compensatory behaviors, OR (95% CI)	Fasting/skipping meals, OR (95% CI)
Mother—child connectedness				
Among males	.92 (.78—1.09)	.89 (.70—1.15)	1.00 (.60—1.65)	.91 (.73—1.14)
Among females	.82 (.76—.89)***	.83 (.74—.94)**	.85 (.75—.97)*	.79 (.72—.87)***
Father—child connectedness				
Among males	1.01 (.85—1.20)	1.19 (.93—1.53)	1.05 (.66—1.67)	.87 (.69—1.10)
Among females	.80 (.72—.89)***	.79 (.69—.91)***	.81 (.69—.95)**	.81 (.73—.91)***

Models adjusted for participant age, participant race/ethnicity, highest parental education, percent federal poverty level in adolescence, family structure, and mother/ father type.

CI = confidence interval; OR = odds ratio.

* $p < .05$.

** $p < .01$.

*** $p < .001$.